The pathology and diagnosis of diseases of the chest; illustrated especially by a rational exposition of their physical signs. With new researches on the sounds of the heart / [Charles J.B. Williams].

THE

PATHOLOGY AND DIAGNOSIS

OF

DISEASES OF THE CHEST;

ILLUSTRATED ESPECIALLY BY

A Rational Exposition of their Physical Signs.

WITH NEW RESEARCHES ON

THE SOUNDS OF THE HEART.

BY

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THIRD EDITION,

MUCH ENLARGED.

LONDON:
JOHN CHURCHILL, PRINCES STREET, SOHO.

1835.

LONDON:
C. RICHARDS, PRINTER, ST. MARTIN'S LANE, CHARING CROSS.

TO

WILLIAM FREDERICK CHAMBERS,

M.D. F.R.S.

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS,
SENIOR PHYSICIAN TO ST. GEORGE'S HOSPITAL, ETC. ETC.

My dear Sir,

I have again the pleasure of offering you a trifling tribute of my respect for the distinguished talents and honourable candour, which uniformly mark your professional course. The ability and discernment which you have long evinced with respect to the subjects of this work, make your approval of its contents a point which I especially desire.

Earnestly wishing you continued health in your eminent and useful career,

I remain, my dear Sir,

Your very faithful and obliged Servant,

CHARLES J. B. WILLIAMS.

CONTENTS.

EXPLANATION OF THE PLATES.

PREFACE.

THE gratifying manner in which the former editions of this work have been received, has encouraged me to devote much attention and care to render it, in its present form, more worthy of the same kind reception. The progress which patient and comprehensive study constantly ensures to pathological science, has furnished many important additions from the works of various distinguished labourers in this field; and these, together with the later results of my own experience and study, have greatly improved, and nearly doubled, the matter in the present volume. Of these additions, I may mention, that the sections *On the Ocular and Manual Examination of the Chest; On Expectoration; On Encephaloid Disease, Melanosis, &c.; On Diseases of the Bronchial Glands;* and the whole of Part III. *On the Auscultation of the Heart,*—are new; and large additions have been made to the sections on *Bronchitis, Peripneumony, Pleurisy, Pneumothorax,* and *Phthisis.*

The experimental researches on the sounds of the heart, contained in Part III. will, it is hoped, give a greater accuracy and precision to the pathology of

that organ, than it has hitherto acquired; and although in the subsequent chapter on this subject I have expressed much doubt about the diagnosis of certain lesions, yet the attempt to distinguish and separate the points that are clear and useful, may lighten the labour and disappointment of the student, and assist future inquirers to clear up the remaining difficulties.

It has gratified me to find that the connection which I have endeavoured to establish between physical diagnosis and pathology, by tracing signs to their mechanism, and the mechanism through its variations to the production of the signs, has been generally approved of and adopted, and I have now only to repeat my conviction, strengthened by further experience, that the easiest way to study auscultation, and the only sure way to profit by it, is through a knowledge of pathology, and of the physical laws of sound.

Within the last few years, auscultation has been rapidly advanced in the estimation of the profession; and I think this is much to be ascribed to the more rational and moderate manner in which its pretensions have been urged. Instead of being blindly put forth as infallibly indicating such and such diseases, its signs are now appreciated only as they inform us of certain physical changes in the organs within the chest; whilst the aid of our other senses, a due attention to the general symptoms, and a careful reflection on all these evidences, are also required,

to enlighten us in our study of disease, and to suggest remedies for its removal. It is with a view to prevent the student from relying too exclusively on the physical signs, that I have in the present edition added a short summary of the general symptoms of each disease, which in very many instances are more important in guiding our practice, than the local signs; and for the same reason, and also because the principles of pathology and diagnosis are more fully considered, I have somewhat altered the title of the work, hoping that it may still prove an easy guide to a knowledge of auscultation, and at the same time explain the general pathology and diagnosis of Diseases of the Chest.

Half-Moon Street, Piccadilly,
March 24, 1835.

PREFACE

TO THE FIRST EDITION.

A DISCOVERY, a new doctrine, or an innovation of any kind, produces a curious agitation in the public mind, which, in a remarkable manner, illustrates the paradoxical and heterogeneous composition of human character. Tossed to and fro by the exertions of its opponents, and of its scarcely less opposing ultra-partisans, it resembles a pendulum; and vibrating irregularly between many disturbing forces, it is driven out of the real sphere of its importance, and from that true point of utility to which its intrinsic weight and worth would cause it to gravitate.

More particularly has this happened in medicine, which, having few standard or fixed points to steady it, has been ever too much at the mercy of contending opinions. The localization of diseases is a characteristic doctrine of the present day; and assuredly such a system would be the most scientific, that could trace the multifarious forms of disease to a few simple primary lesions of tissue, or well defined alterations of function; and that plan of practice the most efficacious that could concentrate its efforts against the very root of evil, and stop at its very spring-source the current of disorder. But we are far from having attained such a perfection; and let caution, therefore, remind us, that hastily to follow a light, which, however pure and real, is yet at distance too remote

to shed its rays upon our paths, is scarcely less dangerous, than to chase an empty ignis fatuus; that to grasp at an object, however perfect and substantial, so far beyond our present reach, is not less futile than to catch at an illusory shadow.

The local study of diseases must not, therefore, remove our attention from their general phenomena; our examination of their physical nature must not exclude the consideration of many constitutional effects, that by reaction may often become converted into causes; and still less should physical signs of doubtful import make us neglect obvious disorder of the system.

But, thus limited, the local study of diseases is more advantageous than the knowledge of their general forms; an examination of their physical signs, when possible, more useful than the perplexing consideration of a host of uncertain and fallacious constitutional symptoms; and when physical signs are wanting, or beyond the sphere of our observation, those constitutional ones are our best guides, which most nearly depend on the physical and unchangeable character of the disease. For the local study of a disease acquaints us with its proximate and essential cause, and this knowledge suggests means for its removal; and by a study of its physical signs, and of those general ones most allied to them, we obtain the most certain method of discovering its existence, and of distinguishing its character.

Further than this, I shall not expatiate on the advantages or disadvantages of the new methods of diagnosis of diseases of the chest. They are now too well understood and appreciated to be in danger of

yielding to the opposition of prejudice, or of falling into oblivion through neglect. Too many ears have been opened to the language of disease, to suffer its warnings to be lost without a listener; too many minds are convinced of the truth of its admonitions, to permit them to pass, as hitherto, unheeded. Those who are disposed to study the signs of auscultation and percussion, will soon find in that study the proofs of their merit and importance; those who will not examine them, are not likely to be more moved by any commendations that I could bestow, than by those that have already been written in their favour.

The "Traité de l'Auscultation Médiate," and the perfect translation of Dr. Forbes, are at length generally appreciated, even in this country, slow to award its meed of praise. The homage paid to the talents of the author, gives me a gratification that almost seems personal; and I doubt not that this feeling is shared by others of his pupils, in whom his urbane and amiable deportment created a sincere regard for the man, as his great mental abilities excited our respect. His great talents are known to the public through the medium of his writings; but those who attended his *clinique* can only appreciate the wonderful acuteness of his perception, and faculty for observation, that enabled him to carry his discovery to the degree of perfection in which he left it; and they, above all, witnessed, felt, and profited by the solicitous interest which he showed to make others partake of its inestimable advantages. They felt in his death the loss of a friend; as science had to deplore the loss of his talents: he has wrought a good work for both; the feeling shall last while they last;

science has recorded his name on her tablets for ever :—

" Illum aget pennâ metuente solvi
" Fama superstes."

Let me say a few words on the objects and plan of the present work.

I have ever found in practice, and it is perfectly conformable to reason, that the easiest and most agreeable way to study physical signs, and to attain the surest criterion of their value and importance, is by considering how they are caused, or what are the relations in which they stand to the physiological and pathological states that produce them. Attempts to discover the rationale of the general symptoms of disease, have been as unsuccessful as our knowledge of the functions or properties on which they depend is scanty and imperfect ; and inquiries of this kind have been proportionately unsatisfactory and unprofitable. But physical signs stand on the broad and intelligible basis of physical laws, and are as readily explained as other simple phenomena illustrated by natural philosophy. It has been my endeavour to exhibit them, as far as possible, in this intelligible view ; to show the mechanism by which the signs are produced, and the manner in which, according to fixed laws, they result as phenomena ; to make a knowledge of the pathology predicate the signs, and a knowledge of the signs indicate the pathology ; and by thus familiarizing the mind with their principles, to enable it to understand the multifarious forms which, by combination, these signs may assume, and to judge of the corresponding physical changes that modify or produce them.

I have not refrained, when the subject seemed to require it, or where I had any new view to offer, from entering on some questions of general pathology. I am not clear that I have been judicious in so doing; for the slight views, that I have given of these questions, may be deemed too superficial and unsupported to be satisfactory; and had they been developed more fully, it would have completely changed the size and nature of the work. These opinions, as well as my acquaintance with the physical signs, are the result of some extent of study and observation, prosecuted chiefly in the wards of La Charité, where Laennec taught, and Andral prosecuted his labours. Most of the facts which I have described have appeared in the works of these illustrious men; and wherever my experience has not enabled me to give the same as the result of my own observation, I have referred to their competent authority. Where, in point of fact, or opinion, I have differed from them or from others, I would wish my dissent to be viewed rather as a question to be answered by others, than as in itself superseding former observations or opinions.

London, May 21, 1828.

The reader is particularly requested to correct with the pen the following *Errata*, as some of them materially injure the sense.

Page 96, line 23, for *habitual* read *mutual.*
— 118 — 19, for *or* read *on.*
— 121 — 4, erase *or.*
— 122 — 5, for *is* read *are.*
— 135 — 21, for *alternatives* read *alteratives.*
— 167 — 9 from bottom, (note) for *distillation* read *dilatation.*
— 168 — 3, (note) for *intimate* read *imitate.*
— 185 — 16, erase the comma after *blood.*
— 198 — 3, for *shifting* read *whiffing.*
— *ib.* last line, for *be* read *is.*
— 203 last line but one, for *made* read *make*

THE

PATHOLOGY AND DIAGNOSIS,

&c. &c.

PART I.—CHAP. I.

ON THE PHYSICAL SIGNS OF DISEASE.

By *physical signs* I mean such as depend on the direct operation of known laws of natural philosophy on our organs of sensation. As they are produced by the physical state or condition of a part, they become indications of that state or condition, as certain, as the laws, of which they are exemplifications, are unerring and sure: and the physical state of a part of the body may be ascertained with more or less certainty, as its physical signs, or relations to these natural laws, are more or less appreciable by our senses.

The organs of *vision*, impressed by forms and properties in relation to light, and perfected by the immediate correction of touch, are, both by nature and habit, calculated to give us a more perfect knowledge of external objects, that can be derived from the other senses. But the number of diseases that come under the cognizance of vision is very limited, as by far the greater part of the body is excluded from its sphere. Derangements of the surface, and of the openings of some of the passages to the interior, can alone be subjected to the direct examination of the eye. Mediately, physical changes of internal organs can be perceived by sight only, when their size,

form, or position, is so far altered as to cause displacement of some external part; and the knowledge that such a sign gives us, although scanty, is often valuable.

The sense of *touch*, or *tact*, will, in the same cases, furnish us with further knowledge as to the form, substance, and constitution of a diseased part; and when perfected by experience, may frequently discover organic changes that are altogether imperceptible to sight.

The sense of *smell* is more rarely qualified to distinguish disease; as its impressions can only be conveyed through the medium of air; yet we shall find that cases are not wanting in which this sense may assist us in diagnosis.

Sound, as it may be both generated and propagated in every form of matter, solid, liquid, and aeriform, may be therefore considered a mean of examination of parts removed from sight and tact, more promising as its sphere is less limited. It is requisite, however, that the object of examination be capable of producing or transmitting *audible* sound; and that changes in the part, cause in the sound thus produced or transmitted, corresponding changes that may be appreciated by the ear. The relations of the organ of hearing to the qualities of external objects, are, in ordinary life, much less exercised than those of tact and vision. Yet continual experience proves to us that the substance or consistence of simple objects is, in some measure, declared by the sound which they emit when struck. The sound of liquids in contact with air, is familiarly distinguished from that of solids in the same medium, and a little more attention discovers the varied sounds which air in motion produces in contact with solids in different forms. The rushing of the torrent, the murmur of the brook, the whistling of the wind, the howl of the tempest, the rustling of leaves, the rumbling of carriages, the trampling of horses, are instances out of thousands in which we recognise in the sound the nature of its physical cause; and the mental process is here

so simple, that the association of the sound with the object that produces it, is scarcely more than an instinctive perception.

Such scanty knowledge of the relations of sound suffices for the common purposes of life: to study them more closely, with a view to discover the nature of objects, were a work of supererogation whilst sight and tact are capable of giving us much more perfect and certain information. But an individual deprived of sight substitutes a perfection of tact, and of hearing and distinguishing sounds, which, in a great degree, compensates for his want of vision. So likewise may we, with equal advantage, so perfect our sense of hearing, as to make its indications available to instruct us of objects beyond the sphere of tact and vision. Now such perfection must in great measure depend on the practice of each individual, as a knowledge of simple sensations cannot be transferred by description; but the study may be much assisted and simplified by a general knowledge of the chief laws according to which sound is produced and propagated. Since the publication of the first edition of this work, in 1828, the science of acoustics has been more fully studied, both on its own account, and for the illustrations which it affords to optics, now also a science of vibrations. The student may therefore with greater confidence be referred to the authorities named below;* but there are still some properties of sound not developed in other works, and, as they are essential to a right understanding of

* Sir J. Herschell's article on Sound, in the Encyclopædia Metropolitana, 1830. The article Acoustics, by Sir J. Leslie, in tbe Edinburgh Encyclopædia, 1830. Dr. Arnott's Elements of Physics, Vol I. 1833: this valuable work will save students from the gross error, hitherto lamentably prevalent, of attempting to understand the complexities of the animal machine, without a previous knowledge of the laws of simpler matter. Some interesting papers on sound, by M. Savart, will be found in the "Annales de Chimie," Vols. 14, 24, 30, &c. and I may refer those interested in the matter to my own communication to the physical section of the British Association for the Advancement of Science, at their late meeting at Edinburgh, and published in the Philosophical Magazine, January, 1835.

the acoustic phenomena of the chest, I shall retain, with some alterations, my original remarks on this subject.

ON THE NATURE AND PROPERTIES OF SOUND.

Sound, *physiologically speaking*, is an impression communicated to our sense of hearing by certain impulses of matter. Sound, in a *physical sense*, may be defined as *motion of a certain velocity resisted with a certain force.* The moving and the resisting forces, acting in opposite ways, constitute the vibrations of sound. All matter is susceptible of sonorous vibrations; but the degrees of this susceptibility are as varied as matter is diversified in form and nature. As a general rule it may be stated, *that it is in proportion to the strength and uniformity of the molecular elasticity* in the matter.

By the term *molecular elasticity*, is meant that force by which the molecules of a body are held at a certain distance from each other, and resist any effort to displace them from it. Thus, glass and steel may be said to possess molecular elasticity in a powerful degree, because any external impulse is instantaneously communicated from particle to particle throughout their whole mass; and it is not lost or broken by the yielding or displacement of the molecules at the point struck. Air, and other fluids, on the other hand, cannot readily be thrown into vibrations, unless the impulse be very abrupt, or applied to some extent of surface, by which it becomes communicated to many particles at once.

I conceive that the motion of matter, producing sound, should be considered as molecular, although the result is the vibration of the mass. Let it be represented thus:—An impulse being impinged on certain molecules, momentarily overcomes the resistance of their inertia, and causes them to start from their place; that force of repulsion, which, existing between the molecules, resists the attempt to approximate them, transfers the impulse from molecule to molecule, and thus ex-

tends it throughout the mass. The impulse that forced these molecules from their position, being overcome by the reaction of the elastic forces (attractive and repulsive), these forces drive them back to beyond their proper station, whence, from the same cause, they again spring back; until, by a series of these alternating vibratory motions, the disturbing force is lost.

The assimilatory or propagating power, then, of these vibrations, depends on the molecular elasticity of the body; that is to say, on the repulsive and attractive forces that subsist between the molecules of which it is composed; and in proportion as these forces are strong to re-act on a mechanical impulse, they will convert that impulse into a sonorous vibration. It is likewise apparent that uniformity, or equality of molecular elasticity, favours the propagation of sonorous vibrations. For if the elasticity of some molecules be less than that of others, the reaction being less, will produce vibrations not consentaneous with those of the others, and may impair, or even destroy them. Let us illustrate this by the vibrations of pendula. Suppose a number of pendula suspended in a line, and in the act of vibration: if these pendula are of the same length, the vibrations will be equal and consentaneous, and will neither interfere with, nor interrupt each other. Such are the vibrations in bodies whose molecular elasticity is uniform. But suppose the pendula of different lengths, and the vibrations, therefore, unequal, the motions would then interfere with and neutralize each other, and this the more effectually, the more varied and irregular they are.*

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* Lest it should be supposed that, in these and subsequent illustrations, I have borrowed, without sufficient acknowledgement, from Sir J. Herschell's Essay, before alluded to, I beg to state, that the above explanations were published nearly in their present form in the first edition of this work, about three years before Sir J. Herschell's paper appeared. I have not the presumption to suppose that that distinguished philosopher ever saw them; but it is satisfactory and gratifying to me, that he has advanced views and modes of illustration very similar to mine.

There are, however, some vibrations that, although they are not synchronous, nevertheless promote each other, and these constitute what are called harmonic sounds. To show how this is effected, let us again refer to the pendulum. We have already remarked that pendula of the same length vibrate synchronously, and may, therefore, promote and strengthen each other. This is the harmony of unison. Suppose one pendulum half the length of the other; it makes double the number of vibrations in the same space of time, and, being regularly in the same ratio of striking two for every one of the other, the vibrations do not counteract each other. This concord or harmony of vibrations of sound, produces the harmonic note of the octave. The same illustration will enable us to conceive the harmonics of the fifth, the fourth, and the third; the ratio of their vibrations being as 3 to 2, 4 to 3, and 5 to 4, of the key note; and in like manner of other harmonics. These illustrations will render intelligible not only the simple sounds of separate bodies, but also the various compound noises which we are accustomed to hear; for, owing to the unequal molecular elasticity of the bodies in which they are produced, these sounds are always compound, and consist of a variety of vibrations, which may increase or neutralize each other according as the arithmetical relations of their motions harmonize or disagree: in the former case the sound will be a prolonged tone; in the latter a mere stroke or knock.

The propagation or conduction of sound from body to body is subject to the same rule; and, in fact, it consists in the transmission of the same impulse, producing sonorous vibrations from one body to another. A sound will, therefore, *cæteris paribus*, be best conducted by those bodies which approach in degree and strength of molecular elasticity the body in which that sound is generated. Thus a sound produced in air will be best propagated by air; one produced in a solid will be most completely conducted by a solid of the same den-

sity and hardness, &c. On the other hand, bodies, very different in density, receive and transmit sonorous vibrations very imperfectly. Thus air transmits, in a very impaired degree, the sounds produced in dense bodies, such as metals; and the sonorous vibrations of air are scarcely received by dense bodies.

The sounds produced by the collision of solids, and transmitted to us through air, are, nevertheless, among the loudest that we hear; but this is by reason of the law before stated, that those bodies are most susceptible of sonorous vibrations, in which the molecular elasticity is greatest, as well as most uniform; and such sounds are incomparably louder when heard through solids, instead of through air. The transfer of sonorous vibrations may, however, be greatly favoured in another way, by bringing a large surface of the solid vibrating body in contact with the air, and otherwise modifying its form, as in the case of bells, &c. This is a separate branch of acoustics, and is not sufficiently connected with our subject to require notice here.

There are many substances that prove bad generators or conductors of sound, from their being of unequal density; and those are worst in which this inequality is greatest. Linen and woollen stuffs are examples: the threads of which they are composed, leave interstices, which contain air very different in density from the solid fibres. In paper and pasteboard, the same fibres pressed closer together, and forming a more solid mass, become a far better conductor. The same is the case with all spongy bodies.

It now becomes apparent why the loudness of sounds does not always appear to us proportioned to the hardness and density of the bodies in which they are produced. Air is commonly the medium through which sounds are conducted to our ears; and this is a body of such tenuity that it much impairs those produced in solids, although, physically, they are

the loudest. We are thus relieved from the danger of injury to our organs of hearing, from sounds that might be too powerful for them to bear; and this happy provision supersedes the necessity of providing them with a defensive apparatus for their occasional occlusion, which we find to be in various degrees necessary for the other organs of sense.

Many of the sounds which we are accustomed to hear, are generated as well as conducted in air, and in this medium they may be of a most powerful kind, but they can be only imperfectly transmitted by solid conductors, which reflect and convert them into echoes. Thus in explosions, and notes of flutes and of other instruments of the whistle kind, the motion of air constitutes the impulse as well as the sound, and a confined column of air is the best conductor. From the recent researches of M. Savart, it is probable that the sound of the voice also originates solely in air; and not as we formerly supposed, in the motions of the glottis throwing the air into vibrations, as in reed instruments. In the sounds of these latter the air is equally the sonorous body, but it is thrown into vibrations by the mechanical motions of a solid which are reciprocally affected by the column of air which they cause to vibrate, and their sounds may be propagated either by a solid conductor communicating with the reed, or by air, as in other wind instruments. Of the same mixed kind is the hum of insects: the rapid motion of their wings produces in air a corresponding series of vibrations, which, when it attains a certain rate of frequency, constitutes sound, the pitch or key of this sound being high in proportion to the degree of frequency beyond this rate. The sounds of cords and bars, although capable of impressing air in a similar manner, more properly belong to the solid material, and, like those from the percussion of solids, are independent of the surrounding medium; but their effect on our ears will necessarily depend on the conducting power of this medium, which if rare and unresist-

ing, as in the case of air, receives and transmits but imperfectly the vibrations of dense and highly elastic solids. A material of intermediate density, but with strong molecular elasticity, will greatly facilitate the transfer of the vibrations of a sonorous solid to the air, and light rigid woods possess these qualities in an eminent degree. Thus the percussion of hard metallic bodies sounds much louder when they are in contact with wood, which by its great molecular elasticity receives accurately the vibrations from the metal, whilst from its lightness or small inertia, these vibrations become sufficiently extensive to take full effect on the air. The effect of the sounding board of musical instruments depends on the same principle, and may be pleasingly illustrated by the common tuning fork, which gives out little sound, after it is struck, as long as it is held between the fingers, but no sooner is it placed in communication with the table or a sounding board, either directly or through a long piece of wood, than its note becomes freely and fully transferred to the air. The same principle, as we shall afterwards see, makes light rigid woods the best material for ordinary stethoscopes.

I am thus led to consider the power of different media to conduct sound, not as an absolute and unchangeable quality, but as dependent on their relation, in point of elasticity of their molecules, to the substance from which they receive the sonorous vibrations.

The reflection of sound has relation to the same qualities of substances, but in a converse way. When, for example, a sound is produced in a very rare medium, such as air, the force with which the vibrations are propagated from particle to particle, is weak, because the molecular elasticity is weak, and being, therefore incapable of communicating its vibrations to any hard, dense, and incompressible solid with which it may be in contact, the resisted impulse is reflected

back to the air itself; and this more perfectly, the greater the difference in density and elasticity between the air and the solid body. The laws of the reflection of sound are nearly the same as those of the reflection of light; the angle of reflection being equal to the angle of incidence; and this analogy greatly facilitates our study. I must observe, however, that the analogy is not perfect in observation; for the greater materiality of the media of the vibrations of sound exposes them to a greater number of disturbing influences, which impair or disguise the operation of the law. Thus, from motion, difference of density, &c., sound seems often to be propagated through air in curves, instead of in straight lines; and from there being often refraction as well as reflection, where there is diversity of matter, sound is more easily diffused than light. The dispersion of sound through air depends in great measure on the condensation of the pulses causing a lateral displacement or aberration from the direct line of the original impulse, which constitutes a true case of refraction of sound. In confined tubes this aberration is prevented, and consequently sound may be conducted through air in them to considerable distances with very little loss.

CHAPTER II.

ON THE PHYSICAL EXAMINATION OF THE CHEST.

It has been well remarked, that no parts of the body require the assistance of an additional sense to discover their state so much as those contained in the thorax. Excluding equally with other parts the scrutiny of vision, and by reason of their bony case more than they beyond the reach of tact, the thoracic viscera would have remained in more than the common obscurity and uncertainty of signs produced by equivocal and inexplicable sympathies, and still more fallacious sensations, had not the sagacious discoverers of auscultation and percussion pointed out the peculiar adaptation of the chest to afford to our organs of hearing more certain indications of the state of its contents. And so effectually is the lacuna filled by the exercise of a sense that may be said to have been hitherto useless in the physical investigation of disease, that the diseases of the chest may now be ranked among those most within our powers of examination. For, unlike some others (the brain for example), the lungs and heart have no such complexity of structure, or obscurity of function, as to render signs of their physical state of little avail to explain their disorder, or to suggest means for their cure. We see in these organs a mechanism of structure admirably adapted for its known office; we know that the perfect state of this mechanism is necessary to preserve the integrity of the function; and we can perceive, when that becomes deranged, how this must necessarily suffer. The signs by which such derangements are commonly distinguished, arise not so much from the diseased part itself, as from the disorder which it may produce in the functions and

sensations. Now, as it is impossible to find a standard by which to judge of the health of a function in individual cases, and as sensations are frequently so elusive as to baffle our attempts to trace them to their source, the common method of diagnosis not unfrequently fails to detect even the existence of a disease; and even when the signs of disordered function and local pain are so distinct and prominent as clearly to prove that disease is present, they generally leave us in more or less doubt as to its nature. They have still their importance, and until lately have been our sole guide in the employment of a practice by no means unsuccessful. Let us not then exclude these from our view, whilst we study other signs which promise us still greater certainty; but whilst due attention is paid to general and functional symptoms, those signs which are identified with the physical nature or essence of the disease, should surely command our careful consideration.

Section I.—*Ocular and Manual Examination of the Chest.*

Much useful information as to the state of the thoracic organs may often be obtained by mere inspection and manual palpation of the chest; and although the signs resulting from this method of examination are neither so numerous nor so precise as those of auscultation, they are yet very valuable, because they are easily obtained, and because they often give a general and decisive character to other more minute indications.

In a healthy and well acting chest, the act of inspiration is performed equally by the elevation of the ribs and the descent of the diaphragm; and the eyes watching the naked chest, or the hands feeling it, will perceive its equal and uniform expansion by these means. If any disorder impede the

free descent of the diaphragm, whether it be pain of this muscle or its coverings, or of some of the viscera below, or whether it be pressure on its under surface, as from abdominal dropsy, tumours, or pregnancy, an increased task will then devolve on the ribs and their muscles, and the respiration being performed principally by the heaving of the chest, is called *thoracic*. This character of respiration is obvious to the eye: and one hand applied lightly, but in close contact, on the chest, and the other on the abdomen, equally perceive it: it becomes then the next matter of inquiry, which of the above mentioned causes is present. Again, in the converse case, which belongs more closely to our subject, pain or increased sensibility of the parietes of the chest, or of its more superficial contents, ossification of the cartilages of the ribs, and occasionally certain changes in the lungs themselves, which will be afterwards considered, make the respiration *diaphragmatic*, or *abdominal*, the ribs remaining comparatively immobile. The diseases of the chest which may render the respiration chiefly abdominal, are pleurodyne, inflammation of the costal and upper pulmonary pleura, and occasionally induration of the lung by hepatisation or tubercles. Of those which render the respiration chiefly thoracic, besides diaphragmatic pleurisy, spasmodic asthma may be reckoned, in which the diaphragm, overcome by the superior force of the bronchial muscles spasmodically contracted, is permanently drawn into the chest, causing a remarkable hollow at the scrobiculus cordis, whilst the respiration is carried on by the intercostal muscles, and others which assist them in supplemental respiratory efforts.

The more useful indications, however, in the external examination of the chest, are those which arise from a want of correspondence, in form or in motion, between the two sides. Any disease which interferes with the respiratory act, and affecting chiefly one side, will produce an inequality

obvious to sight and feeling. For this mode of examination, the patient should be placed with his chest exposed in a good light opposite to the observer, who attentively surveys the chest, and further corrects his estimate of its form and motions, by feeling with his two hands the simultaneous motions of corresponding parts on the two sides, during increased as well as ordinary acts of respiration. To determine the comparative size of the two sides of the chest, the more accurate method of measurement from the spinous process of a vertebra to the sternum may be sometimes employed. Care must be taken that the string or tape be passed over corresponding parts; and it must be held in recollection, that in healthy persons the right side is almost always slightly larger than the left.

Now it is obvious, that any inequality or irregularity in the form or movement of the chest will imply some disease; and, with a little further attention, the same method of examination will give some general knowledge of its seat and character. Thus, if one side appears to be immobile, with a sharp pain or stitch, but without alteration of size, it may be suspected that a pleurodyne or a recent pleurisy is the disease, and prevents the respiratory movements by the pain which it would cause in the affected part. If to the immobility of the side is joined an unnatural fulness, perceptible to the eye and to mensuration, there is perhaps effusion into the pleura, as in advanced states of pleurisy, empyema, hydrothorax, and pneumothorax. If a contraction of the side is joined to the defect of movement, adhesions or the reabsorption of a pleuritic effusion of some standing, is the probable cause. If one side does not partake in the respiratory act, and yet there is neither pain nor alteration of size, it is likely that the corresponding lungs may be hepatised, or otherwise obstructed.

The preceding signs are connected principally with the

middle and lower parts of the chest. Irregularities in the movements and shape of the upper regions are perhaps more characteristic. Thus tubercular disease rarely exists to a considerable extent without diminishing the motion of the upper ribs of the affected side; and adhering, as the diseased lung often is, to the walls of the chest, it not unfrequently causes an angularity and want of symmetry, which are very characteristic. Opposed to this is the effect of pulmonary emphysema, which gives a full and unnaturally rounded appearance to the upper parts of the chest, whilst, both by sight and tact, it can be perceived that the chest does not rise and fall to its natural capacity.

It is sufficiently apparent that this class of signs is too vague and equivocal to be exclusively depended on in the formation of a diagnosis; but as a general guide to the more accurate but difficult test of auscultation, or as a confirming summary of its results, ocular and manual examination of the chest is often highly instructive and useful. Besides in the manner just described, the fingers may with advantage be used to feel the conditions of the parietes of the chest, and especially of the intercostal spaces which are modified by the presence of liquid or air in the pleura, to feel the pulsations of the heart, or of aneurisms of the great arteries, and, in short, to appreciate any change on the surface of the chest which may result from the physical condition of the interior. The deformities of the chest produced by curvatures of the spine, or by improper pressure by the nurse's hands on the ribs during infancy, may be detected by the external examination of the chest, and should of course be carefully distinguished from disease of the interior. The effects of tight lacing in females become also obvious to the eye, in the permanently contracted and less mobile state of the lower parts of the chest, and in the increased heaving of the upper ribs.

Section II.—*On Percussion.*

There are two classes of sounds from which a knowledge may be obtained of the state of the thoracic viscera. One description or class is, for the most part, naturally produced by the motions of the organs within the chest, and is heard by the direct or mediate application of the ear to its parietes. These are the signs of *auscultation.* The other class of sounds is produced artificially by striking the chest; these constitute the signs of *percussion.* These last I shall first notice, not that they are prior in importance, but because they are more simple, and are generally consulted in examination before the fuller and more satisfactory ones of auscultation.

The chest of a person in health yields, when struck lightly by the ends of the fingers, a hollow and somewhat drum-like sound. The resonance thus produced arises from the elastic air contained within, in the spongy tissue of the lungs, which gives a freedom of vibration to the thoracic parietes. But in order that the vibration be produced, these parietes must possess a certain degree of elastic tension; for if they are flaccid, and yield to the stroke of percussion, no sound will be emitted but that slight and obtuse one produced by the fall of the fingers upon the surface. The natural compact of the chest, with its frame of bone, attached by elastic ligaments and cartilages, and invested by a covering more or less tense, of muscles and integuments, is generally well adapted to transmit to its interior the impulse of external percussion: but if the elasticity of the cartilages be in any way lost, or if the integuments become thickened by œdema, fat, or other cause, the resonance on percussion will be proportionately diminished; and these causes of modification of the pectoral

sound must be carefully separated from those depending on the state of the internal organs.

In the natural and healthy state, as the clearness and fulness of the pectoral resonance on percussion depends on the air-filled structure of the lung, and the tenuity and tension of the containing parietes, it is evident that those parts of the chest will sound best that most completely present these conditions. Our anatomical knowledge will therefore point out the different degrees of sound that the different parts of a healthy chest should emit. Thus, the anterior and axillary parts of the chest should sound well; but in most of the posterior regions the thickness of the soft parietes must render the sound more dull, and the same effect may be produced in the inferior parts by the contiguity of the abdominal viscera. For a more specific detail of the natural sound of each part of the chest, I refer to the table of the regions at the end of this book, with its accompanying plate, which it is hoped will assist the student more readily than a detailed description. The names of the regions differ but little from those given by Laennec.*

The manner in which diseases modify the pectoral resonance, is by changing the density of the contained organs.† If,

* Since the appearance of this table and that of the physical signs, in the first edition of this work, similar ones have been published on an enlarged scale, in the form of separate charts, by Drs. Townsend, Hawley, and others. My reasons for not enlarging my tables after these examples are, that I consider them merely as aids to the memory, and that when, by comprehending details, they profess to supersede a treatise, tabular views lose the advantage of being intelligible at a *coup d'œil*, and become no more than a book in an inconvenient form.

† The true explanation of the physical principle of percussion, is not, as it has been hitherto given, the mere throwing the air or other body under the part struck, into vibration; but it is the body struck which vibrates, and derives the character of its vibrations from the density of the matter under it: thus, if this matter is air, the vibrations are unresisted, free, and the sound more or less deep and prolonged: if the matter is a rigid solid, the vibrations are abruptly returned and quick, and the sound is higher, giving the impression of hardness: if the matter is liquid, or a soft solid, the vibrations are destroyed or absorbed, and the sound is merely a sort of short dead *tap*.

for example, a liquid or solid effusion take place in any part of the lungs or pleura, the corresponding portion of the chest will yield a dull, dead sound, and without that hollow resonance which is naturally produced by elastic air underneath. On the other hand, when the aeriform contents of the cavity are increased beyond their usual proportion, as in pneumothorax and emphysema, the natural resonance may be increased to a degree that sounds quite tympanitic.

The practice of percussion requires some manual dexterity; and as on this, in great measure, depends the correctness of its indications, I shall bestow a few observations on the best method of percussion. It is of very little consequence whether the patient be sitting, or standing, or sitting up in bed, provided we hold in mind that all the sounds, bad and good, are rendered somewhat duller in the latter case, by the vicinity of the pillows and bed-clothes, which destroy the resonant echo accompanying sounds in more empty rooms. The same amount of difference may be perceived in different rooms, when percussion is practised in the standing or sitting posture. In some cases of debility, and of painful disease, the patient can bear no other than the recumbent posture; and in the parts where percussion can be practised, the sounds are somewhat more dull in these cases, from the deadening effect which the bed has on them. Thus warned, a little practice will enable the student to avoid error from these causes.

Percussion may be employed immediately on the chest, or mediately on some solid body firmly applied to it. Immediate percussion, which was the only mode practised by Avenbrugger and Laennec, is generally performed with the three first fingers of the right hand closely applied to each other, and so bent, that they may fall perpendicularly and simultaneously on the surface to be struck. To render the stroke of percussion more equal, and to prevent it from producing pain, the patient should retain on the chest a linen or cotton cover-

ing, which must be kept tight and smooth on the points of examination by the fingers of the left hand. This precaution, likewise, by giving a greater firmness to the surface, makes it yield a more distinct sound.

But all these advantages, as well as a greater facility of applying it, pertain more completely to mediate percussion, which for the last five or six years I have generally adopted in preference to the other method. M. Piorry has the merit of having first recommended the use of a pleximeter or percussion plate, which is a disk of ivory or wood, a line in thickness, and about two inches in diameter, with a raised rim, or two little projections, by means of which, it can be pressed closely to the chest by the left hand, whilst percussion is practised on it by one or more fingers of the right hand. The resonance of the internal parts can be elicited much more readily and distinctly through this plate, than from the unprotected surface; and by its aid, the mammary regions in women, and many parts of the back, which give no indications on immediate percussion, yield a tolerably clear sound, which proceeds from the deep seated aereal contents, and which is rendered dull by any considerable increase of solid or liquid in the interior. This method can likewise be used in other cases, where direct percussion is inadmissible or ineffectual, on account of great tenderness, ædema, obesity, or flaccidity of the thoracic parietes. To avoid multiplying apparatus, I formerly recommended the ivory ear piece of the stethoscope as a percussion plate, which being made thin, and lined with soft leather, to prevent the clacking noise produced by the fall of the fingers on its hard surface, will be found to answer very well in common cases. Held by means of its raised rim, with its outer side in close apposition to the chest, it presents its inner surface, covered with leather, for percussion. The most delicate pleximeters, however, are those made expressly for the purpose, without the central

perforation; and I prefer a thin disk of ivory about an inch in diameter, covered on one side with leather, and having on the same side two little projections by which the fingers may press it firmly to the point of examination. The small size of this instrument makes it capable of adaptation to the unequal surface of thin chests, and it is peculiarly advantageous in discovering inequalities of resonance of small extent, such as those arising from tubercles, pulmonary apoplexy, and lobular pneumonia, and in exactly defining the size of the heart.

There is, however, a kind of mediate percussion requiring the aid of no instrument, yet so easy in its application, and accurate in its results, that it has generally superseded pleximeters on all common occasions. I allude to the use of the fingers of the left hand as a pleximeter. If one or more of these, with the back outwards, be applied to any part of the chest, percussion may be practised on them, without annoyance to the patient, and with the effect of eliciting a much louder sound than can be obtained by direct percussion. The pliancy and capability of the fingers, by which they can singly or collectively be made to fit any inequality in the surface of the chest,—single fingers being used where delicacy is required, and all four where only a general survey of the chest is wanted,—render this method so much more prompt and *handy*, that I have no doubt of its general adoption, to the exclusion of other modes.

The judicious auscultator will, however, soon find the advantage of availing himself of each mode in particular circumstances. Thus, on the clavicles, and on the exposed parts of the ribs in thin persons, direct percussion may still claim a preference, whilst in the cases of doubtful resonance before named, the pleximeter will afford more delicate indications. Again, in a general survey of the chest, the application of the flat hand to the chest, both in the way of gentle percussion,

and also of feeling the expansibility of the different portions during respiration, will frequently aid us considerably in estimating the state of the internal organs. With those accustomed to the process, percussion on a healthy hollow chest, gives to the fingers a peculiar vibratory sensation, different from the dead inelastic feel over a part of the chest destitute of air; and this distinction, although scarcely perceptible to the unhabituated, is not without its value to those who by experience learn the advantage of availing themselves of all their senses in the investigation of disease.

A person commencing the practice of percussion, will be guided more safely by the *comparative* than by the *absolute* sounds of different parts of the chest, and although he should lose no opportunity of acquainting himself with the sounds both of percussion and auscultation in healthy subjects, he should in case of disease, more particularly at first, direct his attention to irregularities, or want of correspondence of the two sides in the same subject. In instituting this comparison, he should be careful likewise to practise percussion on corresponding parts of the two sides, and with such an attention to the manner in which his fingers fall, and, if he uses the digital pleximeter, the manner in which this is placed, that any difference of sound may not arise from these fortuitous circumstances. In cases requiring much nicety, it is also necessary to attend to the period of the respiratory act, for most parts of the chest are considerably less sonorous after a full expiration, than when the lungs are more distended with air. Thus the space between the cartilages of the fifth and seventh left ribs, in a healthy well proportioned subject, is perfectly resonant after a full inspiration, but may be made to sound quite dull by expelling freely the air from the lungs. This circumstance, which depends on the margins of the lungs, when distended, overlapping and covering the heart, should be espe-

cially attended to in examining, by means of percussion, the dimensions of this organ.*

Such is the mode of obtaining signs of the physical state of the contents of the chest by percussion. The indications thus obtained, although they only relate to the density of the parts,† are of great value, and alone may sometimes detect disease that all other signs leave in obscurity. But their importance and value are vastly enhanced when they are combined with, and corrected by, the more numerous and precise signs discovered by auscultation; these we now proceed to consider.

Section III.—*On Auscultation.*

The signs of auscultation are those sounds produced in the chest, which may be heard by the direct or mediate application of the ear to its parietes. Now, I shall endeavour (and the same will be my object throughout this little work) to trace these signs to their physical causes, and by thus exploring the relations of diseases to certain and unchanging laws of natural philosophy, to place their characters beyond the doubtfulness and obscurity of sympathetic and sensatory signs.

I have before remarked that the sounds heard by auscultation are, for the most part, produced by the natural movements of the organs contained within the chest. These movements are, those of respiration, to which may be added the voice; and those of the heart. Let us consider the

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* The student will, it is hoped, find the tables and plate, at the end of the work, of use in introducing him to the more obvious variations of pectoral resonance.

† A dulness over certain cavities in phthisis forms an exception; for as it has been remarked to me by Dr. Chambers, this is not caused solely by condensation surrounding the excavations, but probably also by the lax and flabby remains of the lung acting as a damper on the sonorous vibrations.

manner in which these several motions give rise to sound, and we shall then be enabled to perceive *à priori* the modifications in it that disease may produce.

Sounds of Respiration.

The ingress and egress of air in the lungs cause a sound of a peculiar nature, differing somewhat according to the part in which it is heard. This difference arises from a diversity in the size of the tubes, through which the air passes; and from a knowledge of the distribution of these tubes, we may, therefore, easily judge what these differences ought to be. Between the scapulæ, for example, in the upper part of the axillæ and in the upper sternal region, the sound is hollower, more tubular and blowing, because in these regions many bronchial ramifications of considerable size come so near the surface of the lung, that the sound produced by the passage of air through them, is heard more distinctly than the duller and more diffused murmur which has its seat in the smaller bronchi and air cells. It is this latter sound, on the other hand, that prevails in other parts of the chest; for although there is bronchial respiration in these parts likewise, it is not sufficiently near to the surface to be transmitted through the spongy and ill-conducting tissue of the lung.

It is of great importance to be able to distinguish between the sounds which the passage of the air produces in the trachea, in the larger bronchi, and in the extreme bronchi and vesicles; which different kinds of respiratory sound, we shall, with Laennec and Andral, distinguish by the epithets *tracheal*, *bronchial*, and *vesicular*. Rather than attempt to convey an idea of these sounds by description, I will refer for the illustration of *tracheal respiration*, to the anterior and lateral parts of the neck, the superior sternal region, the sternal portion of the infra-clavian regions, and the cervical portion of the acromian regions; of *bronchial respiration*, to

the middle portion of the sternum and those parts of the mammary regions contiguous to it, to the principal part of the interscapular and axillary, and in thin subjects to the inner and lower parts of the scapular, regions; and of *vesicular respiration* to the remaining parts of the chest. Such are generally the situations of the different kinds of respiratory sound; but, as might be expected, the distinction may be much more easily made in some subjects than in others, and the characters can in a corresponding degree be severally recognized.

There is a considerable difference in the intensity of the sound of respiration in different individuals; and this depends partly on the thickness of the parietes of the chest, but principally on the degree of activity of the respiratory function. Increased thickness of the parietes of the chest, by fat or œdema, does not very materially impede the transmission of sound of respiration to the ear, for being nearly of equal density, they form still a pretty good conductor of sound. Nevertheless, the respiratory murmur is most distinctly heard in those parts of the chest where the parietes are thinnest.

The degree of activity in the function in a remarkable manner determines the intensity of the respiratory sound; and the variety which different individuals in this respect present, even in health, is a matter of much physiological interest. We know that in like manner other secernent functions, as those of the kidneys and skin, vary in different individuals, under the same circumstances, in the degrees of their activity, and we may range the present instance amongst them. Were we to inquire still further into the causes of these differences, we should probably be led to conclude that they all have relation to a certain standard of organic activity or irritability, in some manner dependent on the physical constitution of the body. From this obscure point of constitutional difference

(on which we can scarcely do more than generalize), let us turn to the changes in the respiratory sound that may take place in the *same individual* within the bounds of health.

I have remarked that it is more distinct after meals than at other times, which fact accords well with an ascertained point in animal chemistry; and proving that a greater activity of function is at that time required, it likewise furnishes an additional reason why persons affected with habitual dyspnœa, should then most feel the incapacity of their organs. Moderate exercise likewise increases the respiratory sound; but violent exertion has a tendency to produce an opposite effect: for, when the muscles of respiration are exerted beyond a certain degree of activity, the inflation and collapse of the lungs cannot always keep pace with them, and the violence done to the lungs by the effort, together with the load of blood thrown into their vessels by the motion of the various limbs in action, throws the bronchial muscles into a state of irregular spasmodic contraction, which is only gradually relieved by the returning moderation and regularity of the respiratory effort. This is much more remarkable in persons unaccustomed to exertion, and advanced in life, than in the young and active; and I think we should not use too mechanical a term, if we say that this proceeds from the greater *rigidity* of the lungs in the former. This leads me to the remarkable peculiarity that the respiration of very young individuals presents to the auscultator.

From birth till about the period of puberty, the sound of respiration is much louder and more shrill, than in after life; the passage of the air, producing it, seems much quicker, and the function appears to be in an extreme of activity. That the sound of *puerile respiration* (as Laennec terms this modification) proceeds from no peculiarity in the structure of the lungs of children, is proved by the fact that it is occasionally produced in adults, when one part of the lung is called into

increased activity, to supply the defect of another, incapacitated by disease.* So also in adults, after a temporary suspension of respiration, as in reading or continued utterance, the respirations are often attended with a puerile sound; for being more rare, they are made with greater perfection and energy than usual. About the age of puberty, the sound of respiration becomes deeper and less noisy, and in a few years, sooner or later, gradually assumes the character of adult respiration.

This change I am disposed to attribute principally to a greater comparative development of the lungs at that age, rather than to a diminished activity of their function. It is at this period that the muscular system developes itself more fully, and to support the occasional exertion of its augmented power, the organs of the chest acquire an increased capacity, and a more extended sphere of activity. The pulse and respiration becoming slower in the standard of rest, offer a greater range in their dynamometric scale; and although, from increased capacity, their common activity is diminished, they have greater capabilities in reserve to support the occasional exertions of increased muscular strength. We find, accordingly, that in young persons above the age of puberty, increased exertion renders the respiration puerile, (that is, more active), and is therefore easy; and as long as this supplementary power of the lungs is moderately exercised, by occasional increased muscular exertion, it will be preserved; but, on the other hand, it will be lost by disuse; the organs will become rigid in their limited sphere of action; occasional exertion will be attended with the anhelation and spasmodic action of the bronchi before alluded to; and the attacks of disease, on a function that can scarcely bear abridgment,

* I have repeatedly found that tight-laced stays in females render the respiration puerile under the clavicles; the reason of this is sufficiently apparent in what has been stated before. See page 15.

must be felt with a greater degree of severity:—an addition to the volume of arguments in favour of regular and active exercise.

As the sound of vesicular respiration is produced by the perfect penetration of the air into the lungs, its simple and equal presence may be regarded as a proof of the healthy performance of the function; and, as no physical change can interrupt or modify this function without interrupting and modifying the sound, the study of these latter changes will lead us to a knowledge of the physical changes that produce them.

The total absence of respiratory sound in a part, indicates that the air no longer penetrates there, either because something excludes its entry into the pulmonary tissue, or because this tissue is pushed away from the parietes of the chest by an effusion into the pleura. An appeal to the signs of percussion is here necessary to ascertain in what manner the lung is invaded or surrounded. If the sound on percussion remains natural, the obstruction is probably situated in some of the bronchial branches leading to the part, whilst the vesicular texture contains its due quantity of air; if it is dull, there is liquid or solid effusion, either in the vesicular structure of the lung, or in the intervening pleural space; but if it is clearer than usual, there is either emphysema of the lung, or an aeriform effusion in the pleura. The inquiry, thus simplified, can now be specifically directed to the distinctive characters of individual diseases, presenting either of these physical conditions.

Sometimes the sound of natural vesicular respiration is absent, and a kind of hissing *bronchial respiration* is heard instead. Now, as the sound of the passage of the air in the bronchial tubes cannot be usually heard through the spongy and ill-conducting vesicular texture, it must be supposed that either the bronchial respiration is louder than usual in these cases, or, that the tissue of the lung is, by some change, ren-

dered a better conductor of sound. But increased loudness of the bronchial respiration would not explain the absence of the sound of the vesicular. Abandoning, therefore, this alternative, we shall find in the other an explanation of both modifications; for a liquid or solid effusion, at the same time that it obstructs the entry of air into the cells, likewise so condenses the tissue as to enable it to transmit, from its interior, sounds that are not usually heard.

A sound, resembling that of tracheal respiration, is sometimes induced by disease, in parts where vesicular respiration alone is naturally heard; and this phenomenon is caused by the passage of the air in a cavern, or ulcerated cavity, communicating with the bronchi. The sound thus produced, which is called the *cavernous respiration,* is so remarkable, and so like the blowing of air into any little hollow object, that the mind would at once, and, as it were instinctively, refer it to its true cause. The sound is often louder than tracheal respiration, and owes its existence to the resistance opposed to the current of air by the walls of the cavity, whilst its tone and character depend on the size and form. When these are considerable, with simple and tense walls, every sound produced in, or communicated to, the cavity, will be attended with a resonant echo, the tone of which depends on the relation of the diameter of the cavity to the velocity of sound in air; and thus arise the phenomena of *amphoric respiration* and *metallic tinkling,* which will be noticed hereafter.

There is a remarkable class of sounds produced by partial obstructions to the passage of the air through the bronchial ramifications. These sounds, which are called *rhonchi,** may

* I originally preferred the Latin term *rhonchus* (Gr. ῥόγχος) to the French *râle,* and the English *rattle,* because it expresses the subject as well, and is more accordant with the usual style of medical language. It has since been adopted by Dr. Forbes, Dr. Copland, and other of our best writers. Many, however, espe-

be divided into the *dry* and *humid*. The dry rhonchi are those sounds produced by the passage of the air through bronchi, which have some part of their calibre contracted by a substance more or less solid. This contraction may be produced by a partial tumefaction of the membranes of a bronchus; by the pressure of an adjacent tumour; or by some body, such as a portion of thick mucus, within its tube; and the form and size of the isthmus, or contracted point, will determine the nature of the sound. Thus, we often hear an acute whistling sound, which is therefore called the *sibilant*, or *sibilous* rhonchus; and, as we know that such a sound may be produced by air passing through a small circular aperture,* it may be supposed that a contraction of this kind causes it in this case.

The *sonorous rhonchus*, which sometimes resembles snoring,

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cially among our Irish brethren, still retain the French word, the introduction of which would add to those anomalies of pronunciation, which so much injure the simplicity of our language. It would be easy, by reference to quotations, to show that the word râle is familiarly used by the French in a sense different from that to which Laennec applied it; and in conventionally extending its signification, he did not infringe on the purity of his language; but the adoption of the same word into the English tongue, which has its own proper sources of derivation, and rules of pronunciation, is obviously objectionable. The term which I have selected was always used by Laennec in his Latin conversations; and either it may be retained with its Latin termination, so as to correspond with its fit companion *bronchus*; or it may be *anglicised* into *rhonch*, after the analogy of *conch*. If an original English term must be used, the word *wheeze*, proposed by the editor of the Medico-Chirurgical Review, is the least exceptionable. To that editor the profession is indebted for his having with a truly candid and philosophical spirit, at an early period recognized and proclaimed the advantages of auscultation.

* It must be observed, that to produce a whistling sound by the passage of air through a round aperture, there must be a certain proportion between the velocity of the air and the size of the aperture. I must beg again to be indulged in a philological note. Dr. Forbes, and some other writers, use the words *sibilous* and *crepitous*, in preference to *sibilant* and *crepitant*, which I have adopted. But I must plead precedent and analogy to be in my favour. *Sibilant* is already an English word: *sibilous* is not. *Crepitant* is derived directly from the Latin *crepitans*: there is no Latin adjective corresponding with *crepitous*. Again, analogy suggests generally, that epithets implying action, should be expressed by the active participle ending in *nt*, whilst those representing passive or possessive qualities, are more commonly derived from nouns, and terminate in *ous*.

sometimes the buzzing of an insect, sometimes the bass note of a violoncello or bassoon, is rather produced by a flattened contraction in a bronchus of considerable size. This contraction, which leaves little or no gaping aperture, throws the air passing through it into sonorous vibrations, after the manner of the reed of the hautboy, or the lips in blowing a horn or trumpet; or, perhaps, the production of this sound is still more completely represented by the manner in which a celebrated ventriloquist imitates, with his lips, the buzzing of a fly.

The *dry mucous rhonchus* may be said to be a coarse modification of the preceding. It resembles the sound of a click wheel; and is produced by a portion of very viscid mucus attached to the interior of a bronchial tube, which yielding with a jerking resistance to the air forcing its passage, thereby causes a ticking sound. Such is, in fact, the analysis of the sonorous rhonchus; for it only requires that the air should pass more speedily, and the tickings be multiplied until they seem continuous, to convert the dry mucous into the sonorous rhonchus.

The *humid rhonchi* arise from the presence of fluids in the bronchial tubes. The commonest, and the most obvious in its nature, is the *mucous* or *bubbling rhonchus*, a sound which the mind at once refers to the passage of air in bubbles through a liquid. It is more gurgling, coarse, and irregular, when situated in bronchial tubes of large size, because the bubbles are large and unequal. In the smaller order of bronchial tubes, on the other hand, it is more equal and minute. So we can perceive at once what kind of sound this rhonchus ought to have when in the trachea, or in a cavity produced by disease. It is coarse and gurgling, in proportion to the size of the tube or cavity in which it is produced, and the freedom with which the air passes through the liquid; and, when presenting these conditions, the *cavernous rhonchus, gargouille-*

ment, gurgling or mucous rhonchus of caverns, is one of the most remarkable and important signs discovered by auscultation. It would seem unnecessary to inquire farther into the physical nature of the mucous rhonchus, were it not that the inquiry may enable us to distinguish some of its varieties from another rhonchus, which distinction is an object of considerable importance.

The sound of the mucous rhonchus depends on the bursting of successive bubbles of air which pass through a liquid. A bubble is a portion of air contained by a thin film of liquid, which preserves its continuity by virtue of its attraction of aggregation; and the bursting of this bubble is the overcoming of the resistance of this power by some other, so that the air escapes. At the moment of its escape by the bursting of the film, its slight expansion communicates to the body of air, of which it is now become a part, an impulse which, if sufficiently forcible, produces a sound. Now this impulse will be forcible in proportion to the resistance offered by the film of liquid at the moment of its rupture, and will therefore be greatest when the bursting force is applied so quickly and suddenly that it meets with the full resistance of the newly formed film, undiminished by the extenuating power of gravitation.

Now the bubbles of the mucous rhonchus are both formed and burst by the respiratory movements driving the air through the liquid in the bronchial canals; and they will therefore produce most sound in those bronchi through which the air passes most quickly. These are, of course, the bronchi of larger order. If the liquid be thin and watery, the bubbles pass, and burst in quick succession, with an irregular and more or less gurgling sound; but if it be viscid, they are fewer in number, and may be carried on in the tube some way before they burst; and the sound is therefore diffused, more

regular, and rare. The quantity of liquid present in the bronchi may in some measure be estimated by the continuance of the rhonchus. If this accompany only the first part of inspiration and the end of expiration, the liquid must be scanty, for it only interferes with the air when the tubes are in their contracted state: But if the whole of the respiratory act, even to the acme of inspiration, is attended with the bubbling sound, then it must be apprehended that the quantity of liquid is considerable, and extends to the small bronchi.

A little liquid in the smaller bronchial tubes produces the *submucous rhonchus,* a kind in which the bubbles are fine and more crepitant; but they often intermit, and in a full breath are diminished to a slight roughness, accompanying the respiratory murmur.

The rhonchi of which I have yet spoken, with the exception of the submucous, are generally produced in bronchial ramifications, of above or about the size of a crow's quill; it is in those below these, or perhaps in the necks of the bunches of vesicles themselves, that the *crepitant* or *crepitous rhonchus* has its seat. This is likewise a bubbling rhonchus; but it is physically and pathologically different from all the others. The sound is a gentle crepitation, uniform and continuing to the end of inspiration. It is compared by Laennec to the crepitation of salt by heat, and the resemblance is pretty exact when common grain salt of commerce is thrown on a heated iron. It may also be tolerably represented by rubbing tranversely, between the fingers and thumb, a lock of one's own hair close to the ear, or by squeezing a portion of healthy lung. It is probable that in the cases in which the crepitating rhonchus is present, the calibre of the last bronchial divisions is so much diminished (by the interstitial effusion) that the air cannot pass through them, without raising the mucus, more or less viscid, into bubbles; which, being

uniformly small, and bursting regularly, produce a continued succession of minute crepitations.* The more viscid the mucus, the more distinct is the crepitant character of the rhonchus. It is perfectly so in the crepitant rhonchus of recent pneumonia. In pulmonary apoplexy and œdema, on the other hand, the liquid of the bronchi is thinner, and the rhonchus being less perfect in its crepitation, is accordingly called *subcrepitant*.

Thus the rhonchi give positive and direct indications of the state of the bronchial tubes; nor can these suffer materially without either altered secretion or change in calibre producing one or other of these rhonchi. Respiration thus modified is generally more noisy than when free, and a rhonchus, particularly the sibilant or sonorous, may often be heard through the spongy texture of a whole lung. This does not, however, prevent the natural respiration of the healthy parts from being heard: for as long as two sounds differ in nature, the louder will not drown the weaker, unless the disproportion be great. For example, the sound of respiration may be heard, although a much louder sibilant rhonchus accompany it: and a deep seated crepitant rhonchus can often be distinguished through the respiratory murmur immediately below the stethoscope. We frequently hear a mixture of several rhonchi, occupying adjacent tubes; and it sometimes requires considerable attention to separate and recognize them.

The modification of respiration which constitutes cough, may often be consulted with advantage when the sounds produced in the ordinary respiratory act are doubtful or indistinct. Cough consists of a sudden and forcible expiration, succeeded by a deep but quick inspiration. Now as in this case the passage of the air is more forcible and perfect, the sounds produced by it must likewise be rendered more dis-

* We must not omit to notice that another view has been commonly given of this symptom. This subject will be considered in the section on Pneumonia.

tinct than in common respiration; and not unfrequently a cough may force air through bronchi too much obstructed to admit it by the common respiratory effort. The cough may, of course, be modified in the same way as common respiration; and may, therefore, be accompanied by the sibilant, sonorous, crepitant and mucous rhonchi. It discovers the existence of caverns more unequivocally than common respiration does, whether the caverns be empty, and yield only the hollow resonance of air blowing in them, or whether, containing a liquid, they are the seat of the gurgling cavernous rhonchus.

Sounds of the Voice.

The voice is another source of signs by which the auscultator may judge of the state of the lungs. The sound of the voice, although produced in the larynx, is propagated to the air in the trachea and bronchial tubes*, as outwardly it is communicated to that in the mouth and beyond it. Accordingly, if we listen with the stethoscope applied to the trachea or upper part of the sternum, we hear the voice through the instrument, and louder than by the other ear, inasmuch as the voice is outwardly diffused in a large space, but there is confined within a narrow tube: this is called *laryngophony*.

* I do not at all concur in M. Laennec's opinion, that the bronchial tree is a part of the instrument that originates the sound of the voice; for if it were so, disease would affect the voice in a very different manner from what we find to be the case. The hepatization of a lung, or its compression by a pleuritic effusion, should in that case raise its tone to a treble. I have often known them to exist without changing it at all. It is true that large ulcerous excavations do sometimes render the voice deep and hollow; but this is because the want of breath prevents the patient from contracting the glottis sufficiently to produce more acute tones. To receive as much air as possible to fill the healthy cells, as well as the vast excavations in which it is wasted, the glottis either contracts only enough to produce a low bass note, or does not contract at all, and the patient then speaks in a whisper. I conceive that the trachea and bronchi, besides supplying the air for the production of the voice in the larynx, act something after the manner of the resonant cavity in the sounding board of musical instruments, reverberating and giving fulness to the voice, but not essentially producing or changing its note.

If the stethoscope be applied to those parts of the chest under which pass bronchial tubes of considerable size, the voice will be heard there likewise, but it is not so loud, and its articulation is less distinct: this is *natural bronchophony*. Over smaller branches, the articulation is still further confused, and the voice is only heard in a diffused resonance. In the vesicular structure they are both lost, and over this a slight fremitus, which the voice produces throughout the chest, can alone be heard. The vocal resonance does not extend to the smaller bronchi, because they do not afford sufficient space for its vibrations; and also, because their less tense and more membranous tunics yield and destroy the sonorous impulse.

The vesicular texture is, as we have already seen, a very bad conductor of sound; hence it prevents the vocal resonance in the bronchi from being transmitted to the parietes of the chest; except in those parts where bronchi of some size pass close to the surface. It therefore appears that different parts of the chest will present to the auscultator some varieties of this vocal resonance, and it is important that he should be acquainted with them.

When the stethoscope is applied to the larynx or trachea, the voice seems to enter the instrument as loudly as if the speaker's mouth were applied to it. This phenomenon scarcely ever exists to its full extent in any part of the chest unless in a state of disease; and therefore it is useful to distinguish between it and the natural bronchophony, which is to be heard under and near the upper part of the sternum, in the upper part of the axilla, and in the interscapular space. In these situations, the voice is generally louder than that which, proceeding from the mouth, strikes the other ear, but the words seem to be at the end of the tube, and not as in *laryngophony*, to pass through it into the ear.* Such is the

* I consider these phenomena to exemplify the law of vibrating systems, which has been beautifully developed by M. Savart and Professor Wheatstone. When

impression, and although it arises only from a difference in the body of sound, it should be attended to, as enabling us to make an important distinction.

The degree of vocal resonance in the chest differs considerably in different individuals, and the causes of this difference are not obscure. It is loudest, and most distinct and extensive, in those persons that are thin, and have a sharp treble voice; and if these circumstances exist in a great degree, the natural bronchophony may extend to the mesial parts of the scapular, infraclavian, and mammary regions, whilst in the usual places it almost amounts to laryngophony. It is therefore remarkable in young subjects and in females. In those, on the other hand, whose chests are well clothed with muscles and fat, and whose voices are deep, the natural bronchophony is obscure and confined. The vibrations of deep notes cannot be extended to very narrow tubes, because there is not space for their play; and this explains the difference resulting from the tone of voice, and suggests that a change of tone in the same individual may considerably vary the bronchophonic resonance. In all other parts of the chest there is either no resonance, or only that slight vibratory fremitus or thrill that may likewise be felt on the application of the hand to the parietes during the exercise of the voice. This vibration, which accompanies deep tones more than others, is produced by the transmission of the sound, not through the bronchial tubes, but through the common substance of the lung, and is so slight as not to obscure other signs to any extent.

Disease may produce vocal resonance of either kind, in parts where it does not naturally exist. In degree equal to

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the local resonance is very confined or not strong, the sound is merely propagated through part of the air in the tube of the stethoscope, and the sound appears at its proper distance; but when the voice is full and strong at the end of the tube, it throws the whole column into sonorous vibration, which, constituting with the voice in the bronchial tree a vibrating system, extends the full tone of the voice to the very ear.

laryngophony, this accidental resonance is called *perfect pectoriloquy:* and when it simulates the natural resonance under the sternum, it is *imperfect pectoriloquy.*

These symptoms are produced by unnatural cavities in the substance of the lung, to which the sound of the voice is propagated through the bronchi; and their presence is a certain proof of the existence of such cavities. When the stethoscope is applied to a part of the chest under which lies one of these cavities, the words which the patient utters seem to proceed from that spot; hence the term *pectoriloquy.* The distinction between perfect and imperfect pectoriloquy is, as in the case of natural resonance, whether the voice seems to traverse the tube, or to remain at the end; and the physical difference producing the two modifications, consists in the size and situation of the cavity. The most perfect pectoriloquy is produced in cavities of moderate size, which are situated near the surface of the lung, and freely communicate with a large bronchial tube. If the cavity be deep seated, or if its communication with the bronchi be imperfect, the resonance of the voice will not amount to perfect pectoriloquy. True pectoriloquy, produced by a cavity, is generally abruptly circumscribed, so that its limits can be distinctly traced.

Pectoriloquy may be considered a certain indication of a cavity (almost always tubercular), whenever occurring in those parts of the chest where there is naturally no bronchial resonance. When it is heard in the other parts, it is more doubtful, but even there, if it be perfect, distinctly circumscribed, and heard so on one side only, it leaves very little room for doubt.

There is another way in which the vocal resonance may become a sign of disease. As we have noticed that bronchial respiration may become audible by the condensation of the intervening portion of vesicular tissue, so the same cause may transmit to the surface a bronchophony, which in the

healthy state is confined by the surrounding ill-conducting tissue. Hence inflammation, œdema, tubercular and sanguineous infiltration are often attended with an *accidental bronchophony.* Accidental bronchophony frequently differs from that existing naturally in certain parts of the chest; but of this difference, and of that called *œgophony* I shall hereafter speak.

Section IV.—*Methods of Auscultation.*

All the acoustic phenomena of the chest may be heard by the simple application of the ear to its parietes. In this *immediate* method of auscultation, the sound is communicated through the parietes of the chest to the air in the hollow of the external ear and meatus, which being excluded from the access of all other sounds, receives in unmodified intensity every vibration that emanates from the chest. This method has the advantage of being simple, expeditious, and easily acquired; but it likewise has disadvantages to be noticed hereafter, which render it less eligible than mediate auscultation with the stethoscope. We need not, however, entirely abandon immediate auscultation; but in cases requiring little nicety of examination we may often avail ourselves of the greater ease and celerity of this method, particularly in exploring the posterior parts of the chest, where the application of the stethoscope requires a somewhat tedious caution.

The *stethoscope* is an instrument invented by the late M. Laennec, to assist the ear in examining the acoustic signs of various diseases, especially those of the chest. Although its construction is simple, and its application easy, yet we shall not lose time if we give a little attention to analyze its physi-

cal office, and render intelligible the principles of its use. When we bring to the aid of our senses artificial instruments, we can neither perfect their construction, nor fully avail ourselves of their application, without a knowledge of the physical principles on which they assist our organs. No one can make a proper use of the microscope or telescope without understanding the laws of optics; and I hold that the easiest, the most agreeable, and the most certain road to a knowledge of stethoscopic phenomena, is through a study of acoustics.

In acoustic instruments we avail ourselves of the conducting power of bodies of similar density, and the reflecting quality of dissimilar substances, to direct sound into any particular channel. It is with regard to these principles in the first place, and with a certain respect to portability and convenience, that the best stethoscopes are constructed. As the sounds which they have to transmit to the ear are various in origin and intensity, so these instruments have modifications in their make, which, with a view to avoid the multiplication of apparatus, were comprised by Laennec in a single instrument of convenient portability. Thus, to convey the sounds originating in the denser contents of the chest, such as the heart, a cylinder of light but rigid wood, as free as possible from knots and inequalities, answers perfectly. To transmit the sounds produced in the more aereal contents of the chest, this cylinder is perforated longitudinally, so as to contain a column of air, which being perfectly closed from external communication by the ear at one end, and the chest at the other, readily conducts the sound of the voice, or of the respiration produced in the spot which the aperture covers. To concentrate the diffused sound of respiration, and to expedite the examination by making the stethoscope cover as large a space at a time as possible, the cylinder is hollowed out at the pectoral end into a conical cavity, the apex of which terminates in the central canal; so that all the sounds

which enter the excavated end are reflected through this canal into the ear. To reconvert this into a simply perforated cylinder for the uses above mentioned, a perforated plug or stopper is adapted, of size and form exactly filling the conical excavation.

Such are the general principles for the construction of the stethoscope; for further particulars I must refer to the plate, and its accompanying explanation. I would now say a few words on the advantages of mediate auscultation, and of the necessity of the stethoscope to those who wish to avail themselves fully of physical diagnosis in general practice. After what has been said by Laennec and others on this point, I should have hardly deemed it necessary to mention it here, were it not that several writers, otherwise advocates of auscultation, have spoken slightingly of the aid afforded by the stethoscope. Could the naked ear be in all cases applied to the chest of a patient, the utility of the stethoscope might be more reasonably questioned. But it cannot be denied that immediate auscultation is, in many circumstances, impracticable; in some cases being disgusting, in others indelicate, and in infectious disorders unsafe; whilst these objections are not applicable to the use of the stethoscope. Further, if it be said, restrict its use to these cases, we reply, that unless we have more practice with it than can be obtained in these only, the stethoscope would be comparatively useless in our hands; for its indications, although more accurate than those of the unaided ear, unquestionably require more practice to obtain them.

But there are other circumstances, which give to mediate auscultation a claim of preference; and as they illustrate the application of the instrument, I think it proper to mention them. The unaided ear, although capable of perceiving very delicately the sounds produced under the surface to which it is applied, cannot isolate any particular spot; and the sounds

transmitted by the parts adjoining the ear, the temporal zygoma, the mastoid protuberances, &c., as well as the adventitious noises liable to be produced by the friction of the hair of the head and face, are frequently mixed with, and confuse the signs from the spot under examination. On the other hand, by the stethoscope, we can separate any individual point; and by tracing the sounds to their exact position in the organs within, we can accurately determine their value as signs. This faculty is, in some cases, of great importance. It is only by its means that we can discern the limits between a natural resonance of the voice under the clavicles, between the scapulæ, and in the axillæ, and the morbid resonance which an induration of the tissue of the lung produces in parts immediately adjoining these regions. The stethoscope with the stopper, not unfrequently discovers a sound to be merely a natural resonance, which to the open instrument, or to the naked ear, has the character of morbid bronchophony; and the variations in the form of the instrument, furnish a means of distinction between the degrees of pectoriloquy, more accurate than can be obtained by the ear alone (See p. 37.) The capability of the stethoscope to separate the sound from the impulse of the heart, and the facility with which it can be applied to the infra-clavicular and axillary regions, and to the examination of sounds in the carotid arteries and abdominal aorta, which are not within the convenient reach of the unassisted ear, are further and important advantages of mediate auscultation. Although, therefore, with a view to expedition and convenience, immediate auscultation may be occasionally substituted, I am confident that no one who has once trained his ears to the use of the stethoscope, will ever so lightly esteem its aid, as again to abandon it.

A little well-regulated practice in the use of the stethoscope is worth a volume of directions and cautions. By this the

observing student will soon find how necessary it is to keep the instrument closely applied both to the chest of the patient and to his own ear, so that there be no communication between the interior of the tube and the external air; to hold it in such a manner, by the end near the chest, that no extraneous sound be communicated by friction of contiguous clothes or otherwise; to avoid pressing so hard upon it as to produce pain, or interfere with the respiratory movements; to avoid too stooping or constrained a posture, which may cause tinnitus aurium, and render hearing obtuse;* and to conduct his examination of the series of signs with as little fatigue to the patient as the case will permit.

The patient should not have over the chest more than a single garment of linen or cotton, and this should be kept smooth under the instrument. To explore the anterior and lateral regions the patient may be either seated on a chair, or lying near the edge of a bed: the examination of the back must be effected in the sitting posture, with the body bent forwards. It is always the best plan to change sides in order to examine the opposite side of the chest, and not lean across, unless it be for the comparison of corresponding points on both sides, where it is important that the two impressions should succeed quickly to each other. The attentive student will soon find how far these precautions are necessary; and to what degree tact, furnished by experience, may supersede or modify them.

It is generally expedient to follow a particular order or method in conducting a physical examination of the chest. I usually begin with inspection and feeling the chest with both hands, and then proceed to percussion, first on the clavicles,

* Another source of error is the remarkable tinkling sound produced by any strong impulse on the ear applied on the stethoscope, or still more directly on the chest. This sound, which is simply the note of the tympanum re-echoed in the external meatus, misled Laennec himself in several instances. See *Pneumothorax*.

then on the anterior parts of the chest, proceeding from above downwards; next on the lateral portions, beginning at the axilla, which is exposed by the patient raising his arm up to his head. The same parts are then examined, in like order, by the stethoscope, with due attention to the indications just obtained by percussion. For the percussion of the posterior part of the chest, the patient must sit with his head bowed forwards, and his arms crossed over his breast; and after due care in exercising percussion in this more obscure region, the easier test of auscultation may be practised.

For exploring the respiration, cough, and most of the rhonchi, the stethoscope should be used without its stopper. The signs of the voice are least equivocal, when heard with the stopper in; and to determine the extent of a cavernous or crepitant rhonchus, or of a bronchial respiration, it is often useful to resort to the instrument in this form. When the voice is examined, the patient should turn his head away from the auscultator, and speak a few consecutive sentences. For the respiration he should be desired to breathe a little quicker than usual. The heart is examined with the stopper in; but when it is wished to hear the sound of pulsation without the impulse, the stopper should be taken out.

The physical examination of the chest, when adroitly and systematically conducted, is not nearly so tedious as might be imagined, and it is surprising with what ease and expedition it may be performed, after some experience. A few minutes will, in a majority of cases, suffice to furnish us with information far more certain than can be obtained in any other way; and, in cases of obscurity and difficulty, a much longer time devoted to it should not be considered as thrown away. For in all cases time should be deemed of much lower value than a true knowledge of the disease; and I hold it to be the duty of the conscientious physician to consider this, and the employment of curative measures founded upon it, as the paramount objects of his care.

Section V.—*On Expectoration.*

The sputa in pulmonary diseases, although the signs which they give are not the directly physical effect of the lesions, yet have frequently such distinctive characters, and are often so intimately connected with the physical signs, that when consulted together with them they assist in a most essential manner in pointing out the nature of a disease. I shall, therefore, in the descriptions of the pathology and signs, refer to the character of the excretion of the bronchi, whenever expectoration presents it to our view. In this place I shall confine myself to a few remarks on the physiology and general pathology of the act and matter of expectoration.

The act of expectoration is one of the instances of combined movement in the respiratory machine, which by an admirable and harmonious consent between its numerous muscles, unerringly produces such a variety of actions. The function of respiration is of such vital importance, that accumulations or effusions that obstruct it, endanger life itself. The structure of the bronchial tree contributes greatly to the easy removal of any superfluous matter in it that might cause such obstruction, for the sum of the area of its branches being considerably greater than that of its trunk, the trachea, the air commonly finds easy entrance into the air cells, and on its more rapid return in expiration, carries with it the superfluous matter. Thus ordinary respiration tends to prevent, in spite of gravitation, any accumulation in the air tubes; but the excretion is more completely effected by coughing, and special efforts of expectoration. These consist of a quick and forcible expiration, preceded by a deep inspiration, and accompanied with a constriction of the larynx and trachea, the effect of which is to bring any superfluous matter into positions, from which the air, forcibly expired, drives it through

the glottis. It is worthy of remark that expectoration cannot effectually take place without a previous full inspiration by which air is carried beyond the accumulating matter; hence, when this is prevented, either by weakness of the respiratory powers, or by the impermeability of the bronchial tubes, the excretion is suppressed. The first of these causes of obstructed respiration is exemplified in adynamic fevers, which may thus prove fatal; the second occurs in pneumonia, in the stage of hepatisation, and if extensive, must lead to a fatal obstruction of the respiratory function. They probably occur together towards the fatal termination of bronchitis, phthisis, and other severe diseases of the lungs.

Expectoration, in its other sense, namely, the matter expectorated, is a subject well worthy of a careful study; for its characters often furnish signs of the greatest value in the diagnosis, prognosis, and treatment of diseases of the chest. It can scarcely be said that the examination of the sputa is entirely neglected in this country; but I have had frequent occasion to observe that opinions are very loosely and vaguely formed from it, and of a nature quite inconsistent with the present state of pathological science. Thus the presence of pus in the expectoration is often called a sure proof that the lungs are 'diseased;' whilst the far more pathognomonic sputa of peripneumony and the well marked secretion of acute bronchitis are hardly recognised.

The natural secretion of the bronchial mucous membrane is a colourless liquid of somewhat glutinous quality, like a thin solution of gum arabic. It does not much differ in composition from the serum of the blood, and owes its viscidity to an animal substance, which Dr. Pearson, Dr. Bostock, and Berzelius, concur in considering an imperfectly coagulated albumen. This secretion is the basis of most of the varieties of expectorated matter; but unhappily our present knowledge of animal chemistry does not enable us to discover the precise

nature of the changes in composition which produce these varieties. All that we learn is, that albumen in different forms and proportions is present; for whether the expectoration be mucus, serum, pus, tuberculous matter, or coagulable lymph, the chemist can discover in these but scarcely discernible varieties of the same principle. There is a considerable variation in the proportion of saline matter in different kinds of expectoration; and on this depends a distinction, formerly much insisted on, by means of the salt or sweet taste. This criterion certainly fails to distinguish pus from mucus; but I think that an excess of saline matter may be taken as a sign of inflammatory action in the mucous membrane.

It is by its mechanical and visible conditions, however, that expectorated matter is most distinctly characterised; and to examine these fairly, the entire sputa should be collected in one or more convenient vessels of white ware or glass, in which their quantity, colour and consistence, can be minutely scrutinized.

PART II.

ON THE PHYSICAL SIGNS OF DISEASES OF THE LUNGS AND PLEURA.

HITHERTO we have considered physical signs only with relation to the natural or physiological state, and to the general pathology of the thoracic organs: it now becomes our task to study the forms or characters that individual diseases present to the auscultator. To understand the physical signs of a disease, it is quite obvious that we must be acquainted with its pathological characters; for they are naturally inseparable: and I view it as not among the least advantages of physical examination, that it directs our attention through a confusing crowd of uncertain and equivocal symptoms of general derangement, to that substantial and primary lesion, which, if not the starting point of all, is that against which our practical efforts are the most required.

My object will not be to enter into minute details of pathology and morbid anatomy; but as far as my own observations and those of others will enable me, to explain the general physical nature of the changes which, in individual diseases, so modify the conditions of the thoracic organs, as to produce signs available as means of diagnosis, and as guides in practice. With this pathological exposition will be occasionally blended short accounts of the leading general symptons, which, although often equivocal or deceptive in themselves, yet taken in conjunction with the physical signs, greatly contribute to the

useful and practical knowledge of disease. I shall generally confine the text to pathology, properly so called, but I shall not omit to append, in form of notes, a description of the general morbid appearances found after death, by a comparison of which with the physical signs during life, the real nature of a disease is to be known. I begin with the diseases affecting the air tubes of the lungs.

CHAP. I.—DISEASES OF THE AIR TUBES.

SECTION I.—*Bronchitis.*

The pathological cause of *bronchitis, acute mucous*, or *pulmonary catarrh*, is an inflammation and altered secretion of the mucous membrane of the bronchia. There are several varieties of this disease, arising from its extent, or from the state of constitution or complications with which it is associated; but as they pass insensibly into each other, and as the physical signs of all are frequently combined in one, I shall comprehend in this section their general description, distinguishing only the acute and chronic diseases.

1. *Acute Bronchitis.*—Inflammation of the mucous membrane of the bronchi at first causes tumefaction and partial obstruction of their caliber. This partial obstruction or constriction, occurring in individual points, so modifies the passage of air through the bronchial tubes, that it produces sound after the manner of instruments of music. If the whole periphery of a portion of a tube be tumified, the constriction is circular, and the air passing through it produces a whistling sound. This constitutes the *sibilant rhonchus*. If the tumefaction be unequal, so that the constricted portion preserves a flattened

aperture, then a sound is produced after the manner of reed instruments, or rather of the horn or trumpet, by the rapid alternate compression and dilatation of the air passing between two vibrating laminæ or surfaces. Such, I conceive, is the rationale of the *sonorous rhonchus*. The extent of the constriction, its situation, and the secretion lubricating the tube, will variously modify the note and tone. The larger bronchial tubes alone can produce deep or bass notes; but it is plain that they may also yield high ones. When a deep sonorous rhonchus is produced in a bronchus near the surface of the lung, it communicates a slight vibration to the corresponding paries of the thorax, which may be felt by the hand. This mechanical vibration is often perceived internally by the patient himself, although he does not hear the sound that produces it. As the inflammation attacks the larger bronchial ramifications first, the rhonchus is usually grave, and frequently resembles the prolonged note of a violoncello, and sometimes the cooing of a dove.

The sonorous and sibilant rhonchi, then, we find to be the first physical signs of pulmonary catarrh, and these are sometimes present before the cough becomes pronounced, and while the local feelings only indicate a nasal coryza, or a roughness or dryness in the throat. In slight cases, a feeling of chilliness, with occasionally pain of the chest and sore throat, are the only general symptoms at this period; but when the disease is extensive, there are rigours, quick pulse, and other symptoms of incipient fever, with more or less tightness and pain across the sternum, dyspnœa, and dry cough.

After a while the inflamed membrane begins to secrete a somewhat viscid saline tasted liquid, which at first mellows the sound of the rhonchi, but afterwards increasing, interrupts it by the formation of bubbles, which momentarily stop the vibrations, and then burst. These bubbles increase in number

as the secretion increases, and are at last produced in such a continuous succession, that the sound of the former rhonchi ceases, and is replaced by a new one produced by the successive formation and rupture of bubbles in the air tubes. This is the *mucous* or *bubbling rhonchus*. In the larger bronchi the mucous rhonchus is composed of bubbles of unequal size, causing a gurgling sound; but in the smaller tubes the bubbles are more uniformly small, and the rhonchus may be called finer: they are still, however, somewhat unequal; and, even when in the extreme bronchi, they can be distinguished to be liquid bubbles, and quite different from the uniform dry crepitation that constitutes the crepitant rhonchus. The matter expectorated is now transparent, and resembles raw white of egg diluted with water, and if brought up with much coughing, contains many air bubbles. When poured from a vessel, it falls in a stringy or ropy stream. Its viscidity is generally in proportion to the intensity of the inflammation, any exacerbation of the fever always increasing this quality; at the height of the inflammation, and at other times, when the cough is violent, streaks of blood are sometimes seen; but there is never a rusty tinge, as in pneumonia.

With this state of the expectoration, the other symptoms reach their acmé. The cough and heat in the chest become troublesome, and in severe cases the dyspnœa increases, with heaving thoracic respiration; whilst the auscultator perceives the cause, by the extensive mucous rhonchus accompanying the whole respiratory act. In the bronchitis of young children and of old persons, in whom the bronchial secretion is usually early and copious, the rhonchus is often universal, and this circumstance, together with a livid hue of the lips, imports great danger, whether the sensible dyspnœa and cough be urgent or not. The chest still retains enough air to sound pretty well on percussion, mere bronchial effusion being inca-

pable of rendering it dull, except, perhaps, when conjoined with great pulmonary congestion, which immediately precedes death.*

The decline of the disease in favourable cases is marked by a change in the expectoration, which becomes pearly or of a greenish white, and more consistent. This change is first perceived in the morning expectoration, and it is always accompanied by a remission of the fever, and generally of the other symptoms. The physical signs also announce the thickening of the mucus contained in the air passages, by the mucous rhonchus becoming drier and more sluggish, from the resistance opposed to the air in passing through the inspissated liquid. This resistance increases with the increasing spissitude of the mucus, and sometimes amounts to a complete obstruction of the tube; and in this case the sound of respiration ceases in the part supplied by it. More frequently, as the mucus becomes thick, its quantity is diminished, and then it only partially obstructs the tube. This straitening of the calibre may cause a rhonchus, and being soft and incapable of vibration itself, the sound produced is a whistle, in which air is the only vibrating body. Occasionally, however, at this period of the catarrh, a ticking sound is heard, like that produced by the click wheel of a small clock. This is caused by a pellet of thick mucus at the orifice of a bronchial ramification, which acts like a loose valve, yielding, in successive jerks,

* The anatomy of fatal cases of bronchitis, as far as it goes, is in perfect accordance with what we learn from the physical signs. The lungs do not in general collapse on opening the thorax, the escape of air being prevented by the obstructions in the bronchi. These, in most instances, contain a quantity of frothy liquid, like that expectorated during life. Sometimes it is mixed with bloody serum; but this, not having appeared in the sputa, has probably exuded from the surcharged blood vesssls during and after dissolution. Purulent matter is also observed in some very acute cases, especially those supervening in exanthematous fevers, which have proved fatal in four or five days. The mucous coat presents an appearance of an injection, diffused or in patches, of various shades, from a bright crimson to a deep brown red. It is sometimes thickened, but rarely much softened.

to the air pressing for passage. A change in the force of respiration may much modify these several sounds. Thus, the forcible expiration and inspiration accompanying a cough may produce the clicking sound, or even the sibilant rhonchus, in a tube which, in ordinary respiration, is totally obstructed with mucus; it may convert the clicking into sibilation, and this into the simple sound of the passage of the air; the obstacles yielding, in all these cases, to the increased force of the passing air. It is therefore useful to avail ourselves of this simple mode in our examination; for on desiring the patient to cough, the nature of the obstruction may frequently be made apparent by the momentary presence of one of the above signs.

The uncertainty in which the signs of auscultation sometimes leave us is completely removed by percussion. The sonorousness of the chest is never materially impaired by catarrh; and accordingly, the partial suspension of the respiration in a part of the chest, in this disease, cannot be erroneously ascribed to hepatization, or an effusion in the pleura.

The extent, as well as the seat of the catarrh, may be determined by the rhonchi. When the inflammation occupies only the large tubes, as is the case in common colds, the bubbles of the mucous rhonchus are large and uneven, and the sound of respiration may still be heard. But if the rhonchus is fine, and accompanies the whole of the respiratory act, the disease extends to the small bronchi, and if extensive, must impede the function to a serious degree. Cases of this kind proving fatal, are often, in this country, erroneously considered peripneumonic. In some cases of continued fever, the rhonchi indicate a catarrh of various stages in every part of the lungs; they are the sibilant, sonorous, and mucous rhonchi; and when thus mixed, Laennec used to designate them *rhonchus canorus*. Their presence may be considered a very unfavourable sign, and is seldom indicated by the cough or other symp-

toms, being, as it were, masked by the general affection of the system. In general, severe bronchitis is most dangerous at the extremes of life, and this depends, partly on the profuse quantity of the secretion, and partly on the inferior capability of dilatation in the pulmonary tissue at these ages; greater capacity in adults permits supplementary respiration in the healthy parts to supply the defect of the diseased. Besides these, as in all diseases obstructing the respiratory function, the dyspnœa (and hence the danger) will be great in proportion as this function is naturally or constitutionally active in the individual.

If the catarrh terminates in cure, the expectoration becomes thicker, and more concocted, as the ancients termed it. It is voided without irritation, in rounded, distinct pellets, consisting of an opake, greenish mucus. These and the cough diminish, and are confined to the morning, after waking, and a few times in the evening, and at last cease altogether. A relapse is equally marked by a return of the sputa to the glairy transparent state, which must undergo the same change before the disease is removed.

2. *Chronic Bronchitis.*—If neglected, pulmonary catarrh may assume a chronic form. The cough and expectoration then continuing, the latter is usually at first of the same quality as at the termination of the acute stage, but it sometimes becomes diffluent, less viscid, of a dirty brownish colour, and mixed with a serous fluid. The same mucus is sometimes voided in a more inspissated state, and even moulded into the bronchial ramifications.* In severe cases, the sputa frequently are mixed with pus, and sometimes become completely purulent, presenting all varieties in odour and consistence that pus, from other sources, offers. In such instances, there are generally quick pulse, and signs of hectic; and the disease tends

* Cheyne, Andral, and Laennec.

to a fatal termination with night sweats, diarrhœa, emaciation, and all the common symptons of pulmonary consumption. These ensue most frequently on repeated attacks of acute, intense, or humid bronchitis, influenza, pertussis, and severe catarrhal affections during eruptive diseases; all of which effect a serious change in the bronchial membrane. The same change results more directly from the habitual inhalation of various kinds of dust, and occasions the severe chronic bronchitis of needle-pointers, stone-cutters, leather-dressers, and other artisans subject to this influence.*

The physical signs, when carefully studied, often afford us the only means of forming a correct opinion of these cases; but here let me caution the young auscultator against too perfect a confidence in his examinations, and too hasty a conclusion from their results. As the diagnosis is important, so is it often difficult.

The symptoms heard by the ear in chronic catarrh, are the mucous rhonchus, in most of its varieties, shifting and intermitting from time to time, and occasionally the sibilant and sonorous, the presence of which is explained by the sputa; the sound of respiration, sometimes diminished, but usually unimpaired, or even puerile;† the chest, on percussion, yields

* See Cycl. of Pract. Med. Diseases of Artisans.

† The presence of the dyspnœa, in these cases, where there is no obstacle to the entrance of air into the lungs, nay, where the puerile respiration shews it to be more perfect than usual, is ascribed by Laennec to an increased "*besoin de respirer.*" In the present instance, however, I see nothing more in this phrase than an expression of the fact. There is nothing in the state of the system that indicates the want of an increased activity in the respiratory function. The quantity and quality of the urine, and the other excretions, may be taken as pretty correct criteria of the extent of the chemical changes by respiration. I think that we must look rather to the change in the bronchial membrane, and in the nature of its mucus, for an explanation of the point in question. I have elsewhere (Trans. of Med. Chir. Soc. of Edin. vol. ii. p. 100) pointed out an important part which this mucus performs in assisting the action of the air on the blood. It is easy to conceive how a diseased state may unfit it for this office, and impair the chemical function of respiration, however perfectly the mechanical part may be performed. See Cyclop. of Practical Medicine, Dyspnœa, by the Author.

a clear sound, and, examined by the eyes and hands, appears to expand pretty uniformly. It will be perceived that all these signs are negative, and none of them are characteristic of this modification of bronchitis. It is, therefore, in the absence of the signs hereafter to be described, as peculiar to phthisis, that we must recognize the character of chronic catarrh.* As, however, negative are weaker than positive proofs, so must they be multiplied to be rendered certain. If, after having repeatedly examined the patient, at different hours during several days, there are found no gurgling cavernous rhonchus, no cavernous respiration, no pectoriloquy, and no constant absence of the respiratory murmur, and of the sound on percussion, then, in spite of the general symptoms, we may, with tolerable certitude, pronounce the disease to be simply pulmonary catarrh, and a still further multiplication of examinations will remove all doubt.†

The long continuance of chronic catarrh may entail an organic change in the lung, which will sometimes render exceedingly difficult a distinction between its signs and those of tubercular phthisis. The bronchi, long the seat of chronic

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* The mucous membrane is found on dissection to exhibit, sometimes a violet red colour, diffused or in patches, sometimes little or no discolouration, and occasionally even a blanched appearance. Softening and ulceration are rare, except in the cases arising from the inhalation of dust. Partial thickening is more common. But the dilatation of the bronchi is the most remarkable effect; and this is sometimes uniform, so that a tube of the size of a straw suddenly swells to that of a goose-quill; in other cases, the dilatations are partial, and form a series of sacs, of sizes from a pea up to a walnut. The membranes lining these dilatations are sometimes thickened, but sometimes quite the reverse, and in some instances they are indurated, so that the dilated portions are quite rigid. The mucous membrane is however commonly softened.

† This passage I have given nearly in the words of the illustrious discoverer of auscultation; yet, aware as he was of the attention required in the examination, and of the fallibility of a hasty judgment, I have more than once seen himself give proof in point by the failure of a premature diagnosis. If then, one, from knowledge and experience so profoundly acquainted with his subject, was, through inadvertency, led into error, how much more circumspect should they be who have not his experienced tact, and his talent for improving observation.

inflammation, and exposed to the straining influence of repeated paroxysms of cough, become hypertrophied and dilated.

Laennec supposes these dilatations to be produced simply by the accumulation of a thick mucus mechanically distending them: but in this case we should discover more frequently an absence of the respiratory sound in the part: such a sign, although common in the latter stages of the acute disease, scarcely ever occurs in chronic bronchitis. I am more disposed, with Andral, to ascribe them to the altered texture of the membrane; but even this is only the predisposing cause. The physical cause, I apprehend, is to be found in acts of respiration and cough exerting a degree of pressure on the membrane greater than its elasticity can resist. Thus the forcible inspiration which succeeds each fit of coughing, acts with greater effect on these weaker parts: and again, the violent expiration of coughing brings an undue pressure on the same tubes, which, distended in one part, and partially obstructed by the thickening of their membrane in another, are perpetually exposed to a straining influence.* Induration, the effect of another degree of the inflammatory process, sometimes follows, and gives the dilated portions the rigidity that is occasionally noticed in them. The effect of these dilatations must be to obliterate the neighbouring cells, and to abridge the function of the lung; and accordingly, when extensive, the disease causes habitual dispnœa.

Now it may easily be perceived, that these dilatations, if superficial and large, may be the seat of pectoriloquy, cavernous respiration, and the other signs which are generally distinctive of phthisical disease. When the dilatation is uniform, there will be a loud bronchophony, and a *whiffing* sound of respiration. It is only by attention to the progress of such

* These opinions were first advanced by me in 1828. Somewhat similar views have been adopted by M. Roche, *Dict. de Méd. et de Chir. Prat., Art. Bronchite*, 1830.

cases, that they can be discovered not to be phthisical. A tuberculous excavation rarely remains stationary as these do; but enlarges, and gives a pectoriloquy of greater extent, whilst new cavities are formed in other parts. Dilated bronchi rarely affect the shape of the chest, or its sound on percussion, as tubercular disease does. The most common situations of dilated bronchi are in the scapular, mammary, and lateral regions; the infraclavian and acromian are the more usual seats of phthisical signs. The sputa are often remarkably fetid, and of course do not contain the portions of tissue and tubercular matter occasionally seen in phthisis. It must be confessed that cases do occur, although rarely, which leave the most experienced auscultator in uncertainty; but these are exceptions, of comparatively little practical import, and hardly detracting from the value of auscultation. For dilatation of the bronchi is incurable, and when presenting the equivocal form in question, must be connected with such extensive lesion of the bronchial membrane, that the prognosis would be much the same as in phthisis itself.*

Section II.—*Pituitous Catarrh.*

The varieties of catarrh, which Laennec terms, from the nature of the expectoration, *pituitous* and *dry catarrh*, require to be noticed as far as they differ in their physical signs from mucous catarrhs.

In the pituitous catarrh, or humoral asthma, as it is more commonly called in this country, a thin, colourless, glary liquid, is secreted in abundance by the bronchial membrane.

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* For a full account of the general history and treatment of bronchitis, see my article in the Cyclopædia of Practical Medicine, vol. I.

This flux comes on in paroxysms, attended with dyspnœa and cough, which are relieved by a copious expectoration of the liquid. It does not appear that the membrane becomes permanently tumified, unless occasionally by the co-existence of a slight degree of œdema. The dyspnœa and cough are therefore to be chiefly ascribed to the quantity of pituitous secretion.

The respiratory murmur is weak, accompanied with the sonorous and sibilant rhonchi, occasionally modified by bubbles of the mucus, so as to imitate the chirrupping of birds, and sometimes heard distinctly with a liquid mucous rhonchus. When a slight œdema is present, the humid crepitant or subcrepitant rhonchus may also be distinguished, but this disappears in the interval with the other signs. The chest, on percussion, sounds well throughout the attack.

This catarrh may be confined to one or two paroxysms, or it may attack daily for months and even years. Like most other serous fluxes, it is very difficult to remove when once established, and sometimes arises from the development of a number of miliary tubercles in the pulmonary tissue. Its long continuance produces that change in the mucous membrane that commonly accompanies, or is produced by, profuse watery discharges. This is a degree of atrophy which is sometimes attended with perfect pallidity, and sometimes with slight irregular striæ or patches of sanguineous injection.

These circumstances, and the fact that it most commonly attacks those of weakly habit, languid circulation, and little disposed to inflammatory symptoms, indicate the prevailing evil to be a debility or want of tone in the vessels of the bronchial membranes, on account of which the watery parts of the blood transude with little restraint and little modification. The effects of remedies, and the nature of the predisposing causes, confirm this view. Inflammation may have been in the first instance the cause of the loss of tone in the

vascular fibre; and even although it does not afterwards continue, stimulating agents may aggravate the disease, by increasing the local force of the circulation, by which a flow of liquid becomes directed to the weakened part.

Section III.—*Dry Catarrh, or Bronchial Congestion.*

The affection to which Laennec has given the contradictory name of *dry catarrh*, is, perhaps, in its general signs, more allied to asthma than to the preceding diseases. The ordinary symptoms are dyspnœa, varying according to the extent of the disease, and cough, with scanty expectoration of a tough, translucent, ash-coloured mucus. It consists in a sanguineous congestion in the membrane of the bronchi, which causes tumefaction, and partial or complete obstruction in their calibre. There is with this the scanty secretion of thick mucus, which arranges itself in globules, completing the obstruction of the tube.

The stethoscopic sign of this affection is, accordingly, a suspension of the sound of respiration in the part affected, whilst the chest sounds perfectly well on percussion. Sometimes the obstruction is not quite complete, and then there may be a slight sibilant or a clicking rhonchus, which may generally be made audible by the cough.

The severity of this affection depends entirely on its extent, and this may vary from a degree not at all deranging the general health, to one producing severe and oppressive asthma. Many persons, apparently in perfect health, only perhaps subject to some shortness of breath on exertion, present to the auscultator examples of the slightest degree, in a defective respiratory murmur in some part of the chest; and these usually expectorate every morning a small portion of the

pearly mucus that I have described. If the engorgement affect a longer extent of the bronchi, some degree of dyspnœa may be felt, even when the person is at rest, particularly after meals. In a severer case the dyspnœa may last for several days, and is usually relieved by cough and expectoration of a small quantity of the same viscid mucus. These symptoms are still in proportion to the extent of suppression and obstruction of respiration observed by auscultation.

This disease not unfrequently terminates in the pituitous form; or rather, its paroxysms sometimes end in a watery expectoration, with a small proportion of the tough mucus in it. Like pituitous catarrh, it may have its first origin in an inflamed state of the mucous membrane; but from the natural duration of the symptoms, and the effects of remedial measures, as well as from the appearance after death, I have always considered its present cause rather as a passive congestion, and consequently interrupted secretion, arising, perhaps, from deranged nervous influence, than an active inflammation. It is very commonly connected with disordered digestion, or imperfect secretion of bile, with irregular alvine evacuations.*

Section IV.—*Pertussis, Croup, &c.*

The physical signs of *pertussis* do not materially differ from those of bronchitis, and at the commencement they depend on the same cause, inflammation and altered secretion of the bronchial membrane. The febrile character of the first attack, and especially the convulsive mode of cough, distinguish this

* I can bear testimony to the efficacy of the alkaline remedies recommended by Laennec; but their effect is only temporary. The most successful treatment of this and the preceding affection, is that addressed to the pathological state of the mucous membranes, and to its constitutional causes. See the article Catarrh, by the Author, in the Cyclopædia of Practical Medicine.

disease. In the intervals of cough, the respiratory murmur becomes indistinct in some points, and puerile in others; at first a mucous, afterwards a sibilant or sonorous rhonchus are sometimes heard, and the sound of the chest, on percussion, is unimpaired. From this it may be concluded, that the violence of the cough does not depend entirely on the state of the mucous membrane of the air-passages, and an examination, during a fit of coughing, confirms this conclusion. If the ear is applied to the chest at this period, no rhonchus or respiratory sound is heard, except for a moment, between each cough; and during the sonorous back-draught all is silent within the chest. This absence of the respiratory sound, in an inspiration that seems so deep and forcible, is to be attributed to the admission of air being slow and scanty, on account of the spasmodic constriction of the glottis, by which, too, the hooping noise is caused. A spasm of the muscular fibres of the whole bronchial tract may also contribute to the exclusion of air from the air-cells, but I cannot, with Laennec, consider this as the only cause.

The great tendency of pertussis, in young children, to pass into pneumonia or intense bronchitis, makes frequent auscultation of the chest very necessary in these cases. A crepitant rhonchus commencing, or a mucous rhonchus extensively prevailing, should call for decided measures.

Croup is to be recognised by the sudden hoarseness with fever, followed by the characteristic croupy cough, dependent on the albuminous exudation from the mucous membrane of the trachea and bronchi. These signs, although truly physical, need neither stethoscope nor comment to explain them. When the disease extends to the bronchi, the respiration about the sternum, which is naturally bronchial, becomes sibilant or whiffing; but it is often difficult to distinguish it from a similar rhonchus which generally, also, has its seat in the trachea. Laennec gives a solitary example of a bronchial croup, in which the presence of an adventitious membrane caused a dry and

tubular respiration, without the diffused, slightly crepitant sound so marked in children. This, with the sound of percussion unimpaired, if found constant, would (he suggests) be sufficiently distinctive of this form of the disease. The clearest physical sign of inflammatory tracheal croup is, certainly, the detachment and expectoration of the albuminous concretions formed in the air-passages. The suddenness and more transitory nature of the attack, characterises spasmodic croup, or spasm of the glottis; but this sometimes arises also in the course of inflammatory croup, and there is with the former sometimes such a degree of congestion, as to pass into this disease.*

I do not believe that ulcers of the bronchi have any constant sign by which they can be distinguished. They excite a copious mucous secretion from the membrane, which is sometimes mixed with pus and blood. The presence of the same liquids in the bronchi occasions a mucous rhonchus. In these cases the local pain, excited particularly by exertions of the voice, is the most characteristic symptom.

For the diagnosis of polypous and other tumours in the bronchi, I must refer the reader to his own reflections; for as I am convinced that no one can become a good auscultator by the use of his ears and memory only, so do I maintain that by a knowledge of the properties of sound, and a happy generalization of its phenomena, an observer will be enabled to explain and appreciate not only all those signs that experience has hitherto discovered, but those that may also be revealed by future observation. Thus he who knows how the sonorous and sibilant rhonchi are produced, will perceive that a tumour pressing on a bronchus may likewise cause them. He will see, in a hæmorrhage simply bronchial, all the elements necessary to produce the mucous rhonchus, &c.

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* For the best treatise on Croup, see that prodigy of labour and talent, the Dictionary of Practical Medicine, by Dr. Copland.

Section V.—*Spasmodic Asthma.*

Asthma, or *dyspnœa occurring in paroxysms*, may arise from a variety of conditions in the lungs themselves. Thus we have seen that the states of the mucous membranes constituting dry and pituitous catarrh, produce forms of asthma, and I have ventured to ascribe another, (which, as the air enters the lungs freely, Laennec vaguely refers to an increased *want of breath*) to the altered condition of the mucous membrane, and its secretion, caused by chronic bronchitis. Other varieties of asthma we shall find accompany emphysema of the lungs, and extensive disease of the heart. But in nearly all these cases, the asthmatic attack is often more sudden, severe, and transitory, than the above various pathological states, simply considered, can account for; there is, in truth, a disease temporarily superadded, and this occurring separately, constitutes the complaint strictly called Spasmodic Asthma. The existence of this disease has long been *assumed* to explain the phenomena just mentioned; but until Reisseissen demonstrated the existence of muscular fibres in the bronchi, and Laennec, by auscultation, ascertained them to be occasionally affected with spasm, spasmodic asthma was merely a matter of conjecture.

The causes exciting purely spasmodic asthma, are those which impress the nervous system, as strong or peculiar odours, irritations in the stomach or bowels, mental emotions, peculiar states of the atmosphere, and the like. The attack comes on suddenly,—and, if severe, forces the patient to assume a remarkable attitude with his body bowed forwards, and chest contracted, with the sensation of a weight on it, or a tight cord across it; the face becomes suffused and anxious, the veins turgid, with the other characters of ordinary dyspnœa.

During the paroxysm the chest sounds ill on percussion, and the respiratory murmur is indistinct, even on the most forcible respiration. But if the patient, after holding his breath a little while, be desired to breathe again quietly, the spasm will be overcome as it were by surprise, and the entry of the air into the cells will be heard in a clear and sometimes puerile sound. This may be best effected in slight attacks, or where the spasm is permanent, as recommended by Laennec, by desiring the patient to read aloud, or count as many numbers as he conveniently can without taking breath, and then to breathe at his ease. But after one or two inspirations, the spasm regains its hold, and the respiration becomes as dull as ever. The diminution of the respiratory noise here, obviously proceeds from the obstruction opposed to the entry of air into the small bronchi and vesicles, by the tonic contraction of their muscular fibres. By the same contraction the lungs are in a manner collapsed within the thoracic cavity, and the parietes of the chest, pressed by the atmospheric weight on them, lose that sonorous elasticity produced by a fulness of aëreal contents. The chest thus contracted to the size of the collapsed lungs, may be compared to a drum, the parchment of which is pulled in by transverse strings. The free vibration is thus checked by these unyielding frœna. Conceiving, as I do, that the contraction of the bronchial muscles is a sufficient cause of the phenomena of spasmodic asthma, I cannot support the hypothesis of the active dilatation of the lungs, even supported as it is by such high authorities as Laennec and Dr. Copland. The supposed active protrusion of the lung through wounds in the chest, is far better explained by the irregular pressure of the respiratory forces, in such an unnatural condition of the parts, than by an assumed expansive power, of which the structure of the lungs affords not the slightest proof, and which is opposed by all we know of animal dynamics. There is doubtless a *mechanical elasticity* in the

lungs, which may expand them after they have been unnaturally contracted by the antagonising action of the bronchial muscles; but this is not vital expansibility.

The dyspnœa produced by spasm of the bronchi is often of long continuance, and may, to a certain extent, become habitual. In such cases the system accommodates itself to the diminished supply of air, and the respiratory function is less called into action; but slight causes, either reproducing the want in the system, or increasing the spasm, will be sufficient to bring back the dyspnœa. Of the first class of causes are exertion, the sudden application of cold, &c.; of the second, emotions of the mind, and sympathetic irritations, produced by certain ingesta in the stomach and intestines. This second class includes usually those which originally produce the disease. I have seen a remarkable and exquisite case produced by the slow introduction of lead into the system, but such a form of saturnine neurosis is, I believe, rare.

It may be also readily perceived, that a congested state of the lungs, or an undue injection of the bronchial membranes, may excite the bronchial muscles to spasm; the former, by the irritation of the mass of the blood; the latter, by its exalting the sensibility of the membrane; and thus, violent exertion, diseases of the heart, and other affections of the lungs and of their membranes, may occasionally develope fits of asthma.*

This affection, when complicated with partial dry or pituitous catarrh, may be partial, affecting one lung only, or one more than the other, and the signs of these diseases will then be observed in some parts of the chest, while those pathognomic of the spasmodic affection will be heard in others.

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* For an attempt to define the pathological principle, *irritation*, of which these are illustrations, see the author's article on that subject in the Cyclopœdia of Practical Medicine.

CHAPTER II.

DISEASES AFFECTING THE TISSUE OF THE LUNGS.

SECTION I.—*Peripneumony.*

PERIPNEUMONY consists in an inflammation of the parenchyma of the lungs, and, according to the changes produced in the tissue, it is divided into three stages.

The first is that of simple inflammatory injection, in which the size and apparent number of the blood vessels is greatly increased. Our knowledge of minute anatomy does not permit us to specify with certainty the exact and essential seat of this inflammation; but I am disposed, from a consideration of the signs, and from many minute examinations of the effects on the tissue, to refer it principally to the important plexus of blood vessels, which in fact constitutes a chief part of the parenchyma of the lungs. It is its seat in this plexus, which, as constituting the organ of the minor circulation, through which the whole blood of the body must pass for a vital purpose, stands in most peculiar and important relations to the functions of the heart, between whose compartments it is the communicating channel—that gives to pneumonia its remarkable and serious character. From this plexus it usually extends more or less to the mucous lining of the bronchi, and to the pleura investing the inflamed part, but these extensions are uncertain and trivial compared with the essential disease of the vascular parenchyma.*

* On dissection, the lung in this stage is found to be of a livid red colour, of various shades; it is increased in weight, and pits on pressure, but it is still somewhat crepitant, and usually floats in water. When cut into, it still presents its

In this stage of the inflammation, the distended vessels, and perhaps a serous effusion in the interstices, press on the minutest bronchial ramifications, and partially obstruct the ingress of air into the cells to which they lead ; whilst the viscid secretion of the mucous membrane, simultaneously inflamed, filling the calibre of the tubes thus narrowed, only yields to the air in respiration forcing its way through it in successive bubbles. This bubbling passage of air through a viscid liquid, contained in an infinity of tubes of equally diminished calibre, causes that regular and equable crepitation which constitutes the true *crepitant rhonchus*. If the inflammatory infarctus be not so general as to prevent the air from entering without obstacle into many of the bronchial cells, then, besides the crepitant rhonchus, the natural sound of respiration will be heard. On the other hand, the inflammation increasing, and passing into the second stage, causes a total obstruction of the cells, and all sound of vesicular respiration, and even of crepitant rhonchus, is confined to the end of each inspiration, or to the act of cough, and at length ceases. The progress of the inflammation is, therefore, now marked by the gradual cessation of the crepitant rhonchus.

The sound on percussion is only slightly impaired in the first part of this stage; but as the engorgement advances, and the proportion of air in the inflamed spot of lung is diminished, the sound becomes more dull in the corresponding part of the chest. If the disease be extensive, manual examination may also often detect a deficiency in the motion of the ribs on the affected side.

The general symptoms of this stage are, fever, with more

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spongy structure, out of which generally exudes abundantly a spumous bloody serum. Sometimes it is softer and very livid, and only a little spumous blood flows from its cut surface. Its integral cohesion is diminished, for the texture may be easily broken down between the fingers. The mucous membrane of the small bronchi is of a deep red colour.

or less pain in some part of the chest, cough with viscid expectoration, accelerated and sometimes oppressed breathing. The decubitus is generally on the back; the pulse quickened, but very variable in its character; the fever is generally of the inflammatory kind, but is occasionally typhoid.

Pneumonia modifies the secretion of the bronchial mucous membrane in a very remarkable manner. At the commencement of the disease there is frequently no expectoration, or it is simply catarrhal, being composed of a mucus of moderate tenacity; but as the crepitant rhonchus becomes marked, the sputa assume their characteristic form. They are semi-transparent, tenacious, and run together, forming one mass of a reddish yellow, or rusty tinge of various shades. As the disease advances, this tenacity increases. At first it does not much exceed that of the white of an egg, and when poured out, the sputa fall in glutinous strings, but at the height of the first stage they are frequently so viscid, that inverting the vessel, and even shaking it in this position, will not suffice to detach them from it. The same tenacious property imprisons in the mass a multitude of little air bubbles, which sometimes produce a spumous appearance. The colour may vary, in numberless gradations, from a light reddish or greenish yellow, to a deep orange, a dingy red, or rusty hue. All these tints proceed from various proportions of the colouring matter of the blood, which transuding from the engorged pulmonary vessels, becomes intimately combined with the secretion of the bronchial membrane. Quite different from these are the sanguinolent sputa, that sometimes occur in catarrh, in which the blood appears in distinct striæ. The intimately combined tint, and the glutinous viscidity of peripneumonic expectoration, give to it a character perfectly pathognomic, and sufficient in itself to prove the presence of the disease. Moreover, the degree of viscidity announces, with tolerable precision, the intensity of the inflammation; and whenever, after having become thinner

in the course of cure, the sputa regain their former viscidity, a relapse into the disease is indicated. But although the presence of these sputa indicates with certainty the existence of pulmonic inflammation, we cannot draw an opposite conclusion from their absence. They rarely appear until the second or third day, sometimes not till later, and in some cases have not been observed at all.

The second stage of peripneumony is that in which the lungs present that change in the tissue which is called by Laennec hepatization.* This change consists in the effusion

* *Ramollissement rouge* of Andral.—A hepatized lung presents the following characters after death: Externally it is of a deep red colour, which internally is studded with a number of lighter small granular spots, with patches of whiter colour, marking the vessels, interlobular septa, &c. less affected with the inflammation. It sinks in water, and is no longer crepitant, but, inelastic to the touch, breaks readily under the fingers, and may, by a slight pressure be reduced to a red pulp. When cut into, it is often mottled, and paler than in the first stage. The little granular points are best seen on tearing the lung, and they then seem to be the most solid parts. They appear to consist of the little bunches of vesicles, (in which, according to Reisseissen, each minute bronchus terminates) whose membranous tunics have become so swelled by the deposition of a soft albuminous matter in them, as well as from the increased size of their blood-vessels, that their cavities are obliterated. For a full consideration of this and other parts of the anatomical and pathological history of Pneumonia, the reader is referred to the author's article on that subject in the Cycl. of Pract. Med. from which I will insert here one extract.

"On inspecting, by aid of a simple lens, the margin of a slightly inflamed spot of lung, numerous vessels may be seen, distended with blood, passing across, around, and between the vesicles; and as the scrutiny is extended to a part more inflamed, these vessels are so multiplied and confounded with each other, as to be no longer separately discernible. In this state, it is impossible to distinguish whether the tunics of the cells, or the tissues which connect them, are most affected, for they all appear one mass of redness, in which are seen the cells irregularly diminished in size, and containing bloody serum with bubbles of air. The interstitial cellular texture, where it can be separately discerned, namely, between the lobules, and around the larger bronchi, is generally less vascular, and of a lighter colour than the other parts, and in some instances is nearly free from the inflammation. The lining membrane of the minute bronchi, although generally of a deep red colour, is sometimes bluish red, as if from redness under it, rather than in it; and on tracing these tubes higher, the presence of this inflammatory mark is very uncertain. These examinations, and some pathological considerations, induce me to regard the capillary ramifications of the pulmonary artery and veins as the proper seat of pneumonia, and that these may involve more or less of

of a semi-solid albumen in the interstitial tissues, which, pressing on and obliterating the cavities of air-cells and smaller

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the tissues through and around which they pass. Thus, through them, the tunics of the air-cells, particularly the submucous, commonly become the seat of inflammation, whence are formed the granulations of ordinary hepatization. When, again, the inflammation is confined more to the intervesicular plexus and tissue, which appears to be the case in the more congestive form of inflammation, where vessels larger than capillaries are involved, a uniform, non-granular form of hepatization is produced. If, as is commonly the case, the inflammation has extended to the extremities of the bronchial arteries, which are said by anatomists to anastomose with the pulmonary, the mucous membrane lining the vesicles, and minute bronchi, partake of the inflammatory action, and exhibit it in the manner peculiar to mucous membranes, by the secretion of a viscid mucus, and afterwards of pus. So likewise, when the inflammation reaches the surface of the lung, it is generally, but not constantly, propagated to that portion of the pleura which invests it, and derives its vessels from it, and this extension of the inflammation may give a pleuritic character to such instances of the disease. But it is in the extensive capillaries of the lungs, through which the blood of the whole system is continually passing,—it is this affection of so considerable and important a portion of the circulatory system in the immediate vicinity of the heart, that causes the severe and intense character of pneumonic inflammation; and the more constantly we hold this in view, the better shall we understand the pathological history of the disease, and its important relations to remedial measures."

In connection with these views of the minute pathology of pneumonia, may be considered the physical cause of the crepitant rhonchus, on which various opinions have been formed; and in doing this, as some subjects must be anticipated, I would recommend the student not to read this note, until he shall have duly studied the subsequent sections.

It is agreed on by all auscultators, that œdema of the lungs, and the margins of hœmoptysical engorgements, may produce a rhonchus of the crepitant kind, and it naturally occurs to us, that these lesions resemble pneumonic engorgement, in the pressure to which they subject the vesicular parenchyma. If we compare with these instances the very close representation of the crepitant rhonchus, which the simple pressure of a healthy lung will produce, we are led at once to an explanation of the mechanism of this sign. This pressure forces the air from the tissue, but it so narrows the tubes and cells, that the air can only pass out of them in successive bubbles, the escape of which produces minute crepitations. This sound, Laennec tells us, differs from the crepitant rhonchus only in not being so strong, there being no preternatural fluid present in the air-cells. In œdema, or in effusion of blood into the substance of the lungs, we have interstitial effusion and narrowing of the minute air-tubes and cells, and from the nature of the expectoration we may conclude that there is also an increase of liquid within them; the rhonchus that accompanies them is accordingly described by Laennec to consist of moister and somewhat larger bubbles, and he terms it subcrepitous. Lastly, we have the peripneumonic engorgement, in which anatomy assures us that there is

bronchi, destroys the spongy texture of the lung, and converts it into a more or less solid mass. Such a condition of the

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the same narrowing of the air-tubes and cells by the swelling of the interstitial blood-vessels, whilst the observation of the sputa leads us to conclude that the interior of these tubes is during life lined with a viscid secretion;—this produces the drier and stronger sound of the genuine pneumonic crepitation. I have given the description of the sounds on the authority of Laennec, because as a practical auscultator, he has certainly never been surpassed; and yet he had no *fixed* views as to the cause of the crepitant rhonchus. (See Dr. Forbes' transl. pp. 52, 212.) M. Andral considers this sound to be nothing more than a finer modification of the mucous rhonchus, the latter becoming crepitant when its seat is in the minutest bronchi and vesicles, the narrow dimensions of which render the bubbles which compose it finer and more equal. This has also been ingeniously advocated by M. Piorry, and in this country by Dr. Spittal and others, who consider the crepitation to arise from the bursting of minute bubbles in a serous liquid, the same probably as that which flows after death from an inflamed lung, when an incision is made into it. I have doubted the accuracy of this explanation, because the character of the expectoration in pneumonia does not warrant the supposition that there is during life any other secretion into the air-cells in the first stage than the characteristic viscid secretion of the mucous membrane which lines them. I have before pointed out the tendency which the natural respiratory movements have to throw all superfluous secretion from the smaller bronchi into the larger, until they are brought under the influence of the act of expectoration. (See p. 43.) Now if, as those who found their opinions solely on morbid anatomy maintain, there were a serous effusion into the air-cells in the first stage of pneumonia, there ought to be more or less of this serum mixed with the sputa, as in some cases of pulmonary œdema; this, however, is not the case, for the expectoration is a glutinous mucus from the onset of the inflammation, and instead of becoming more serous, increases in viscidity as the inflammation becomes more intense. Moreover, we venture to assert (after Laennec) that there is a mucous or liquid rhonchus of the fine bronchial tubes, the character of which is sufficiently distinct from the rhonchus of pneumonia to merit its separation in kind as well as in degree from the latter sign. This fine mucous rhonchus, which I have observed in pituitous catarrh, and in the general bronchitis accompanying continued fever, occupying the base of the lungs, the common seat of the crepitant rhonchus, is distinguished from this by the greater inequality of the bubbles which compose it; they appear to roll through a liquid, without breaking with that regularity which distinguishes the crepitation in pneumonia. There are besides, little hissing or whistling sounds mixed with them, which convey to the mind the impression of a moveable proportion of air and liquid in the tubes. If this sub-mucous rhonchus is to be distinguished from the subcrepitant of pulmonary œdema and apoplexy, it is by the greater irregularity of the minute sounds which constitute it; but there may be gradations of the two which cannot be distinguished. But I still maintain that the crepitant rhonchus of peripneumony is, with few exceptions, sufficiently characterised by its pure equal crepitation, unmixed with hissing or any sounds

air-cells precluding any further ingress of air into them, what stethoscopic signs can we have to indicate this stage of inflammation in the living body? Here still a consideration of the physical state of the organ will teach us to expect, *à priori*, the same phenomena that experience has revealed. We have already had occasion to observe that the healthy lung, from its being composed of conductors of very different powers, (air, membrane and liquid) is a bad conductor of sound, and is, therefore, incapable of transmitting to its surface slight sounds, remote in the interior. But now that the tissue is rendered more uniformly dense by hepatization, it becomes a better conductor, and transmits a sound, (usually unheard) of the air passing to and fro in the larger bronchial ramifications. This is the *bronchial respiration* of Laennec and Andral; and specifically marks the second stage of pneumonic inflamma-

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of liquid, to render it a valuable and available means of distinguishing this disease in its earlier stages. In conceiving the mechanism of this rhonchus according to my explanation, we must take into account the force with which the air passes through the narrowed tubes, and we shall then perceive why the bubbles crepitate drily, and the liquid is not carried before the air passing to and fro, as it would were its viscidity less and its quantity greater. It is stated by Dr. Spittal of Edinburgh, that the crepitation of peripneumony may be most closely imitated by the bursting of bubbles on the surface of fluids of the tenacity of serum, and hence he ingeniously deduces that the crepitant rhonchus depends on the mixture of air with such a fluid in the lungs: but I must remark that the cases are not analogous: in the one, the bubbles rise and burst merely from their own levity; in the other, an active moving force is constantly driving and breaking them through an infinity of minute tubes. As there is no serous expectoration in the first stage of pneumonia, I cannot but regard the serum found in the lung after death, as in great measure the result of the cadaveric separation of the serum from the blood in the engorged vessels; and I would still, as formerly, refer the crepitant rhonchus to the forcible passage of air through the narrowed air-tubes and cells lined with a viscid secretion. As the vesicular parenchyma becomes obstructed by the deposition of albuminous matter from the inflamed vessels, this crepitating entrance of the air becomes less, and is heard only at the acme of inspiration, or during the more forcible efforts of coughing, and at length, when the stage of hepatization is complete, ceases altogether.

I feel great satisfaction in adding, that Dr. Chambers has long entertained opinions on the crepitant rhonchus of peripneumony very similar to mine, and he does not admit that a serous effusion accompanies the commencement of the inflammation.

tion. This sound, when once heard, cannot be mistaken. It resembles that produced by blowing through a crow's quill, and is frequently so loud as almost to amount to a whistle. This sound, acute and defined, forms a remarkable contrast with the dull, diffused sigh of natural vesicular respiration. In other parts of the lung, if the disease is extensive, the respiration is puerile.

Another nearly as characteristic sign is given by the voice. When the stethoscope, with its stopper in, is applied to the diseased part, the voice is heard to resound there in a tone modified, as if speaking through small tubes. The voice does not, as in pectoriloquy, appear to enter the tube of the instrument; and the sound of the voice is not heard in distinct words, but in notes of various continuance, not always synchronous with the words uttered by the mouth; and the intervals are often alternated with what may be called whiffs of bronchial respiration. It is obvious that the extent and intensity of these sounds must greatly depend on the number and size of the bronchial tubes in which they are heard. They are therefore most distinct when the hepatization occupies the summit or the neighbourhood of the root of the lung, and extends to the surface. On the other hand, when the surface or the centre alone is hepatized, these signs may be altogether wanting.

The sound on percussion is now, and in the subsequent stage, perfectly dull; and, if a considerable portion of the lung is hepatized, it may be perceived by the sight or feel, that the corresponding portion of the chest partakes imperfectly in the respiratory movements.

The sputa sometimes continue rusty and glutinous, but more commonly become mucous or serous, or cease altogether. When hepatization takes place, the fever is generally somewhat lowered, and the pulse loses strength; but it remains quick, and is often running or wiry; the dys-

pnœa is unabated, the face is sometimes patched with red, and in the worst forms shews some lividity. The disease, when extensive, often proves fatal in this stage, without going on to suppuration.

In the third stage the diseased lung becomes infiltrated with a purulent matter, which is generally consistent at first, but soon acquires the liquidity of common pus.* In this stage the bronchial respiration and vocal resonance usually cease, and are sometimes supplanted by a gurgling mucous rhonchus, indicating the presence of a liquid in the principal bronchial trunks. The expectoration is dirty, mucous, or purulent; in scorbutic persons it is a brown serous liquid. The pulse now becomes very weak and thready, and the strength generally sinks rapidly.

In the rare case of the formation of an abscess in the hepatized lung, the passage of air through the liquid will be indicated by the gurgling or cavernous rhonchus: and when the cavity has been emptied of the pus by expectoration, pectoriloquy and the cavernous respiration will be added to this sign.

Gangrene is also a rare termination of peripneumony. The gangrenous portion, being softened and ejected by expectoration, will produce a cavity which will be indicated by the usual signs of cavernous rhonchus or resonance. The distinctive physical sign of gangrene is the fœtid odour emitted from the diseased part in respiration and cough, and

* This changes the colour of the diseased lung from the red of hepatization to discoloured yellow, drab, or stone colour, which is frequently mottled with red portions in the second stage, and with the black pulmonary matter. Thls is called by Laennec, *hepatisation grise*, as it retains the weight, granular texture and compactness of hepatization; and by Andral, *ramollissement gris*, because a slight pressure reduces it to a thick purilage. In a more advanced degree, the colour is of a straw, or sulphur yellow, owing to the greater quantity of liquid pus; the texture is considerably destroyed; more rarely, in certain parts, it is entirely so, constituting a kind of diffused abscess, which has been known to occupy a considerable portion of the lung. Circumscribed abscess is still more rare.

the expectorated matter is also extremely fœtid. This change is usually attended by a collapse of the features, and great prostration of the vital powers.*

Thus far we have traced peripneumony in the changes in the pulmonary textures, as indicated by its pathological signs. The severity of the case may be judged from the extent of the disease, and the advances which it has made.

It is in the first stage of inflammatory injection that auscultation proves pre-eminently useful, in assuring us of the existence of a disease that no other symptoms could discover. The presence of the crepitant rhonchus may be taken as a warning to resort to energetic antiphlogistic measures, which in this stage will seldom fail in arresting its course. The disappearance of this sign, and sometimes the presence of the bronchial respiration and resonance, announce the increasing danger and progress of the disease, as they indicate its advance to the second stage. The diseased structure is, however, still susceptible of a return to the healthy state, and the view which we have taken of the morbid anatomy of this stage suggests, in addition to means directed against the inflammatory orgasm, the important advantage with which sorbefacients, such as mercury and tartar emetic, may be used. Occasionally the passage of the disease to the third stage may be inferred by the substitution of a mucous rhonchus for the bronchial respiration; but there is often no sign distinctive of purulent infiltration. The duration of the disease, and the nature of the expectoration, may often lead us to suspect it,

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* The anatomical characters of grangrene of the lung are various. The colour is sometimes like that of a simply engorged lung, with a greenish tint. Sometimes it presents a greenish-brown, or a dark-brown aspect. In its progress the gangrene produces a softening and complete deliquescence of the pulmonary tissue; but the sphacelic fœtidity is the characteristic sign. It is sometimes found combined with the suppurative stage, in other cases in the midst of a hepatized lung; and Chomel thinks that the first stage may, under the septic influence of the effluvia of sewers, pass immediately into a state of gangrenous softening.

and would render our diagnosis proportionately unfavourable. Recoveries have however been effected, not only from this state, but even from gangrene, by the sloughing of the dead matter, and the formation of a cavity which has afterwards become cicatrised as an abscess. The chances in favour of this rare and happy event will be in proportion to the small extent of the disease, and the functional strength of the subject.*

The resolution or retrogression of peripneumonic inflammation, is attended by a succession of the same physical signs that marked its progress, but in an inverted order. Thus, in a hepatized spot, where no sound of the ingress or egress of air has been heard, or perhaps only a bronchial respiration, a slight crepitant rhonchus begins to be distinguished at the end of each inspiration, apparently produced by the air again gaining a straitened admission through a few of the bronchial tubes, whose calibres have been partially restored by the reabsorption of matter effused round their parietes. This sign increases in intensity as the resolution proceeds; the bronchophony and bronchial respiration are first increased by the free passage of air through them, but subsequently diminished as the lung reacquires its spongy texture, and becomes a worse conductor of sound.† After a while the natural respiratory murmur is heard mixed with the crepitant rhonchus, which,

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* For admirable delineations of the anatomical appearances of this, and other diseases described in this work, see Parts 1 and 2 of Dr. Hope's coloured Illustrations of Morbid Anatomy, which the student will find a faithful and valuable key to nature.

† A hepatized lung in progress towards cure becomes paler and moister; if it has been solid for several days, the restoration of the vesicular structure is partial and irregular, and it continues for some time firmer and less elastic than natural. The signs of restoration from the third stage are a removal of the yellow pus, and the substitution of a more liquid serum for the pus. This afterwards becomes drier and the texture vesicular; but for a long time the lung retains a dirty yellow or greenish colour.

from the thinner quality of the bronchial secretion, is less dry and regular than in advancing pneumonia, and gradually becomes mucous, or sometimes sub-crepitant, from an atonic œdema succeeding to the inflammation. As the texture becomes more permeable to the air, the natural respiratory sound increases as the rhonchus diminishes, and the healthy function of the lung is thus gradually restored. But here again the signs obtained by auscultation are invaluable, as they alone indicate with certainty the absence of the disease. The dyspnœa may have been removed, the cough may have ceased, the expectoration may have become simply catarrhal, the pulse nearly natural, and all febrile symptoms abated; and yet the auscultator detects the lurking disease in the persistance of the crepitant rhonchus; and as long as this continues, a slight exposure to cold, or an irregularity in regimen, may cause a relapse, which, in a subject already reduced by depletion, may be more difficult to cure than the original disease. The subcrepitant rhonchus, without any attendant signs of fever, often ceases on the exhibition of a mild tonic joined with an expectorant.*

I have here described the access, progress, and cure of pneumonia in its general well-marked course; it will now be necessary to advert to certain varieties in the signs produced by situation, extent, and complication of the pneumonic inflammation.

When the inflammation occupies the central part of the lung, and particularly of the base, without extending to the surface, its signs may be beyond the reach of the ear, or discernible only by a very careful examination, hence the necessity of attending to the sputa and other indications. When, however, the inflammation occupies a considerable portion of the organ, the signs are very evident, and the sound of the

* For an account of the treatment of pneumonia, see my article before quoted.

respiration in the healthy parts is much louder than usual, and is called *puerile* from its resemblance to the noisy respiration of children, or *supplementary*, from its being increased to supply the defective entrance of air in the diseased parts. The intensity of this supplementary respiration will depend, besides, on the extent of the disease, on the natural activity of the respiratory function, the want of breath, so different (as we have before indicated) in different individuals.

Percussion gives no indication when the inflammation is central; and it requires much practice to detect by it a small circumscribed inflammation on the surface. Its indications are always doubtful at the posterior and lateral margins of the lungs, on account of the vicinity of the abdominal viscera.

M. Andral has remarked, that in the complication of pneumonia with catarrh, the loud mucous rhonchus of the latter so completely obscures the crepitant rhonchus of the former disease, that this may escape detection; but Laennec was of opinion that there are few cases in which the practised auscultator cannot distinguish the presence and seat of both symptoms.* On this point I would admit as true, that the ear, by practice, acquires a great facility in separating, and listening to a single sound from amongst several others, perhaps superior in loudness, as we are habitually accustomed in the din of a city to distinguish and be attentive to each of the multitude of sounds of various intensity that beset our ears:† but the safe and useful signs of auscultation are not

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* When mixed with other rhonchi, the crepitant rhonchus is best distinguished at the end of each inspiration, that being the part of respiration the most purely vesicular.

† We effect this, I apprehend, by the voluntary tension or relaxation of the tympanum, by which it is made more susceptible of the vibration of one particular sound, by being brought in unison or at least in harmony with it. Thus discordant sounds, or those not separated by harmonic intervals, are easily distinguished, but harmonic sounds being blended with each other, are with more difficulty separated, and this difficulty is in proportion to the perfection of the harmony. This fact is

such as are to be gained by such refined powers; and it is better in such cases to depend rather on the general symptoms, particularly the state of the expectoration; which, when even a slight pneumonia is added to a bronchitis, will seldom fail to exhibit in some points a rusty tinge.

The curious modifications of pneumonia, resulting from its complication with considerable degrees of pleurisy, will be noticed after that disease.

Section II.—*Emphysema of the Lungs.*

The affection of the pulmonary tissue that generally goes by this name, is rather a dilatation of the air cells than an effusion of air, as in emphysema of the surface of the body: the latter form, however, does occasionally present itself in the lungs in what is called *interlobular emphysema*. To understand more fully the nature of the alteration constituting pulmonary emphysema, it will be requisite to study the manner in which it is produced.

In cases of chronic catarrh, particularly of the dry kind, the small bronchial ramifications become so obstructed by the swelling of their membrane, or by the secretion of a viscid mucus, that the air can only be forced through them into the vesicles by a considerable effort. Now, as in ordinary respiration, the inspiration (a muscular effort) is more forcible than the expiration, which is principally effected by the elastic force of the cartilages of the ribs, and the weight of the abdominal viscera, the former may prove sufficient to overcome the obstacle to the introduction of air into the

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of importance in auscultation, the indications of which may be obscured by similar extraneous sounds. Thus a tinnitus aurium in the auscultator, or the rustling of the clothes of the patient, may prevent the respiratory murmur from being distinguished, whilst a sibilant rhonchus may at the same time be heard with its usual force.

vesicles, while the latter is inadequate to effect its expulsion. Successive portions of air, expanding by the increased temperature, are thus introduced and incarcerated in the cells, which are thereby kept in a state of continual dilatation. This, the mode assigned by Laennec, is, perhaps, a principal cause of the dilatation of the air-cells; but I would suggest that other causes co-operate, and other changes are produced, before the emphysema becomes permanent.

The forcible action of the expiratory muscles in coughing will exert a pressure on the dilated air-cells. This pressure may overcome the obstacle in the bronchi, expel the air, and restore the cells to their natural size. But the obstruction may have increased, and then the pressure will expand the cells in the direction of the adjacent yielding tissue. The dilated cells will thus encroach upon the adjoining healthy tissue, and cause its obliteration (a new obstruction in the bronchi), or the rupture of its cells. Add yet another cause, which may occasionally act, and we shall have found explanations enough of the frequent occurrence of a disease, the very existence of which has not, till of late, been suspected. In dry chronic catarrh, the general starting point of emphysema, small particles of viscid mucus form a kind of moveable obstruction, which, falling into a bronchial ramification, instantaneously and effectually plug up the tube. Now, suppose this to happen in a tube at the termination of an expiration; inspiration takes place, but this pellet of mucus acts as a valve, preventing the entry of air into those cells supplied by this tube, the consequence is, that the air in the surrounding cells presses in to fill the vacuum, by dilating or rupturing their membranous tunics.

Other circumstances, producing partial pressure and obstruction among the air cells of the lungs, such as miliary tubercles, may in a similar way lead to the developement of emphysema.

M. Andral has, with his usual sagacity, pointed out another

cause which may give rise to a state of the lung resembling emphysema; namely, the atrophy resulting from disuse. Thus in old persons, where the respiratory process becomes less extensive, or in portions of lung to which, from obstruction, the air has but imperfect access, the septa of many of the cells become attenuated and absorbed, and many minute vesicles are thrown into few coarse ones; but I would remark, that this is rather an anatomical than a pathological state, in as much as it does not constitute well marked disease, as the other kinds of emphysema of the lungs.

Such a variety of causes, acting and re-acting upon each other, tends to produce this organic derangement. How strikingly does this prove the exactitude and perfection with which the machine must act to preserve health, since so slight a deviation may entail such disorder; and how wonderful that the equilibrium is not more frequently lost! Besides the simple dilatation of the air vesicles, there appears to be sometimes an intervesicular emphysema, which causes the occlusion of some bronchial branches. To this, and to an increased rigidity of the tunics of the dilated cells, is to be ascribed the tense elasticity so remarkable in an emphysematous lung;* hence, too, the incapacity of the lung to perform its function with effect, so that persons thus affected have more or less habitual shortness of breath. This incapacity is also manifested during life, by the absence or diminution of the respiratory sound in the part. This leads me to consider the physical signs of emphysema.

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* The ordinary appearance of an emphysematous lung is a remarkable coarseness of the vesicular texture as seen through the pleura; it is also elastic, lighter, and less crepitant than usual, and does not collapse. In a greater degree the enlarged cells look like the vesicular lungs of cold-blooded animals, and occasionally raise the pleural surface of the lung into rounded inequalities. Single vesicles, like fish bladders of various sizes, sometimes project from the surface or margins of the lung, in some instances attaining an extraordinary bulk.—*See Dr. Hope's Morb. An. Part* 2, *and Cyclop. Art. Emphysema.*

It not unfrequently happens that emphysema is present without producing any other signs than those of dry catarrh or asthma; namely, a diminished sound of respiration, with slight sibilant or mucous rhonchus, and clear sound on percussion; and then the permanency of the disease can alone serve as a distinction. But if the emphysema be more extensive, it will give to the chest an unnaturally rounded form, with increased intercostal spaces, and greater resonance on percussion. If one lung only be affected, the corresponding side alone will present this appearance: it will be larger than the other, and emit a clearer sound on percussion. The stethoscope may discover from time to time a dry crepitation of a peculiar kind, and which pretty closely resembles the sound produced by inflating forcibly the cellular membrane of meat. This sound, which is by no means commonly present, is chiefly caused by the motion of air in the intervesicular texture, or particularly under the pleura, during the respiratory movements, and is clearly the same as that which may be produced by pressure on subcutaneous emphysema. The dilated vesicles themselves, are, however, sometimes its seat, and I apprehend, in this manner. Some of the obstructions before alluded to, prevent the air from entering into some portions of the tissue, until a certain extent of inspiration has dilated the tubes to a particular point; it then rushes in, by expanding quickly the dry and flaccid cells, producing the sound in question. It is called by Laennec the *dry crepitant rhonchus, with large bubbles,* but the name is objectionable because there are no real bubbles, and because it essentially differs from the crepitant and all other rhonchi. *Emphysematous crackling* would be a more distinctive term. More rarely, the sound resembles the friction of a pully, or that of two pieces of leather rubbed together, and this is usually confined to inspiration. I have sometimes heard these sounds produced not only by the action of respiration, but also by the impulse of the heart.

They plainly result from the friction of the projecting portions of elastic emphysematous tissue against the chest, or against each other.

The expectoration is usually that of dry catarrh, but often more liquid, and of a dirty grey colour.

Emphysema of the lung, as it commences gradually, and proceeds slowly, is not attended with any immediate danger; but it produces an habitual dyspnœa, which incapacitates the body for exertion, and which various temporary causes may convert into severe and oppressive asthma. It also renders its subjects obnoxious to serious, and even fatal, effects from contingent pulmonary disease, which in a healthy lung might be borne with comparative impunity.

Interlobular emphysema rarely accompanies the last affection. More frequently it occurs separately, and is caused instantaneously by violent straining, or by some analogous exertion of the respiratory organs. As its name implies, it consists in an effusion of air into the cellular tissue, between the lobules composing the lobes of the lungs, and under the investing pleura. This emphysema causes a slight elevation on the surface of the lung, from the spaces which it has made between the lobules; and this elevation rubbing against the costal pleura in the motions of respiration, causes a stepitus, which I have already compared to that produced by rubbing together slowly and forcibly two pieces of leather. It is usually most perceptible at the end or acme of inspiration, but may accompany both inspiration and expiration, and then is sometimes heard in such regular jerks, that it resembles the steps of a person mounting and descending a ladder, and is called by Laennec, *friction of ascent and descent.* The impression conveyed to the ear is exactly that of a body rubbing along the ribs, rising in inspiration, and descending in expiration. The friction likewise often communicates a corresponding vibration to the thoracic parietes, which may be felt by the

hand. The patient himself is sometimes sensible of a kind of cracking in his chest. Interlobular emphysema may produec at first some difficulty of breathing, but rarely to a serious extent, and is spontaneously cured in time by the gradual absorption of the air.

Section III.—*Œdema of the Lungs.*

This is, properly speaking, a serous effusion in the interstitial tissue between the air-cells, and vascular rete, by which these are connected together. When contained, however, by membranes of such extreme tenuity, it is not surprising that some serum should, by transudation, pass into the air-cells themselves.*

Now this liquid, by swelling up the interstitial texture, so presses on, and partially obstructs, the smaller bronchi, that the air passing through the liquid contained in them produces a kind of humid crepitation, like that heard on approaching the ear to a liquid in gentle effervescence, as bottled cider, or ale, when freshly poured out of the bottle. This is the *sub-crepitant* rhonchus. It differs from the crepitant in the bubbles seeming less regular and distinct, and more humid, but it must be considered different only in degree; for the two pass by insensible gradations into each other. Its most usual seat is in the inferior dorsal, lateral, and inframammary regions. The respiratory murmur that is heard with this rhonchus is feeble, particularly in comparison with the energetic action of the respiratory machine. The resonance of the

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* Accordingly it is so found on examination after death. An œdematous lung does not collapse; on opening the chest, it feels weighty, and pits on pressure, but is still crepitant. Its vesicular texture is less perceptible than usual. When cut into, it exudes a clear yellowish serum, scarcely frothy, which appears to proceed from all parts equally. It is, however, highly probable, that it is secreted in the interstitial texture, which is internal, and therefore serous, and not from the membrane of the air-cells, which is mucous.

chest on percussion is often not much diminished, but it is distinctly so where the dyspnœa is oppressive, and the œdema profuse and extensive. The expectoration is usually copious, consisting of a slightly viscid, colourless liquid. This, when present, will distinguish the disease from the first stage of peripneumony; but sometimes there is little or no expectoration, and then the diagnosis must be drawn from the general symptoms, the absence of fever, and the progress of the case, as the physical signs are so nearly the same.

Œdema of the lung is rarely idiopathic. It most frequently accompanies organic diseases of the heart of long duration, and humoral catarrh, in which cases it is often the immediate cause of death. If often co-exists with the general anasarca which sometimes succeeds to febrile affections, particularly the exanthemata, and is the cause of the dyspnœa sometimes occurring after scarlatina, rubeola, &c.

There is one complication of œdema which renders it very difficult to recognise, namely, with emphysema of the lungs. When this is present, the sound of respiration is so obscure that it is difficult to recognise any other sign than an occasional sibilation, whilst the sound on percussion is very good. A forcible inspiration after coughing, or retaining the breath for a while, will, however, frequently discover the disease, by rendering audible the subcrepitant rhonchus.

It is always important to be able to discover the presence of œdema in the lungs, for, although usually a consequence of other disease, it is always to be considered a principal object of treatment.

Section IV.—*Pulmonary Apoplexy or Hæmorrhage.*

This appears to consist in the effusion of blood into the parenchyma and vesicular structure of the lung. Whether this effusion is always in consequence of the rupture of vessels,

or is sometimes simply an hæmorrhagic exudation, has not been distinctly ascertained; but the former cause would seem better to explain the suddenness and quantity of the hæmorrhage, and the circumscribed form of the lesion. It is, however, highly probable that the textures are, in most cases, softened or altered by disease, before the rupture takes place. M. Andral considers the hæmorrhage to be simply from the bronchial membrane, and that the spots of pulmonary apoplexy are merely collections of the exhaled blood coagulated in the vesicles and fine bronchi.

The blood effused may coagulate before it reaches any large bronchial ramification, and, in that case, there will be no hæmoptysis, but more commonly the reverse happens; more or less blood is spit up, or, if in large quantity, more properly, as Laennec observes, vomited; for the discharge is produced by a convulsive action of the abdominal muscles, exactly after the manner of vomiting. At length, however, the hæmorrhage is checked by the formation of a coagulum, which, pervading completely a circumscribed portion of the pulmonary texture, constitutes the *hæmoptoic engorgement* of Laennec.*

When a point of the lung is thus affected, the respiratory murmur will, of course, be no longer heard there; and if the engorgement be of large size, there will be a corresponding dulness of sound on percussion, in that part of the chest. The compression of the tissue immediately around, and the presence of a bloody serum in the vesicles, proceeding from the

* These spots of pulmonary apoplexy, which generally are from one to four inches in diameter, are of a dark blood, or brown red, colour, sometimes spotted with lighter granules; the coarser parts of the pulmonary structure alone can be distinguished in them, but even these partake of the same tinge. Unless they are very recent their consistence is firm, and they contain little or no serum. Sometimes there is an obvious detritus in the centre, formed of grumous blood alone, in which no texture can be traced; and in some formidable kinds of pulmonary apoplexy, which prove fatal by hemorrhage, the whole affected portion of the lung is lacerated and broken up by the effusion of blood into it.

coagulum, occasions a crepitant rhonchus, which is therefore heard around the spot where the respiration is inaudible. This symptom, however, seldom continues long after the commencement of the disease, but, once heard, it distinguishes it from a simple bronchial hæmorrhage.

During the hæmoptysis, as in the latter disease, the blood in the bronchi causes a bubbling rhonchus, which Laennec distinguishes from that produced by mucus, in the bubbles bursting in more frequent succession. The stethoscopic symptoms with hæmoptysis are amply sufficient to indicate the nature of the disease; but when hæmoptysis is not present, the sputa and general symptoms must be referred to, to establish the diagnosis between it and peripneumony.

The extent of the apoplectic engorgement, rather than the quantity of blood brought up, indicate the degree of danger to be apprehended; for a large quantity of blood may pass through a small rupture in the pulmonary tissue, and unless this be so great as to threaten inanition, which is not often the case where prompt measures are resorted to, the lesion is more of the nature of a simple wound than a change of structure. It is where the spots of apoplectic engorgement are numerous or large, that we have to apprehend some ulterior cause than a simple rupture; and whether this be a more frangible state of the pulmonary tissue, depending on the presence of miliary granulations and other precursors of tuburcular formation, or be some modification peculiar to the disease called pulmonary apoplexy, it must be viewed as partaking of the danger of the general or constitutional alterations of tissue, that are very little within the control of medicine. Dr. Carswell has shewn that crude tubercles are sometimes deposited so as to press on and obstruct the pulmonary blood-vessels, and they may thus cause congestion, and ultimately rupture. Hypertrophy, and other diseases of the heart which cause an undue congestion of the pulmonary vessels, occasionally give rise to pulmonary apoplexy, and hæmoptysis.

CHAPTER III.

DISEASES OF THE PLEURA.

Section I.—*Pleurisy.*

Inflammation of the pleura, when severe, is early accompanied by a serous effusion, and it is this circumstance that gives to the disease its most remarkable pathological and diagnostic characters.

Exquisitely marked, as this disease is described to occur, by the acute pain of the side, with fever, catch or oppression of the breathing, hard pulse, decubitus on the affected side, dry cough, &c., there are few practitioners who have not proved the fallacy of each of these symptoms; and, as we shall presently point out, the auscultator finds but uncertainty in them all.

At the first attack, before there are any signs of effusion, if the pain be very acute, the sound of respiration will be somewhat impaired on the affected side. This is, however, merely in consequence of the respiratory action being restrained on that side by the pain, and is equally observed in pleurodyne. When the pain is very acute, it almost entirely restrains the motion of the ribs of the affected side, which may be perceived on inspection, or by the hand applied, to be nearly immobile, the respiration being carried on by the other side, and by the diaphragm. These circumstances are reversed when the diaphragmatic portion of the pleura is affected.

The secretion of liquid by the inflamed pleura generally commences from almost the beginning of the attack, and in-

stead of being, as commonly supposed, a termination of pleurisy, it is a concomitant, or rather, a part, of the disease; as the secretion from the bronchial mucous membrane is of bronchitis. It is, however, the opinion of some pathologists,* that, as in the latter disease, there is a period of diminished secretion, or a peculiarly dry state of the membrane at the commencement of a pleurisy, and that this defect of the usual lubricating liquid occasions a friction between the pulmonary and the costal pleura, which is sometimes sensible to the hand, and more frequently to the ear, applied to the chest. Hence, a sound of friction, like that of rubbing together two pieces of leather becomes a sign of incipient pleurisy. I have certainly heard this sound; and although it cannot be considered pathognomic, since it occurs also in emphysema of the lung, its presence may be admitted as a sufficient reason to make us suspect incipient inflammation. When effusion takes place, its first signs are obtained by percussion. The resonance of the chest is commonly diminished first in the inferior dorsal and lateral regions, corresponding to the base of the lung. As the effusion increases, the dulness of sound gradually extends upwards, and becomes more pronounced.† Some-

* Reynaud, Andral, Stokes, &c.

† The following are the appearances on dissection in different stages of an acute pleurisy: The inflamed pleura presents many points or somewhat elevated patches of a diffused redness, and a number of red vascular ramifications are likewise seen distributed under it. Different parts of the membrane are covered with a white, or gelatinous coagulable lymph, and a yellow or greenish serous or seropurulent, and sometimes sanguineous liquid is found in the cavity. This liquid, if scanty, occupies principally the lower and posterior part of the chest; but when abundant it envelopes the whole lung. The lung is found compressed, flaccid and less crepitant, in proportion to the quantity of liquid. Sometimes reduced to a size not greater than the hand of the subject, it is pushed by the effusion into a small space against the mediastinum and spinal column. In some cases the lung is bound by old adhesions and is then pushed in a different direction. When the adhesions are above, the lung is displaced upwards by the effusion; when the lower parts adhere (a very rare case,) the effusion occupies the upper part, and so on, the lung being always pressed against its points of attachment.

times the transition from the dull to the healthy sounding parts is so abrupt, that a horizontal line will exactly divide them, and this, when well marked, is a very characteristic sign. A change of position will also alter this line in a manner quite distinctive, and which can happen only in liquid effusion, the dull sound always accompanying the liquid as it gravitates to the lowest parts.

M. Reynaud has pointed out another effect of effusion, which may furnish a diagnostic sign, in its intercepting the slight fremitus or vibration which accompanies the voice in all parts of the chest.* The hand applied to a healthy chest, readily feels this general vibration; but a layer of liquid interposed between the lung and the chest, acts as a damper, and prevents the transmission of the vibration. The sound of respiration likewise is rendered more obscure as the liquid accumulates between the lungs and thoracic parietes; but the collection of liquid must be considerable before it becomes extinct.

Before this, however, another effect is produced which gives rise to its peculiar signs. The pressure of the effused liquid condenses the tissue of the lung, by which we have formerly seen it is rendered a better conductor of sound, and transmits noises, usually unheard, of the passage of the air and voice in the bronchi. But this bronchophony, and this sound of bronchial respiration, before they can reach the ear, must pass through the serous stratum between the pleuræ, and are differently affected by it. A respiration is usually heard becoming bronchial as the effusion increases up to a certain point, but then, as the bronchi themselves become pressed by a further increase, it becomes faint, and at last ceases. If the stratum of liquid is thin, the bronchophony traverses it, but, by throwing it into vibrations, is itself modified, rendered sharp and tremu-

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* See Page 35.

lous, and as if produced at the surface of the lung. The voice, therefore, instead of being as from the mouth, or even simply diminutived, as in bronchophony, resembles the tremulous bleating of a goat or lamb. This modification of the voice M. Laennec therefore called ægophony.* Its most distinctive mark is its tremulous or subsultory character. In bronchophony the natural pitch of the voice is sometimes raised, but in ægophony it is constantly and considerably so, and is thus rendered squeaking and wiry.

Now as this modification of bronchophony can be caused only by an effusion in the pleura, it may be regarded as a pathognomonic sign. But even in this case three conditions are necessary before it can be produced. 1. A certain condensation of the pulmonary tissue: 2. The presence of a thin stratum of liquid between the condensed lung and the thoracic parietes: 3. Such a proportion between the mass of this liquid and the pitch and strength of the vocal sounds, that it may be thrown into vibration by them. The necessity of this latter condition is shown in the fact that certain tones of the voice are ægophonic, and others not; some transmitted with only bronchophonic modification, and others changed to the sharp tremulous tone of œgophony. The change in the note or pitch of the voice in this instance, as well as its intermittent or tremulous character, probably depends on an alternate reduplication and counteraction of the vibrations as they pass through the thin stratum of liquid; the grosser motions into which this liquid is thrown sometimes concurring and sometimes interfering with the sonorous pulses. It may therefore be stated generally, that the tremulous or subsultory sound of the ægophonic voice is produced by successive undulations of the liquid, the result of an irregular transmission of the sonorous vibrations.†

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* From *αιζ*, *a goat*, and *φωνη*, *a voice*.

† M. Laennec considers that another cause may contribute to the production of

It may be concluded from this account of the proximate or physical causes of ægophony, that this symptom cannot usually continue for any length of time. The liquid is either so much increased that the bronchi themselves become compressed; or, it is re-absorbed, so that the cause of ægophony is removed. The latter case is indicated by a return of the natural respiratory murmur in the part, and a sonorous resonance on percussion. In the former case, all sounds are lost, and the chest sounds uniformly dull, except in a small space close to the vertebral column, against which the lung is compressed. The effusion is sometimes so rapid that a few hours duration of the disease may produce this state. There is, however, almost always one period at which the effusion unites the conditions necessary for the production of ægophony; and as the progress of the disease is slow or rapid, the duration of this period will be long or short. The situations in which it is most frequently heard may be included in a band about three inches broad, running from below the inferior margins of the scapula, in the direction of the ribs, to the sternum. It is most pure in the anterior and lateral parts, being often mixed with a natural bronchophony in the dorsal regions. Sometimes, however, it is heard in nearly every part of the affected side, the collection of fluid being but moderate. This universal

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ægophony; namely, the flattening of the bronchi by the pressure of the effusion, whereby they are converted into little reed instruments, all set a-piping by the sound of the voice. Besides that this explanation is unnecessary, I must object also that it is untenable. The reed of the bassoon and hautboy sounds only on the passage of *air* through it, and did the flattened bronchi represent it in this instance, the respiration, and not the voice, should make them sound.

That ægophony is bronchophony modified by its passage through a thin layer of liquid, and not by a smaller degree of compression of the lung, as supposed by Dr. Law, (Cycl. Pr. Med. Art. Pleurisy) is proved by the fact noticed by M. Reynaud, that if an egophonic patient leans forward far enough to make the lung float into contact with the posterior walls of the chest, the ægophony in the interscapular region becomes converted into bronchophony. (Journ. Hebdom. Dec. 1829.) M. Laennec succeeded to a certain degree in imitating the phenomenon, by applying a thin layer of liquid in a bladder over a naturally bronchophonic region in the chest of a young man.

ægophony never continues long, unless where the lung is prevented from collapsing before the increasing effusion, by old adhesions retaining it at a little distance from the costal pleura, in which case, after a time, the respiratory murmur returns, the pressure not being sufficient to exclude totally the air from the vesicles. Adhesions may, in other ways, modify the signs of pleurisy. Not unfrequently the apex or subclavicular lobe of the lung adheres closely to the costal pleura; an effusion can here never destroy the sound of respiration under the clavicle, and the same thing may occasionally happen in other parts of the lung; the sound of the respiration remaining, however great the effusion in those spots, where an adhesion protects the lung from pressure.

It sometimes happens that the pleurisy and its effusions are quite partial, being confined to the tissues between the lobes, or to a part on the surface by adhesions. The accompanying pain and ægophony, will generally characterise these circumscribed pleurisies.* If these symptoms are absent, the diagnosis will be difficult, for the same partial absence of respiratory murmur and pectoral resonance, might result from other causes; a flatness or slight prominence of the intercostal spaces, and, if the subject is thin, a perceptible fluctuation, will sometimes suffice to distinguish a collection of liquid from partial hepatizations or tubercular deposits.

Whenever the effusion is abundant, and has been rapidly produced, the respiration on the healthy side will become puerile, or supplementary. Now as the sound of this respiration is sometimes heard on the diseased side, through the liquid, it will be necessary to guard against the error of mistaking it for a faint respiration on that side. On listening

* They most frequently occur in phthisical subjects, being excited sometimes by tubercles, and sometimes by the bursting of a vomica into the pleura. The effusion is commonly purulent, and may, particularly when interlobular, be mistaken, on dissection, for an abscess of the lung.

attentively to the sound, it will be easy to perceive that it increases in intensity as the ear approaches the healthy side, and that its loudness there will sufficiently explain its source. The continuance of a real, although faint, respiration, in a space of about three fingers breadth along the spinal column, corresponding with the compressed lung, will also furnish a standard of comparison, by which the other sound may be distinguished. Besides these tests, the ear, by practice, acquires the power to distinguish at once a sound faint by distance, and one faint in origin.

Another important physical sign, that indicates an abundant effusion, is an enlargement of the affected side. This, although when measured from the spinous process of a vertebra to the sternum, it seldom exceeds an inch, or an inch and a half, is very obvious to the eye: an observer, placed opposite or behind the patient as he sits up, or stands naked, may detect the want of symmetry of even less than half an inch in extent. Mensuration of the chest, as described in a former section, will more exactly indicate the difference. Another effect of a large collection of liquid in the chest, is to displace the viscera and thinner parietes in a remarkable manner. Thus the intercostal spaces are less depressed than on the other side, or may even protrude, and this in thin subjects is a very distinctive sign. An effusion on the left side will often displace the heart, and make it pulsate under, or even to the right of, the sternum. The liver will be pushed downwards by a large collection of fluid on the right side. These signs are important, because they distinguish this disease from pulmonary hepatization, a state very liable to be mistaken for pleuritic effusion, but which produces no such displacements. A useful criterion of this kind, drawn from percussion on the sternum, has been pointed out by Dr. W. Stokes: a copious effusion on one side will displace the sternal mediastinum, and render the whole sternum dull on percussion. A hepatized

lung, on the other hand, will not encroach on the mediastinum, but lying under one half of the sternum will render that half dull, whilst the other half will remain resonant as usual. These distinctions, and the noisy bronchophony in the scapular, lateral, and mammary regions, in hepatization, will suffice to form a diagnosis between the two morbid states in question.

Now, when the disease has arrived at this state of copious effusion, having been attended with more or less pain of side, dry cough, dyspnœa proportionate to the rapidity of its course, and the usual febrile symptoms of acute inflammation, all these symptoms may disappear; the pain and cough gone, the pulse nearly natural, the appetite returned, and the dyspnœa but slight, felt perhaps only on exertion; in short, the patient may appear convalescent, and yet, strange to say, one side of his chest is full of water! In this state, if he be kept quiet, and limited to an antiphlogistic regimen, with mild mercurials and diuretics, sometimes with blisters on the affected side, there will be the symptoms of a slow and gradual absorption, which we shall presently describe; and the patient may in time be restored to real health. But if, relying on his sensations, and deceived by a false and illusory feeling of health, he discards medicine and returns to an active life, with a full and generous diet, the consequences may be disastrous. Either, the acute disease may be rekindled from its smothered state, may excite an increase of circulation incompatible with the crippled state of the organs, and thus produce effusion in other parts, and consequent suffocation; or, the pleurisy may continue in a chronic form, perpetuating the effusion, in the form of empyema, becoming a part of the habit, and so altering the condition of the membrane by the deposition of tubercular or other accidental productions, that it becomes incapable of absorption, or of return to its healthy condition. Hence organic disease will run its resistless and irremeable

course, wearing down the strength by hectic, and wasting the body by atrophy, until life, unable to dwell in such a dilapidated tenure, ceases to hold it from its kindred earth.

Such may be the fatal consequences of relying on fallacious general symptoms. Let us seek in auscultation and percussion, the beacon to warn us of the latent danger, and to guide us to the employment of means to avert it. The absorption of the fluid is indicated by the gradual return of the respiratory murmur; first, in those points where it had persisted latest; afterwards in others; and last of all in the parts where the accumulation had begun. It is very faint at first, and becomes stronger in time; but, generally, a very long period is required to bring it on a par with that of the healthy side: sometimes so slow is the absorption, that many months are required to dissipate a collection of fluid that was formed by a pleurisy of a few days' duration. In other instances, however, the absorption is nearly as rapid as the effusion, and in these cases a returning ægophony also announces the diminution.

As the absorption proceeds, there is sometimes heard a sound of friction, like the leather-creaking which occasionally accompanies the dry stage of pleurisy. This is produced by the approximation and habitual friction of the pleuræ, the surface of which is covered with a false membrane, rougher or drier than usual. This sound of friction seldom continues long, for the surfaces soon become united by adhesions.

When the effusion has remained long, the ægophony seldom returns; for, from the long continued pressure, the bronchi, in which it is produced, lose their elasticity, and do not immediately recover a sufficient calibre to cause that resonance of the voice which constitutes bronchophony. On account of the same loss of elasticity, and slow restoration of aereal texture in the lungs, the chest never recovers its sound on percussion proportionately to the return of the respiratory murmur.—

Sometimes, when a contraction of the cavity takes place, as afterwards described, the affected side remains nearly as dull as ever, after the complete absorption of the liquid. But when the complaint has been of short duration, the sonorousness returns perfectly, although more slowly than the respiratory murmur.

In the double pleurisy, where both sides are simultaneously affected (a very fatal form of the disease), the indications given by percussion are less certain; for both sides sounding equally ill, the standard of comparison is lost; but the upper parts of the chest remaining sonorous, and the exact demarcation between these and the line of the effusion, will still characterize the disease; and by these signs, auscultation will assist us to detect the cause of the oppressing dyspnœa, which, unless the most energetic measures are employed, may soon end in suffocation.

The danger in acute pleurisy depends on the rapidity and quantity of the effusion. If the ægophony continues, it is a proof that the effusion is moderate, and nearly stationary, which promises an easy cure. In leucophlegmatic habits, the effusion is usually very abundant, and its absorption slow; hence these present the most unfavourable cases. After the system has sustained the immediate effects of the effusion, a transition to the insidious chronic state must be the source of apprehension, and this as long as there are no signs of a reabsorption. If, from the return of the respiratory murmur, or of ægophony, to a part in which it had ceased, it is found that the diminution has commenced; then, if no fresh excitation be applied, a gradual cure may be expected with confidence. But if after the cessation of the acute symptoms, the absorption not having begun, or being arrested in its course, a slight fever rekindles, generally with evening exacerbations, attended with more or less cough and mucous or mucopurulent expectoration, then it is to be feared that the disease has taken the inveterate and intractable habits of the chronic

disease, and the prognosis becomes so much the more unfavourable.

Let us bestow some attention on the changes that accompany the different terminations of pleurisy.

The serous effusion is not the only product of the pleuritic inflammation, although it is the only one that is indicated by physical signs. A plastic or coagulable lymph is at the same time exsuded by the inflamed membrane, and becomes the basis of a variety of products. Now as this takes on different forms, according to the modifications of inflammation, it will be useful to enquire whether this variety may be referred to some general pathological law.

The secretory action of the vessels of a part in health is twofold: first, the *action of assimilation or nutrition*, by which the tissue is perpetuated in its kind, and preserved, notwithstanding the operation of an opposite power, absorption; and secondly, the *external secretion*, or action by which some parts of the blood are separated from or out of the tissue. The first is the slow and more perfect act of living structure, varying in the different elementary textures of the body, but always preserving their individual identity. The latter, in its simplest kind, is little more than a mechanical transudation of the more watery parts of the blood, such is the secretion of serous and cellular membrane; but when modified by complicated structure and vital* energy, the pro-

* Shall I say *nervous?* So the analytic experiments of Sir B. Brodie, Dr. Philip, &c. seem to indicate; but, perhaps, not yet in a manner sufficiently decisive. To clear this matter, labour and thought yet are wanted. Still let us guard against the error of considering the term vital as explanatory, or as implying an ultimate thing, not to be analysed or resolved into simpler. I would rather employ it as a generic term, comprehending, not only the unknown power operating in the living body, and *hitherto* unexplained by any physical laws; but also the operation of physical laws through a *mechanism* or *organization*, peculiar to living bodies. The progress of physiology, which has already transferred many living actions from the former to the latter class, may ultimately penetrate the mist that obscures the remaining *terra incognita* of vitality, and find there nothing so unearthly as not to be reducible to the chart of animal physics. Be it understood, I speak here of organic actions only. The sensorial or animal powers require a distinct consi-

duct of this secretion is often more peculiar and characteristic than that of the assimilatory of the same texture: this is instanced in the kidneys, liver, and other excretory organs.

Such are the healthy secretory functions of all tissues. Now if the vascular action of the part be increased in different degrees, these functions will be proportionally exalted. The first effect will be, an increase in the external secretion; in great measure the mechanical result of an augmented flow of blood. The same degree of increase will also in time affect the slower process of nutrition, causing simple hypertrophy, or increase of natural substance. These changes, although frequently produced by disease, do not differ in nature from the physiological phenomenon of growth. On a further increase of vascular action, however, the effect becomes more peculiar to a pathological state. Besides a greater change in the external secretion, the tissue itself becomes more or less thickened or altered, being at first softened, but if the cause continue long, afterwards indurated; the one being the effect of acute, and the other of chronic inflammation. Now what are these but modifications of the nutritive or assimilatory secretion? It is augmented; new matter is deposited in the tissue, first in a liquid state, diminishing its molecular cohesion; hence the softening of acute inflammation: the cause continuing for a longer space of time, and no re-absorption taking place, the new matter, according to its accustomed process, becomes solid, and uniting in firm molecular adhesion with the tissue, increases its solid substance: hence the induration from inflammation of longer continuance. Now this change of nutrition differs from simple hypertrophy, or growth, in the more condensed arrangement of the solid particles, and

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deration, and then appear like the properties of matter, too simple for analysis; like geometrical lines or points, too elementary to admit of definition. Any explanation, therefore, applied to these can be but sophistry, for if mental, it would be to argue in a circle,—if material, *ignotum in ignotius.*

"Nec scire fas est omnia."

presents the first instance of structural disease, peculiarly pathological. Let the acute vascular action be yet increased further, there will be an overflow of the nutritive secretion, which, no longer confined to the texture, will now become external, and will be effused with the liquid secretion, in form of lymph or pus, varying according to the degree of inflammation, and the changes already induced. The matter thus effused is fibrine or albumen, the coagulable parts of the blood; for as Berzelius, and Prevost and Dumas have shewn, these are but varieties of the same substance. Enough in the abstract;—now let us apply these general pathological laws to the case before us.

The pleura is a simple lamina of membrane, so thin, and of context so simple, that it is scarcely susceptible of thickening. As soon, therefore, as it is sufficiently inflamed to augment the nutritive secretion of the vessels, this soon overflows outwardly; and with the increased external, or serous exhalation, an *albuminous exsudation* takes place on the surface of the membrane. This exsudation is the basis of all the factitious membranes, accidental productions, and diseased formations found in the cavities of the pleura.* Let us now examine how these varied products result from different degrees or modifications of inflammatory action.

In the acute form of pleurisy, the inflammatory orgasm is

* I consider inflammation of a serous membrane to be the most simple of any. In more complicated textures, its varied effects arise from the difference of structure. Why does an inflamed mucous membrane throw out pus and not coagulable lymph? The reason is obvious. Its internal or nutritive secretion being increased beyond the degree causing simple hypertrophy, an interstitial effusion takes place, which causes a thickening of the membrane, and mechanically restrains its further effusion; so that if the inflammatory orgasm continue, the external overflow of the nutritive matter will be in a disintegrated form, in separate particles, or globules, and far less susceptible of consolidation and organization. Although, however, the difference in structure may thus modify the products of inflammation, the intensity of the orgasm of the vessels, as well as the nature of the blood which they convey, are other causes of variety to be taken into account; and we incline strongly to the belief that all these are sufficient to produce every kind of new or accidental tissue.

intense, and the nutritive secretion increased to the utmost; the albuminous exsudation is abundant, easily organizable, and capable of nearly as high vitality as the membrane that secreted it. In fact, the vessels under the influence of inflammation produce at once, what in health they are required to do slowly, and to supply decay—the materials of their own membrane. The simultaneous exhalation of serum separates the lymph thus thrown out into thin laminæ, and these becoming vascular, in the manner described by Sir Everard Home and Dollinger, are further perfected into a number of new serous membranes, forming bands, or a network of adhesion between the pleuræ that produced them.* These adhesions, the result of a decidedly acute inflammation, are so exceedingly common, that it is rare to open a body without them. Being loose and mobile, they often produce little or no inconvenience or impediment to the respiratory motions.

It is different with another modification of the disease, which Laennec has denominated hæmorrhagic pleurisy. The albuminous effusion has then mixed with it more or less of the colouring matter of the blood. Now this impedes the process of organization, for the colouring matter in no way contributes to the formation of texture, and must be absorbed before the organization can be perfected. As the new product is of slow generation, so is its vitality of a lower degree; the excitation or orgasm of the vessels having been much diminished before it could be extended to the new sphere of action, the matrix of the new tissue. Hence there is produced, not a new serous membrane, but one of a fibrous or fibro-cartilaginous texture, of inferior vitality, and wanting the soft

* In some cases, the fluid of acute pleurisy has been found in a gelatinous state; which, on standing, will separate into crassamentum and serum, and this seems to depend on a fine cellular network of recent lymph, retaining the serum within it, as it is also sometimes seen under the arachnoid coat of the brain. This lymph, in case of cure, probably constitutes those reticulated adhesions which are occasionally met with between the pleuræ.

and yielding mobility of the serous tissue. I believe that this character in some degree extends to adhesions formed by pleurisies, slow in progress, that are not hæmorrhagic, and Dr. Forbes concurs with me in this opinion.* Now, what will be the consequences of the formation of such a membrane? The lung will be bound down by it in the compressed state to which it has been reduced by the accumulation of the liquid effusion, which, in these cases, is always abundant and of long duration. The liquid being diminished by absorption, the lung, thus restrained, can but partially recover its expanded state; and one of two things must take place to fill the void left by the retroceding effusion: either, the thoracic parietes will be drawn inwards into close contact with the diminished lung; or, the place of the effusion will be occupied by an aeriform exhalation. The first is the most common case, and constitutes the contraction of the chest so well described by Laennec.

In a subject thus affected, the contraction is discovered at once by the eye, and may be proved by a measurement, compared with the healthy side. The ribs are drawn closer together, the shoulder lower, and the muscles appear less prominent than on the healthy side. When the contraction is considerable, the person leans a little to the affected side, which causes something like a limping in his gait. The chest having on this side lost the elastic freedom of equal tension, is no longer fully resonant on percussion, and it requires mediate percussion to elicit any sound of hollow at all. The sound of respiration is, however, preserved with a diminished intensity, except in the inferior parts, where it is generally obliterated.†

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* Trans. of Laennec, 3rd Ed.

† This is, of course, because the inferior lobes of the lungs have been so much compressed, that they become obliterated, and are perfectly impermeable to the air. They appear, on dissection, flaccid, like a uniform muscular tissue, sometimes red or livid, and sometimes of a light fawn colour.

The general symptoms of hæmorrhagic or contractile pleurisy are frequently not well marked, and it is always very long in its progress towards cure. Several months are required for the dispersion of the effusion, and the full contraction of the chest. This being effected, the new fibro-cartilaginous membranes of the costal and pulmonary pleuræ come in contact; and as they are not, like serous surfaces, adapted for mutual friction, by the irritation and consequent increase of the nutritive secretion of their vessels, a gelatinous matter is deposited between them, which in time becomes a bond of union, consisting of fibro-cartilage of lower vitality, or even of real cartilage.* Before this is effected, the action of the coated pleuræ one against the other, will often occasion the leather-creaking sound of friction before described.

This adhesion may be considered a cure; for persons thus affected, although they have a shorter breath than others, labour under no habitual dyspnœa. Catarrhs, and other affections impeding respiration, will be more severe in their effects on such subjects, as they have less room for abridgement of the function. On the other hand, they may be considered more exempt from pleuritic attacks in future, as the gluing together of the pleuræ renders the pleuritic effusion impossible on that side.

I have mentioned another event of the confinement of the lung by a fibro-cartilaginous membrane, namely, the filling up the space left by the effusion with an aeriform exhalation. This mode of termination, although not noticed by Laennec

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* It is in this middle lamina that the ossific process frequently operates. In one instance, I have seen this exemplified in the formation of a bony lamina, of considerable thickness, on each side of the lung, and covering it like a cuirass. The general tendency of fibrous and cartilaginous textures, whether natural or morbid, to ossification, and the acceleration of this process by inflammation, so sagaciously pointed out and beautifully described by M. Andral, in his works and lectures, may be comprehended in the view I have attempted to give of the effects of increased determination of blood on the different secretory powers of vessels.

or any other author, has fallen under my own observation; and, I believe, is likely to occur in most cases where the hæmorrhagic pleurisy has been partial, and the effusion confined by ancient adhesions. A partial cavity is formed by the effusion, which, on its re-absorption, cannot be filled by any contraction of the thoracic parietes. It therefore becomes filled with gases which are frequently exhaled by serous membranes. This case is different, it must be remarked, from those described by Laennec, in which the pneumothorax is, as it were, active, and in which the gases themselves (perhaps evolved by the decomposition of the pleuritic effusion), and not a fibro-cartilaginous membrane, continue to keep down, by pressure, the reduced lung. The pneumothorax is here the disease, but, in the former case, a consequence of the cure. I shall return to these cases in the section on pneumothorax.

Let us now examine the effect of a third degree or modification of pleuritic inflammation, constituting what is called *chronic pleurisy*. The inflammatory orgasm, or, as we consider it, the exalted function of the vessels, although sufficient to cause an overflow of the nutritive secretion, is not adequate to extend itself by vascular communication with the organizable materials thus thrown out: these being, therefore, retained by no bond of union, become, in succession, detached from the pleura in small flakes, and mixing in great abundance with the serous effusion, constitute the liquid of *empyema*. In many cases there is a sort of partial organization in the portions or layers of lymph, which adhere to the pleura; and these seem to prove barriers to the reabsorption of the effused fluid. If an acute inflammation has preceded the chronic disease, the gradations in the organization of the lymph may often be traced by the scalpel; that immediately investing the pleura, presenting the firmness of ordinary false membrane, while the portions on the surface are in shreds, very friable, easily detached, and presenting little or no trace of organiz-

ation, the effused fluid also abounding in fragments of the same substance. In subjects of the tuberculous diathesis, the matter effused is sometimes of the curdy kind which constitutes the riper forms of tubercle.

Empyema, then, is produced by a chronic inflammation of the pleura; and is neither the result of the suppuration of the lung, as was formerly supposed, nor, in fact, is the fluid formed always real pus. The difference is, however, less than this description would at first suggest, since both consist of albuminous globules floating in a serum; but in the liquid of empyema the globules are often united in small flakes, and the serum is more abundant, both of which peculiarities may be explained by the structure of the membrane that secretes it.

In its physical signs, chronic pleurisy does not materially differ from the acute disease. The effusion is recognised by the dull sound on percussion, total absence of the respiratory murmur, the enlargement and immobility of the affected side, which are often more remarkable than in the acute disease, on account of the large quantity of effused fluid. Ægophony is rarely present; for generally the disease is either a sequel of an acute one; or, if idiopathically chronic, it has commenced so insidiously, that the effusion has exceeded the ægophonic degree before it attracts attention. The general symptoms are usually such as practitioners in this country would consider indicatory of phthisis; and joined, as it often is, with chronic bronchitis, and sometimes purulent expectoration, it is impossible, without the aid of the physical signs, to distinguish between the two diseases. In addition to the various signs of extensive effusion before enumerated, that of drawing off a drop of the liquid by acupuncture may be mentioned; and this is a measure very proper to be premised to the operation of parencentesis, both to put beyond doubt the nature of the disease, and to indicate a safe spot for the insertion of the trochar. This operation, under the guidance of the phy-

sical signs, has proved successful in a considerable number of cases, in which it was not probable that recovery could have taken place without it.*

In fatal cases, which Laennec rates at the proportion of a half, the last stage presents complications with peritonitic and gastritic disorders, and the patient dies in a state of extreme emaciation. Where the predisposition exists, chronic pleurisy often excites the secretion of tubercular matter, and perhaps also the formation of tubercles in the pulmonary tissue. It is by such peculiar predispositions or constitutional depravities, that the products of inflammation or increased vascular action are changed from new membranes and pus, to scirrhous, cerebriform, and tubercular formations. An increase in the nutritive secretion must be the basis of all these new productions; the manner in which this is modified by peculiar constitutions is involved in the mystery of secretion; but the increase is certain. Now, for an increased secretion, there must be an increased determination of blood, and although this may have but a minor share in the morbid developement in comparison with the more mysterious operation of diseased fluids or deranged nervous influence, yet as a state over which remedial agents have more controul, it is practically a point of great importance. But the local increase of vascular action may be, and generally is, conjoined with a debility of the general circulation; hence, local depletions, or still better, the counter-action of derivants and counter-irritants, avail more than the more general antiphlogistic treatment.† The most successful constitutional treatment in most of these cases, is that of a mild tonic and alterative nature, with due attention to the free action of the secreting organs, rather than any reducing or starving plan.

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* For much valuable information on this subject, see Dr. Townsend's article "Empyema," in the *Cyclopedia of Practical Medicine.*

† *See Cycl. of Prac. Med. Art. Counterirritation*, by the Author.

Section II.—*Pleuropneumonia.*

It very frequently happens that pneumonia is attended with some inflammation of the pleura; and again, in pleurisy, there is sometimes an extension of inflammation to the pulmonary parenchyma. The frequency of these combinations, and the difficulty formerly experienced in distinguishing between them, led to the use of *pneumonia* and *pleuropneumonia* as synonymous terms. We may now, however, advantageously restrict the latter name to inflammations which attack both the tissue and the membranes to a considerable extent. This complication, when nearly equal, instead of presenting a more aggravated case, rather, as M. Laennec remarks, mitigates the severity of both diseases; and this from a cause purely mechanical. The pressure exerted by the pleuritic effusion moderates the inflammatory action in the lung; and again, the lung, in some degree consolidated by the inflammatory process, not yielding to the encroaching effusion, sets limits to its accumulation. If, however, the intensity of a pneumonia is diminished by a contemporaneous pleurisy, its duration is probably prolonged; for the process of resolution is always much slower in this than in the simple case. This is because the interstitial effusion is more solid, and less mixed with the serous exhalation produced by common inflammation, and which must generally assist in the discussion of the denser products. On the other hand, a pleurisy, coinciding with pneumony, will be of easier and speedier cure, inasmuch as the effusion is less abundant.*

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* It is by inflammation, thus modified by pressure, that is produced that change in the lung, called by Laennec *carnification*. The tissue of the lung in this state has the colour and consistence of flesh, is no longer crepitant, and presents no traces of the vesicular structure. In this respect it resembles the non-granular form of hepatization, which I have described as that in which the interstitial

The signs of pleuropneumony are, as may be expected, a combination of the signs of pneumonia and pleurisy. The crepitant rhonchus will be heard in all those parts of the lung, affected with inflammation, that are not pushed away too far by effusion. It may, therefore, be looked for at the root of the lung, and all round the middle regions of the thorax; and it may sometimes be heard in other parts. Again, the ægophony, the sign of the pleurisy, is commonly to be found at the root of the lung, in the interscapular regions; and here it is generally combined with a noisy bronchophony proceeding from the large bronchial ramifications, but it may extend through the whole ægophonic region, that is from the lower margins of the scapula along the ribs to the sternum. This combination of ægophony and bronchophony M. Laennec compares to the squeaking voice of punchinello. The comparison is pretty exact, but not quite adequate to represent the impression. Besides the punchinello voice, composed of a buzz and a squeak, there is a tremulous or vibratory character in the sound, which seems alternately to approach and recede from the ear in sudden jerks. These signs, as they usually continue throughout the disease, render it very easy of recognition. Sometimes the sound closely resembles that produced by speaking with a comb covered with paper applied to the lips. But it is the vibratory or tremulous character of the sound which most constantly depends on the presence of liquid, and is therefore the surest indication of the existence of pleurisy. Any one who has attended carefully to these signs, and has examined the position of the

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plexus and tissue are the only seat of the inflammation; and this variety of consolidation, in which the ulterior effusion into the coats of the air-cells is restrained by external pressure, affords an additional proof that the cells are not the primitive or essential seat of pneumonia. The progress of this form of inflammation is slow, and it rarely proceeds to the third stage, which Laennec describes in these cases as flabby, dry, and yellowish, with some discernible vesicles containing a concrete pus.

effused fluid after death, cannot assent to an opinion lately broached, that ægophony cannot be distinguished from bronchophony, and is not dependent on the interposition of a layer of liquid.

The occurrence of extensive peripneumony with copious pleuritic effusion is comparatively rare. It is more common that one disease has the predominance, and is attended only with a slight degree of the other. Inflammation occupying part of a lung, is frequently extended outwardly to the pleura, which becomes covered at that point with a thin coating of coagulable lymph, and secretes a seropurulent liquid; and it is a remarkable fact that lymph is often effused also by the corresponding point of the costal pleura, the inflammation being propagated by contiguity.* The ægophony on the one hand, and the crepitant rhonchus with rusty peripneumonic expectoration on the other, will easily distinguish such a case. But if the whole of a lung be inflamed, and converted into a solid mass, although there be no liquid effusion in the pleura, the principal sign that will distinguish the case from that of a copious pleuritic effusion, is a more noisy and almost pectoriloquous bronchophony at the root of the lung. But if this case has been observed during its progress, the characters of the first stage of pneumonia must have sufficiently announced its nature.

Pleurisy is sometimes accompanied by a circumscribed, and even lobular pneumonia, modified in the manner above de-

* This is one instance, out of many of the same kind, which seem to indicate that the assimilatory power of inflammation, or perhaps, even its proximate cause, is of a nature more mobile and subtle than can be explained from any known modification of vascular action. The Broussaians resort to the term *irritation*, less exceptionable only because more vague, and greatly misused in the extent to which they apply it: "Res non verba quæso." *See Cycl. Prac. Med. Art.* IRRITATION. These, and many other phenomena seem to approximate certain vital properties to electric or galvanic influences. Such an explanation is, however, yet in the uncertainty of remote prospect; we have not yet arrived at the ground of its proofs.

scribed. This inflammation will generally be announced in some corresponding point of the chest, where the stratum of pleuritic effusion is thin, by the crepitant rhonchus. In short, it is easy, from a knowledge of the pathology of pleuropneumonia, to predicate all the varieties in its physical signs, as they partake more of a pleuritic or of a pulmonary inflammation.

Section III.—*Hydrothorax.*

It was formerly the common opinion, and is even now believed by many, that idiopathic hydrothorax is a very common disease, producing a formidable array of symptoms, and often causing death by suffocation. In these late years the erroneousness of this opinion has been shewn; on the one hand, by the study of pathological anatomy, which has discovered, in the supposed cases of simple hydrothorax, extensive organic disease, without any effusion; and, on the other hand, by auscultation and percussion, which have not only proved the same during life, but have likewise taught us that hydrothorax, when it does exist, can have but a very small share in producing the symptoms that have hitherto been ascribed to it.

In fact, simple and idiopathic hydrothorax, or dropsy of the pleura, causes but one general symptom, dyspnœa, and this to no great degree, unless the dropsical effusion be very abundant, and of sudden formation. A symptomatic hydrothorax is sometimes produced a short time before the fatal termination of organic diseases of the viscera; and, excepting dyspnœa, these cases are attended with no symptom that is not frequently present in the same diseases, terminating without hydrothorax. In short, I need only refer to the history that I have given of pleurisy, to shew how very slight and uncertain are the symptoms of even an abundant effusion in

the pleural cavity. The physical signs are the only certain tests of its presence; and, in the present instance, they will be equally infallible: I need scarcely observe, that they do not differ from those of the pleuritic effusion. An idiopathic hydrothorax is to be distinguished from this latter case by the absence of fever and other constitutional symptoms, peculiar to pleurisy.

Symptomatic hydrothorax will combine, with the common signs of pleuritic effusion, those of whatever organic disease it is the consequence; and this will generally be found to be some lesion of the circulatory apparatus, by which its function is extensively impeded. Laennec states that it scarcely ever supervenes earlier than a few days before the fatal termination of such diseases, and may, therefore, be considered the immediate harbinger of death, the agony of which it increases by dyspnœa.

Section IV.—*Hæmothorax.*

Besides in the case of hæmorrhagic pleurisy, formerly mentioned, blood may be effused into the sac of the pleura from a wound, by the rupture of an aneurism, by pulmonary apoplexy, and by a passive transudation. As long as this blood remains liquid, it must produce the same effects as we have described of serum in the same situation. When coagulated, it would render obtuse the sound of percussion, and diminish the respiratory murmur in proportion to its quantity; and might, perhaps, produce bronchophony, but not ægophony, for that, as we have seen, is the result only of a liquid effusion. Some, therefore, of its physical signs might enable the practitioner to distinguish hæmothorax from other solid or liquid formations in the thoracic cavity; but the faintness and syncope, or other general symptoms of hæmorrhage, at its first attack, would also assist him in the diagnosis.

Section V.—*Pneumothorax.*

Pneumothorax, or a collection of air in the pleural sac, may be either active or passive. It is active when the air, whether exhaled from the pleura, or generated by the decomposition of a liquid effusion, by the force of its own accumulation presses back the lung towards its points of attachment. It is passive when the air, either, entering by a communication with the external air, supplies the place of the lung diminished by its own collapse, or, generated within the sac, fills up a cavity left by a reabsorbed collection of liquid, after those cases of pleurisy, in which the lung is bound down by a fibro-cartilaginous membrane, and the parietes of the chest cannot by their collapse obliterate the cavity. This division includes and defines all the varieties of pneumothorax.

The most common of all these is that variety of the passive form which results from a fistulous communication between the pleural sac and the bronchi. This communication is usually caused by the tubercular ulceration in phthisis, extending itself through the pleura. The pneumothorax, in this case, is usually accompanied by some pleuritic effusion, excited by the entrance of softened tubercle or some extraneous matter, or merely of the air, through the fistula. The perforation is, therefore, often the immediate result of a fit of coughing, and is attended with sudden acute pain, dyspnœa, and cough. I have seen the intercostal muscles thrown into strong spasmodic contraction on the first entry of air into the pleura. These symptoms happening in a phthisical subject are very characteristic of perforation of the pleura; and the altered form of the affected side, together with the other remarkable physical signs, render the disease one easy to be recognised.

Active pneumothorax is of rare occurrence, and is generally, like symptomatic hydrothorax, with which it is sometimes

conjoined, the precursor of death. It sometimes accompanies the pleurisy excited by the bursting of a tuberculous vomica into the pleura, where there is no communication with the bronchi.* It has been known to occur with common pleurisy.

The physical signs of pneumothorax are very characteristic, but they vary considerably, according to the form of the disease. In all the varieties, but particularly in the active kinds, the tympanic sound of the chest on percussion is increased, so that the diseased sounds as well as, or even better than, the healthy side; the reason is too obvious to require explanation. Hence percussion alone may be a source of error. Auscultation will correct it, and a certain diagnosis may be deduced from their conjoined indications. The sound of respiration will, by the pressure and interposition of the air in the thoracic cavity, be obliterated in all parts except at the root of the lung; whereas, in early pleuritic effusion, it may still be heard. Where, therefore, one side of the chest is very sonorous on percussion and yet no respiratory sound is heard, it may safely be concluded that a pneumothorax exists. The only case in any degree approaching to this is that of emphysema, but in this the sound of respiration is only diminished, not entirely destroyed, and the presence of an occasional sibilation, and still more certainly its pathognomonic sign, emphysematous crepitation, will clearly distinguish this disease. Add to these, that the sound of respiration, absent in other parts, remains audible at the root of the lung in pneumothorax, while no such difference is observable in emphysema, and the respiration on the healthy side is generally in the former case very puerile. In pneumothorax, as in pleuritic effusion, the lung is sometimes retained in contact with the chest here and there by adhesions; and at these points the respiratory murmur is not obliterated. Hence, in

* Louis, Recherches sur la Phthisie.

such cases, the necessity of examining every part of the thoracic surface before a correct knowledge can be attained of the physical state of the organs contained in it. In thin subjects the intercostal spaces are rendered elastic and apparently filled out by the air in the pleural sac, and present to the eye and to the feel a state very different from that of the healthy side. In active pneumothorax of the left side, the heart is sometimes pushed under or to the right of the sternum; and there may be other signs of displacement as mentioned in pleurisy.

When there is a collection of both liquid and air in the pleura, the chest is of course less sonorous on percussion than in simple pneumothorax; but then the inferior parts sound very dull, while those above emit a clear sound, and the transition from one to the other is often very abrupt. On practising percussion, too, in different postures, the presence of a liquid will be discovered by its rendering the sound obtuse always in the most dependent parts of the thorax; while the air, rising to the superior parts, gives them a tympanitic resonance. This effect of change of posture is far more obvious in this case than in pleurisy.

It is in these cases that the presence of the liquid becomes frequently perceptible to the ear by the sound of gurgling or fluctuation, into which it is thrown by the respiratory movements, or any sudden motion applied to the thorax. Here it is, therefore, that the *succussion*, or saltatory agitation, employed by Hippocrates, furnishes a physical sign of pneumothorax with liquid effusion. The best method of obtaining this sign is by the patient himself making a lateral jerking, or a half rotatory motion with his trunk, in the sitting posture. If he be too weak for this, the succussion may be effected by another person applying his hands to the shoulders. On applying the ear to the chest, in such a manner as not to interfere with the motion of succussion, the fluctuation of the

liquid will be heard as in a cask or vessel partially filled, when it is shaken; and a reference to this analogous case will suggest to the operator the most effective method of producing the phenomenon. The roughness of this kind of examination should preclude its employment in all cases of great weakness or painful irritability; for although I believe with Laennec that it is much less fatiguing than would at first be supposed, yet the excitement that it produces must, in these cases, be necessarily injurious.

There are yet other signs by which the auscultator may recognise the presence of air in the thorax. They are of singular character, and have been considered of difficult explanation; but I apprehend that the hitherto too much neglected study of acoustics will furnish us with a key to open the mystery. Every one knows that an empty room yields a kind of reverberation or prolonged echo to any sound made in it. Need I explain that this echo is the sonorous vibration repeatedly reflected from the walls around.* The echo is in such cases chiefly in unison, or in the same note with the original sound. But substitute for the room of many cubic feet capacity, a cavity of only a few cubic inches, the sides of which are still good and uniform reflectors of sound, and then a sound, communicated to the air within it, will be so rapidly

* The more perfectly and uniformly reflective the walls, floor and ceiling of the room are, the more complete and durable will be the reverberation; one built entirely of stone, illustrates this in perfection. The form has little to do with the present question, except that the more uniform the surface, the less is the original sound changed; and a hollow sphere, therefore, best presents this condition. The complication of form would modify the sound; and dissimilarly reflecting composition would neutralize or destroy it. Hence a room crowded with people, with various furniture, yields little or no reverberation. Every room has its proper pitch of echo, which will respond to any sound; and this may be roughly calculated, by dividing the velocity of sound through air (1130 feet per second) by the prevailing diameter of the room. Thus in one 10 feet square the note of the echo would be that of 113 vibrations per second. But a little cavity of three inches would yield the acute note of 4520 vibrations in a second, and could not reverberate a lower tone to any note however low. The peculiar echoes between two parallel walls, in empty barrels, bottles, cups, &c. are illustrations of this law.

reflected by its parietes, that its successive reflections will strike the ear more frequently than the vibrations of the original sound, hence this reverberation or echo will be in a tone raised in proportion to the smallness of the cavity, and prolonged in proportion to the reflecting power of its walls. Such an acute note rapidly dying away, strongly resembles the tinkling sound produced by bodies of powerful molecular elasticity, such as glass, metals, &c. The elements necessary to produce such a metallic tinkling, therefore, are, first, a cavity of uniform reflecting parietes; and second, the communication of a sound, or of a soniferous impulse to the air contained within it.*

Now it may be perceived that pneumothorax may combine

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* There are many other instances of the production of this metallic tinkling in the body, and all of them unite these conditions. It may be heard by the stethoscope applied to the stomach of a person swallowing water by teaspoonfuls. It may often be heard in the intestines, distended with gas by the sudden motion of liquid in them. A somewhat similar sound, too important to the auscultator to pass unnoticed, occurs in the ear, on closing it with the palm, and lightly tapping on the back of the hand. Now this sound in the ear is sometimes excited by an external impulse during auscultation, particularly immediate, and might be readily referred to the chest of the patient. Laennec himself, not aware of the facts I have stated, has fallen into this error. He describes in his second vol. p. 445, a *cliquetis métallique* occasionally heard in the precordial region of persons affected with violent nervous palpitations; and considers it a sign of the presence of some bubbles of air in the pericardium. He has shown me instances of this symptom, and I have since convinced myself, by repeated observation, that it is produced solely in the ear by the impulse communicated to the air within it. Other instances of its production, as on applying the hand to the stethoscope or naked ear, and rubbing together in different ways the fingers, which Laennec refers to the presence of air in the capsules and sheaths of the tendons, may be clearly traced to the same cause by any one who will take the trouble to vary the experiments. For example, lay the ear flat upon the table, and tap the table with the the finger, or something of analogous density, and the stroke will still be heard accompanied with the same metallic clink. For a long time I considered this sound in the ear to be a tinkling echo in the external meatus; but finding that its note was not raised on diminishing the cavity by introducing a body into the ear, and that the sound ceased on forcing air strongly through the eustachian tube, I am now inclined to consider it as the note of the tympanum itself. In two cases of slight deafness in one ear, this note was sensibly lower in that ear, which would indicate a laxer state of the tympanum.

these conditions; let us study what forms are most favourable to their union. The cavity is always present in pneumothorax, but it is best calculated to produce the tinkling echo when its parietes are tense and regular, as when the lung is bound down by a fibro-cartilaginous membrane. If the parietes of the cavity are loose or flaccid, they do not reflect, but absorb, the vibrations, and then no echo is produced; just as we see curtains or loose drapery destroy the reverberation of a room.

The soniferous impulse may be produced within the cavity, or may be communicated to it from without. One cause of sound within the cavity would be a portion of liquid contained in it, dropping on change of posture, or in the motions of respiration, from the upper to the lower parts. Such a case of metallic tinkling is rare, but Laennec records an instance, and I have myself met with one. The sound is, in this instance, as Laennec describes, like that of a drop falling into a decanter, a fourth full of water, followed by a prolonged ringing. The symptom in this case of course indicates the presence of liquid as well as of air in the pleura. The bursting of air bubbles in this liquid would also occasion the tinkling sound, and in such a case succussion might probably be made to form bubbles: it is also possible that they may be generated by decomposition of the liquid, as suggested by Dr. Spittal, where there is no communication with the bronchi.

Sounds, external to the cavity, may cause the tinkling echo within it; and they may be communicated in several ways. Thus a fistulous communication with the bronchi may transmit a sonific impulse to the cavity on the occasion of speaking or coughing, and this is by far the most common case of tinnitus metallicus. It is here most perfectly produced, when the fistulous communication is short, but narrow, and the bronchus is of considerable size. The sound is then heard immediately after the cough or utterance, like that yielded by a wine-glass when struck by a pin, and is of longer

or shorter duration. Sometimes, but more rarely, the same tinkling sound accompanies respiration. It is very probable that the more prolonged ringing in these cases depends on air passing through the liquid in successive bubbles; these bursting in the cavity, cause a sound, which, reverberated in its tiny echoes, becomes tinkling or silvery.* If the fistulous communication be large, or if there be several, and above the surface of the liquid, the tinkling is changed into a hollow buzzing sound, like that produced by blowing into an empty bottle; wherefore Laennec calls it *amphoric buzzing.* This is most distinctly heard during respiration, but likewise accompanies the cough and the voice. It is obviously caused by the passage of air in and out of the pleural cavity. This passage of air deranges the reverberations that produce the tinkling echo within the cavity; hence this amphoric respiration either impairs, or completely destroys, the tinnitus metallicus. Let the observer bear in mind these circumstances, and he will then perceive how metallic tinkling may be succeeded by amphoric respiration, or an enlargement of the fistulous communication with the bronchi; and how the converse may result from its being contracted, or covered with liquid, and that both will cease on its occlusion. To some degree the same effects may proceed in certain cases from different degrees of force in the respiratory movements, which may of themselves open and shut the fistulous aperture; and I have known each of these phenomena presented in the same

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* The suggestion that the bursting of air bubbles may be concerned in the production of metallic tinkling, is due, I believe, to Dr. Spittal of Edinburgh. (See his Treatise on Auscultation, 1830.) Some French writers have adopted a similar notion. But air bubbles cannot produce metallic tinkling without the resonant cavity; and as it will be seen in the text, when the resonant cavity is present, any sound transmitted to it is followed by the tinkling echo, and a slight change of circumstances may develope in it the *amphoric buzzing*, or as Dr. Spittal better terms it, *amphoric respiration.* Hence it appears unnecessary to separate the varieties of the phenomenon.

patient, according as he coughed or breathed with effort, or spoke quietly and breathed with ease. In all cases on record, this form of pneumothorax has been accompanied with more or less liquid effusion, and probably this is a necessary attendant of the pleurisy excited by the access of air or extraneous matter through a fistulous communication with the bronchi. That may therefore be anticipated, which I have had frequently occasion to observe in practice, that this liquid, by occasionally covering more or less completely the fistulous aperture, may also modify, diminish, or destroy, either of the symptoms which I have been describing. Hence change of posture, by altering the situation of the liquid, may assist the auscultator to the discovery of the symptom; and in some cases might even lead him to calculate the situation of the fistulous aperture. I think it important to remark that the fistulous aperture may, in some circumstances, act as a valve, admitting air by little and little into the pleural sac during inspiration, but by the pressure of expiration being closed so as to prevent its egress. This would make the case much worse, because the air would then accumulate and press on the lung, as in active pneumothorax.*

Laennec considered metallic tinkling a pathognomonic sign of pneumothorax with liquid effusion and fistulous communication with the bronchi. The accuracy of this opinion I have been led, by *theory* and *experience*, to call in question. From the theory that I have given, it may be judged that although such a lesion may be the most frequent cause of this symptom, it needs not such a complicated state to produce it. The cavity being present, as we have said, in pneumothorax, a cause of the tinkling echo in it may be found in the transmission of a sound or sonific impulse through solid parietes.

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* Dr. J. Johnson's case, described in the *Med. Chir. Rev. No.* 20, appears to have been of this description. The patient was relieved temporarily two or three times, by giving exit to the air, by paracentesis.

For example, the voice or cough may communicate such impulse whenever, by an induration or condensation of the pulmonary tissue, it is rendered capable of conducting to the cavity the sound from any large bronchial tubes. Such a condensation, we have seen, may be produced by inflammation, and by the pressure of a liquid effusion, and might, doubtless, in some cases result from the pressure of the air of the pneumothorax itself. Add to these the case of pneumothorax, combined with such tuberculous excavation of the lung that pectoriloquous resonance is transmitted from it to the pleural cavity, but without fistulous communication. I have also heard the pulsations of the heart cause the tinkling echo in pneumothorax.

Laennec himself furnishes two examples, which further prove the point. In one, the tinkling was produced in pneumothorax, where there was no communication with the bronchi or external air, by the dropping of a liquid in the cavity.* Another case presented the tinkling echo *after the voice*, although there was no communication with the bronchi, but only with the external air through a puncture in the thoracic parietes (tome I, p. 113.) These cases remove two of the conditions specified by Laennec, as necessary to the production of metallic tinkling; namely, communication with the external air; and transmission of the voice through a fistulous opening in the bronchi.

The following case, which fell under my observation in the ward of M. Lerminier, at La Charité, shews that the presence of liquid effusion is likewise dispensible. A boy, fifteen years of age, had been for some weeks affected with pectoral disease, with cough, shortness of breathing, scanty expectoration, &c. but these had somewhat abated, until a few days before, when they had become considerably aggravated. When

* The case before alluded to, tome II, p. 348, of Laennec.

I first saw him, he had, besides, much fever, quick pulse, pain of side, and other symptoms of an acute attack. On percussion the left side sounded well every where, but in the inferior or lateral and posterior region, where it was rather duller than usual. The right side was very sonorous on percussion anteriorly and laterally, below the fourth rib; less so above, and posteriorly. On auscultation, the respiration of the left side was distinct superiorly and anteriorly, but mixed with crepitant rhonchus laterally and posteriorly, particularly in the inferior parts. No bronchophony or bronchial respiration. On the right side the respiration was puerile below the clavicles and in the axilla, and above the spine of the scapula; became less distinct somewhat below, and was quite inaudible below the fourth rib, the part most sonorous on percussion. In this also, after cough and utterance, a distinct metallic tinkling was heard, which appeared not to be affected by change of posture. The next day the same symptoms were present, but the crepitant rhonchus had extended upwards in the left lung; and the patient seemed worse. Tinnitus as before. Died in the course of the day. On dissection, about eighteen hours after death, about the inferior and anterior half of the right side of the chest was filled with air, some of which escaped with a hissing noise on first incision; the lung was bound down to the whole of the posterior, and the upper portions of the lateral and anterior parietes of the thorax, by a pretty firm fibro-cartilaginous membrane, which also thickened the costal pleura of the cavity. *There was not a drop of liquid in the cavity, and there was no communication with the bronchi.* In the inferior lobes of the compressed lung were found three hydatids, contained in, but not connected with, a cavity lined with a fibrous membrane. The tissue of the lung was flaccid, and compressed in their vicinity, and bounding the cavity containing air, but above it was healthy and crepitant. The lower lobes of the left lung were found in a

hepatized state, which passed superiorly into simple inflammatory engorgement; and still higher up the tissue was healthy. There were no tubercles in either lung.

The history of the case, and the rationale of the symptoms, is obvious. The effusion of a former pleurisy had been absorbed, but the lung being bound down by a tense false membrane, could not expand to refill the cavity; hence it became filled with air exhaled into it, constituting a variety of passive pneumothorax. The tinkling echo was produced in this cavity by the voice and cough, transmitted through the pulmonary tissue condensed by the former effusion and by the hydatids. I have since met with a case of pneumothorax produced in this way, and Dr. Stack notices a similar sequel of pleuritic effusion.* Dr. Forbes, in his valuable notes to Laennec's work, also adverts to a case of distinct metallic tinkling, in which, on dissection, no communication between the bronchi and the cavity could be discovered.† But in comparison with the other form of pneumothorax, these cases are rare.

Active pneumothorax, or that which results from a generation of air within the pleura which presses the lung, may become a fit subject for the operation of paracentesis, and there are instances on record of its having been performed with the effect of averting impending suffocation.‡ Where there is fistulous communication with the bronchi, the relief can be but temporary; and the coexistence of tubercular disease must give to the case a hopeless character. The form of passive pneumothorax which supervenes to fill a void left by a partial pleurisy, has no other bad effect than that of permanently contracting the sphere of the pulmonary function.

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* *Dublin Hosp. Rep.* vol. iv. p. 90. † 4th edit. 1834, p. 56.

‡ Dr. Johnson, *Med. Chir. Rev.* No. 20, and Dr. John Davy, *Phil. Trans.* 1823.

CHAPTER IV.

PHTHISIS PULMONALIS.

Section I.—*Pathology.*

The disease termed *phthisis pulmonalis* is produced by the formation of a particular matter called *tubercle*, in the tissue of the lungs. It would perhaps be more consistent with the order of the work, if I had placed this disease among those affecting particularly the pulmonary texture; but I am induced to allot to it, and to analogous affections, this separate chapter, because the previous examination of other simple diseases will better enable us to understand the pathology of this complicated one.

I shall first trace the progress of the changes which morbid anatomy has shewn tubercles to undergo in the progress of the disease, and afterwards inquire into their nature and origin.

The lungs of those who die phthisical, present some, or all of the following changes:—

I. Small, roundish, semi-transparent bodies, of greyish or ash-coloured hue, of different shades, of a size varying from that of a pin's head to that of a hempseed, and of hardness nearly equal to that of cartilage. These little bodies are dispersed about the substance of the lung in variable numbers, here isolated, and in a tissue otherwise healthy; there agglomerated together in clusters, and surrounded by texture more or less diseased, and often surrounded with the black pulmonary matter. Some are found, on close examination, and on incision, to differ from others, in having within them,

generally, but not always, about their centre, an opaque yellowish white spot, which, traced in different ones, may be observed to be small in some, larger in others, and in some to constitute the principal part of the little miliary body.

II. A diffused induration of the pulmonary tissue, in colour and consistence resembling the preceding, but of greater extent than they, and confined to no particular size or form, but it is usually near parts occupied by the tubercles or excavations to be presently described. The texture of the lung can no longer be detected in the part thus affected, and, when cut into, it presents a moist, homogeneous shining surface, and is totally impermeable to air. Sometimes the induration is inconsiderable, although the other characters remain ; and in rarer cases the change seems to be produced by the infiltration of a matter nearly gelatinous, and slightly sanguinolent, which more or less obliterates the pulmonary texture. This might be considered a distinct alteration from the others, were it not that their coincidence and mutual gradations seem to identify them.* The tissue thus affected becomes, like the miliary granulations, invaded by the yellowish white spots; only these spots here are irregular as to form and number, and they appear to be the commencement of a process by which this grey induration becomes more or less converted into—

III. Opaque masses, of a yellowish white colour, of various size, generally of a roundish form, and of consistence at first considerable, and nearly equal to that of the matter in which they were produced; these are what are called *crude* tuber-

* This gelatinous effusion, according to Laennec, is generally observed accompanying the miliary granulations before mentioned, particularly secondary ones, produced after others have suffered the changes to be described immediately. To me it appears to be a modification of coagulable lymph, not unlike that sometimes seen effused on the pleura and peritoneum, under the less acute forms of inflammation, and which, as Dr. Carswell remarks, also sometimes contains tuberculous matter.

cles; but, in the course of time, they gradually become, in consistence and colour, like soft cheese, and at length attain the liquidity of pus; this change generally beginning towards the centre. The miliary bodies become likewise converted into little opake yellowish granular tubercles when isolated; but when in a cluster, frequently run together into one tuberculous mass of considerable size. The tuberculous masses formed in the diffused induration may, of course, be of form and size as varied as those of the matrix in which they are developed. But the opaque tubercle seems likewise sometimes to increase of itself, to extend beyond the limits of the previous induration, and to encroach on the surrounding tissue. In some few instances this progress is limited by the formation of a fibrous cyst. The yellow tuberculous matter is also produced in some cases, without the previous induration, especially in glands, in the ovaries, and in the vesicular structure of the lungs; and a tuberculous infiltration of this kind is not unfrequently found in the lungs and bronchial glands of children.* After the softening of the tuberculous matter, it is evacuated by a fistulous communication, generally into the bronchi, and rarely into the pleural sac.

IV. After the softened tuberculous matter is evacuated, an ulcerous cavity is left, which presents to the anatomical observer a great variety of form. The walls of the cavity are sometimes simply the pulmonary tissue, in an abraded and sloughy state, or sometimes more or less red with inflammation. The result of this inflammation (excited probably by the irritation of the tuberculous matter) is, during life, on

* Dr. Carswell regards the mucous surfaces as the principal seat of tuberculous matter; the serous surfaces, he says, also present it, and it may occur in blood or in lymph recently effused; but in no instance, he says, is it deposited in the molecular structure of organs. (*Cycl. of Pract. Med. Art. Tubercle.*) Andral considers the cellular tissue its chief seat, but that it may occasionally occur on mucous and serous surfaces. Lombard supposes it to be restricted to cellular tissue. I shall advert to this subject again.

the one hand a dark-coloured homogeneous condensation of the pulmonary texture surrounding the cavity; and, on the other, the secretion of liquids of mixed tubercular, purulent, serous, and sanguinolent matter, and, in some cases, of a coagulable lymph, by which a kind of lining is formed within the cavity. The effect of this latter process is shown in its various stages of advancement in the cavities of different ages that are met with on the examination of phthisical lungs. The coagulable lymph becomes, in time, converted into a mucous lining, which often thickens into a fibrous or even a fibro-cartilaginous membrane, the thickness and firmness of which are generally in proportion to its age.

The cavities are very various in size and form. Some would not contain more than a pea, while, occasionally, a cavity is found occupying a whole lobe. Cavities of large size are most commonly formed by the re-union of many smaller ones, produced by the evacuation of their softened tuberculous contents. The ulcerative process, as it is necessary to open a communication between cavity and cavity, and between these and the bronchi, so also often extends the limits of a cavity by encroaching on the healthy tissue. In this way a lobe is sometimes reduced to a mere sac, composed of the pleura, and a thin layer of condensed pulmonary tissue. In other instances the ulcerative process has been less active, and the cavity may then be sinuous, multilocular, or composed of several small cavities communicating with each other. Occasionally, bands of condensed pulmonary tissue, crossing a cavity, or vessels the caliber of which is generally obliterated, are all that remain of the divisions of former small ones. Cavities recently formed, or which have only recently communicated with the bronchi, usually contain besides some remains of the tuberculous matter, in the form of a curdy liquid, pus, and a greyish or brownish grumous liquid, secreted by the inflamed parietes. Sometimes a little blood is found mixed

with these, but this is seldom to a great extent, as the vessels which terminate in the cavity are almost constantly closed by coagula, and the adhesive inflammation. The effect of this stopping of the principal vessels is sometimes to cause a partial gangrene and sloughing of the tissue around an excavation; this necessarily increases its extent, and occasions the fœtor which phthisical sputa sometimes present.

The quantity of matter secreted by tuberculous cavities is diminished by the formation of a mucous or fibrous lining, and when this is complete, the secretion is nothing more than a scanty serous, or sero-mucous liquid. Thus far of the anatomical history of phthisis pulmonalis; more particulars will be learnt from its physical signs.

It appears, then, that the principal changes of the tissue of the lung in phthisis pulmonalis are of two kinds; first, an induration of a grey, or greyish brown colour; second, the production, generally in this induration, sometimes elsewhere, of a yellowish white opake friable matter, at first rather hard, but becoming gradually softer, until it attains nearly the liquidity of pus. Now, let us endeavour to discover what is the essential nature of each of these changes, what it is that causes them, and in what relation they stand to each other.

Now, in the grey induration of the pulmonary tissue, whether granular or diffused, two things are remarkable; first, that there is an increase of substance, for the spongy texture of the lung is solidified and obliterated; secondly, that its substance is harder than the healthy tissue; the first denoting the deposition of a greater number than usual of molecules by the nutritive secretion; and the second proceeding partly from the same cause, and, perhaps, partly from these molecules being unusually solid. Now this increase of substance implies either increased secretion or diminished absorption; that the latter is not the cause is obvious, from the circumstance that parts of the healthy tissue are often

absorbed away at the same time; and that increased secretion is present is proved from the fact, that the particles deposited present new characters, and do not constitute a mere accumulation of the matter of the natural tissue. Now to produce an increase in the nutritive secretion, according to a general pathological law, there must be an increased determination of blood to the part. Whether this amounts to inflammation can only be determined by defining inflammation more exactly than has hitherto been done. In the mean time, if we compare the indurated state in question with the acknowledged results of inflammation, we cannot fail to see a close resemblance between them. Inflammation we know to cause an external overflow of the nutritive secretion; and I have before* endeavoured to shew why the acute form should generally produce a soft tumefaction, and the chronic an indurated increase of substance; facts sufficiently established by Andral and others. Thus the hard compact granular hepatization, considered by Laennec as a result of chronic peripneumony, occurring around gangrenous and tubercular excavations, is sometimes of a livid brown or grey colour, and of the consistence of the grey induration. But as I have shewn that there is a non-granular form of acute hepatization, so it is reasonable to suppose that there may be a diffused or uniform kind of solidification resulting from chronic inflammation. To such a state, the grey induration, called tuberculous by Laennec, so exactly answers, that Andral, Chomel, and Louis, concur in considering it a chronic form of hepatization. When it is the sequel of the acute disease, there is often a redness in the induration; but where the morbid cause has been of long continuance, and unconnected with the more sthenic degrees of vascular action, the texture is grey, dense, and variously modified by the black pulmonary matter in it. The more uniform

* *See* Section on Pleurisy, p. 99.

and harder masses, occasionally present, may be traced to be the interlobular septa in a state of indurated hypertrophy. In these bloodless and almost cartilaginous portions, we see the exact characters of matter, of which the miliary granulations of Bayle are minute samples. Moreover, as we have been enabled to trace the various diffused consolidations of the pulmonary tissue through the many gradations from acute hepatization down to grey induration, so M. Andral has found miliary granulations presenting the same series of gradations; being sometimes soft and red, in other cases livid and harder, whilst the same lung may contain also the granulations similar in size, but pale or grey, and of various degrees of hardness.*

On the whole, then, it seems more philosophical and satisfactory to consider the miliary granulations, as well as the grey induration, of the lung, as the result of an overflow of modified nutritive matter in the tissue, produced by a certain degree of increased vascular action,—than, with Laennec, to call them the first stage of tubercle,† or, with Dr. Carswell, to suppose them to be constituted by a grey semi-transparent inspissated mucus secreted in the air cells.‡ Let us now examine the ulterior changes.

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* *See* Dr. Hope's Coloured Illustrations of Morbid Anatomy, Part I. Fig. 15.

† Much as Laennec has done in elucidating the history of phthisis pulmonalis, his opinions on tubercles and other diseased productions have always appeared to me artificial and unsatisfactory. Tubercles, an accidental tissue, are produced, or, according to some of his expressions, spring up, in a healthy tissue, without any aid of the vessels of the part; are changed from a greyish semi-transparent to an opake yellowish white, and pass from a state of cartilaginous hardness through intermediate gradations, into that of imperfect liquidity; and all this by mechanism perfectly unknown, and in a manner entirely unexplained. It is too, in my opinion, an undue assumption, when he identifies the granulations of Bayle with the yellow tubercle, bodies quite different in their physical character, only because the one is generally in time converted into the other.—As well might cartilage be called bone, or (if the example does not involve a *petitio principii*) inflamed cellular texture, a stage of pus.

‡ If this opinion were correct, we should expect these granulations to be pro-

We have found that, after a time, the indurated grey semi-transparent, but still organized mass, presents whitish opake points or spots, which increase in size, and at length convert more or less of the mass into a friable substance of a yellowish white colour: this generally retains at first a considerable consistence, but afterwards gradually losing it, becomes that soft and grumous curdy matter, known under the name of *matured tubercles.*

To illustrate this point further, we may with advantage refer to analogous changes on the surface of a simple membrane, such as the pleura. In the latent and more protracted forms of pleurisy, we have had occasion to remark that the lymph first effused forms a dense tissue of low vitality, and approaching fibro-cartilage in hardness and colour. If the irritation still continues, this new structure throws out a lymph of still lower vitality, in friable shreds, and in some cases in form of a curdy matter totally incapable of organization, which, mixing with the effused serum, constitutes one kind of empyema. Now, such a process in the pulmonary tissue would produce all the changes which we have been describing in the successive production of grey induration, crude and softened tubercle. Thus a portion of this tissue (whether a single vesicle or part of a lobe) under the influence of chronic inflammation, or local increased action analogous to it, becomes indurated by the effusion of lymph susceptible of a low degree of organization. The irritation continuing, or other circumstances determining an increased flow of blood to the part, the new structure evolves in the looser parts of its substance a still less organic form of albuminous matter, but as this cannot (like that from the pleura) be thrown off, it presses on

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duced in dry catarrh, in which the mucus of the cells and bronchi attains its greatest degree of spissitude; but this is by no means the case, and I may add that tough mucous expectoration is by no means common in the early stages of phthisis, in which the granulations are developed, and when the sputa, if any, are thin and glairy.

its indurated matrix, and causing its absorption, accumulates in its place: thus is produced the conversion of the grey induration into crude tubercle.

But the semi-transparent induration is not always transformed into crude tubercle. Sometimes it is the seat of vomicæ which contain a dirty or bloody pus; and although even in this the curdy matter of softened tubercle is sometimes seen, it is plain that these vomicæ result from a more direct and speedy process of ulceration or irregular suppuration, another analogous result of continued increased action in the condensed tissue.

The preceding remarks apply to the circumscribed indurations, or miliary granulations, as well as to the diffused forms. The constant shape and size that these miliary granulations present, led Andral to conclude that they are individual vesicles, or the terminal sacs of single bronchi in the state of induration from chronic inflammation; and this opinion appears the more probable from the fact before stated, that this distinguished pathologist was able in many instances to distinguish similar points red and soft, as if from acute inflammation.

But the grey induration is not the only source of tuberculous matter. It is sometimes deposited in a hepatized lung, either in circumscribed and roundish masses, or in a more diffused form, infiltrated through the inflamed tissue. The latter state is compared by Dr. Carswell to boiled liver. I have seen in the lungs of a child the yellowish opake tuberculous matter deposited in parts of a lobe completely hepatized, whilst the uninflamed portions of lung were quite free. Dr. Alison and M. Andral relate similar cases, in which it is impossible to evade the conclusion, that the deposition of tuberculous matter was occasioned by acute inflammation, perhaps of slower progress than usual.*

* The connection between inflammation and tubercle is further shewn in the

Lastly, tuberculous matter is certainly sometimes found in tissues bearing no marks of inflammation or other disease; and although in most of these cases the structures thus invaded are either peculiarly vascular, or subject to congestions of blood, (as the bronchial glands, the lungs of children, the spleen of monkeys) yet no proof can be shewn of the antecedence of any inflammatory process.

Whatever may be the cause which determines the deposition of tuberculous matter in these cases, we know that pus is sometimes secreted in parts unaffected with inflammation, witness the purulent deposits in the viscera after great surgical operations, and the profuse discharges of matter from the bronchial membrane, which after death is sometimes, in such cases, found paler than usual.*

The origin of tuberculous matter in these cases, is much elucidated by the fact that it is also sometimes found in the blood itself, in coagula in the spleen and in the heart, and also in fibrous concretions within the vessels. This circumstance tends to shew that the fibrinous portions of the blood are liable to be converted into tubercle, independently of any action of the vessels, and that the mere secretion of this matter from the blood may be the cause of its deposition without irritation in the various tissues before named.

It is probable that this diseased state of the blood is the principal element in what is called the tuberculous diathesis, from which there is disposition in vessels of different degrees of activity, to deposit tubercle instead of lymph, and when

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similarity of their exciting causes, as proved by several experiments. Thus M. Cruveilhier caused the formation of tubercles in the lungs of rabbits, by dropping a little mercury into the trachea; (*Nouv. Biblioth. Medicale*, Sept. 1826) and M. Flourens produced a similar effect on young ducks and chickens, by exposing them to continued cold less severe than that which would produce acute inflammation. (*Ann. des Sciences Nat.* 1828.)

† M. Andral notices what I have myself seen,—pus in the centre of fibrinous coagula in the heart, in cases where no purulent matter could be found elsewhere in the body. (*Path. Anat. Transl.* vol. i. p. 503.)

this diseased state is in excess, the tuberculous matter is excreted from the blood without any increased vascular action.* Whatever in these cases determines the first deposition of tubercle, will, with greater facility, determine its growth by the addition of fresh tubercular matter to a ready formed nucleus.

The next process to be considered is that of the softening of tubercles. This appears to take place in the mode sagaciously pointed out by my friend M. Lombard, of Geneva.

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* M. Andral, among modern pathologists, first pointed out the diseased condition of the blood as most probably connected with the tuberculous diathesis; the deposition of the morbid matter being a process of secretion, and subject to the influences which affect secretion in general. Dr. Carswell has adopted a similar opinion, and Dr. James Clark has further sagaciously investigated the functional and constitutional disorders which seem to give rise to this morbid condition of the fluids. (*See Cycl. of Pract. Med. Tubercular Phthisis.*) I am myself disposed to consider tuberculous matter, pus and coagulable lymph, only as varieties of the albuminous matter existing in the blood, and differing from each other in mechanical condition and consequent capability of organization, rather than in chemical composition. In this view, if the blood is perfect, the difference of the secretion will depend on the degree of vascular action, and the structure of the tissue; but if the blood is imperfect, or diseased, then whatever be the degree of inflammation, perhaps the source may be incapable of furnishing more than one or other form of secretion, and this inability may extend to the process of reparatory nutrition, and perhaps even to the external secretion from surfaces. To take two extremes. In inflammatory fevers, the blood abounds in fibrine of high vitality, which is readily and abundantly thrown out to form the organized products as described in the section on acute pleurisy. In the scrofulous diathesis, or tuberculous cachexia, (as Dr. Clark names it) the fibrinous portions of the blood are deficient in vitality, and if thrown out, even by inflammation, instead of uniting and becoming throughout organized like lymph, here and there remain in the separate particles of friable tuberculous matter, whose relation to its containing tissue is rather that of a dead extraneous body, than of an assimilable product. If the cachexia be in excess, or the vitality of the blood very low, then portions of its fibrine may be continually losing that organized globular condition which is proper to them, and they may be excreted in various tissues as circumstances may determine, and independently of any inflammatory action. The nature of this work prevents my pursuing the applications of these views; but I may mention, in support of them, the recent microscopic observations of M. Gendrin, who found tuberculous matter to consist of separate particles, without the globules which are seen in the fibrine of the blood, and in effused lymph, and which, by uniting in rows, constitute the organized layers of the latter substance. (*Histoire Anatomique des Inflammations, tome* 2, *p.* 595.)

The tuberculous matter acting as a foreign body on the portions of tissue which are in contact with it, and which in the case of tuberculous masses occur through their substance, occasions the secretion of pus or serous fluids, which either divide the tuberculous matter into clots, or reduce its whole substance into a purilaginous state. The manner in which increased vascular action will contribute to hasten this process is obvious; and experience often proves that tubercles may remain long unchanged in the lungs until an attack of pneumonia or bronchitis determines the changes by increasing the irritability of the tissue and developing activity in its vessels.

More rarely tuberculous matter undergoes another change; instead of softening, it becomes gritty and hard, from being almost entirely composed of earthy matter, consisting chiefly of carbonate and phosphate of lime. This transformation is often attended with a puckering of the surrounding tissue, as if to fill up a space left by the change. This circumstance seems to prove that the animal part of tuberculous matter may be absorbed; and it is probable that the calcareous tubercles in question result from the accumulation of insoluble earthy salts, left by successive quantities of tuberculous matter deposited, and again absorbed.

I think, then, that it is of sufficient importance to distinguish three ways in which the lungs may become infested with tuberculous matter.

I. By the tuberculous conversion of the indurations, whether granular or diffused, which we have been induced to consider the result of a chronic inflammation of the pulmonary tissue; this being the natural termination of such inflammation, as suppuration is of acute inflammation.

II. By tuberculous suppurations of other inflammations of the pulmonary tissue; this substitution of tubercle for pus being determined by the prevalence of the tuberculous

diathesis, in which there probably exists a degenerated condition of the fibrine of the blood.

III. By secretion in tissue bearing no marks of other lesion, the tuberculous matter being here apparently deposited through excess of tuberculous diathesis.

It is these last, the most constitutional, elusive, and obscure forms of the disease, that most bid defiance to our therapeutic means; and it is, perhaps, in great measure, in proportion as these combine themselves with the other form that the diseases becomes rapidly and certainly fatal. However, a predisposition to the chronic inflammations is scarcely less lethiferous, in giving spring to its irremeable course; and what adds to the difficulty of checking the disease is, that developed by constitutional weakness, and accelerated by various degrees of local vascular action, it may run as it were, in a double channel. And here I would remark, that this view suggests what is sanctioned by experience; that neither an antiphlogistic nor a tonic treatment can exclusively suit the generality of phthisical cases; but that a mild tonic and nutritive regimen combined with local antiphlogistic measures, especially counter-irritation, and with whatever sedatives and alternatives the state of the functions may indicate, will constitute the best treatment, and that can only be called the least unsuccessful.

It would be an interesting, and, perhaps, practically instructive task, to trace the mode of the operation of the reputed occasional causes of pulmonary consumption, in opening one or other of these ways for the entry into the system of this demon of destruction. It is by this mode of inquiry alone, that a knowledge of a truly rational system of prophylaxis can be obtained; and so, if cure will ever be within the reach of human power, it is to be found only by the study of the pathology, and of the properties of external agents with relation to it.*

* The Essay of Dr. James Clark before alluded to, is a valuable practical con-

The systems, or general plans in which medicine has been arranged, have been always framed on dogmas too exclusive, and observations too limited, to comprehend the varied and complicated forms of disease.

It is no partial observer that can form for us a philosophical and comprehensive system of medicine. It is not the mechanist; for, although the body is a machine, it is much more. It is not the chemist; for although the body is a laboratory, it is much more. It is not the vitalist; for the body is not disobedient to physical laws. It is not the humoralist; for the solids have also their specific properties. It is not the solidist; for the fluids may change of themselves, or be changed from without. It is not the empyric; for neither bodies, nor even the body, are always the same. Nor is it the morbid anatomist; for his dissections teach him little of causes, or of their relations with effects. It is to him who is not one, but all of these; who views the animal body as a machine of its own kind, obeying physical and chemical laws in unexampled complication, and further disguised by a combination with others peculiar to living structure; and who, *duly regarding all these powers,* seeks, in a change in their relations, the causes and the cures of disease; it is to THE PHYSIOLOGICAL PATHOLOGIST that I would look for the improvement of medicine; and to the combined exertions of many such, for the ultimate achievement of its greatest possible perfection.

SECTION II.—*Signs of Phthisis.*

In the early stage of phthisis, when the rational symptoms are seldom more than those of bronchial disease, or other pulmonary affections, such as short cough, with little or only

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tribution to these objects, and it is to be hoped that the train of probable constitutional and external causes of the origin of the disease which his sagacity has there developed, will become the subject of such careful study and extended observation, as may fulfil the inquiry which I allude to above.

pituitous expectoration, occasional pain or tightness in the chest, slight dyspnœa, or, rather, tendency to anhelation on exertion, more or less quickening of the pulse, and other symptoms of irritative fever, &c., the presence of tubercles, or of the indurations that precede them, will produce physical signs more or less appreciable, according to the situation and extent of the diseased parts. Thus the miliary indurations, even in considerable number, may be scattered through the tissue of the lung, without producing any distinct diminution or change in the resonance of the chest, or the sound of respiration. But if, (and it is the most common case) the induration or tubercular deposition be partial, or affect one part much more than others, then the sounds of respiration and percussion will be distinctly modified, and particular signs will be produced in the diseased parts. What these signs are, will appear from the physical change in the organ.

It requires more consolidation than is usual, in the doubtful stages of the disease, to produce any perceptible irregularity in the form or motions of the chest; but when considerable induration or tubercular deposition does exist in the apex of a lung, the upper part of the chest on that side may be seen and felt to move less in inspiration than the other.

Where the texture is considerably solidified by the disease, there the elastic resonance of the chest on percussion must be diminished, and the sound of vesicular respiration more or less obliterated; whilst those of bronchial respiration, and vocal resonance, are transmitted in an increased degree. Now, in by far the majority of cases, the principal accumulation of granular indurations, and of tubercular formation, takes place at the apex of the lung, which is immediately under the clavicle, and a small space below it.* If, therefore, this bone,

* The greater prevalence of tuberculous disease in the upper lobes of the lung is ascribed by Dr. Carswell to the inferior mobility of these parts favouring the accumulation of tuberculous matter, which he considers to be an excretion from

when struck about its middle, yields a dull sound, or duller on one side than on the other, it is exceedingly probable that the lung is in that part affected with phthisical degeneration. Great care must be taken to strike both clavicles at the same point, for the natural resonance is always less according to the distance of the point struck from the sternum. It is therefore generally expedient, to avoid error, to have the parts uncovered, and to tap the two clavicles alternately at corresponding points, with the middle finger, or the knuckle of the fore-finger.* When the disease is extensive, this dulness of percussionary resonance extends to the infraclavian region. In this part, gentle mediate percussion with the finger or a small plexi-meter, will give the most distinct signs. If the chest be struck forcibly, the parietes, overcoming the resistance of small or superficial solid deposits, will vibrate freely, and yield the deep sound of a healthy chest; but if struck mediately with moderate force, and with one or two fingers only, the irregularities of resonance produced by partial or superficial

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the mucous surface of the pulmonary vesicles, and which he supposes to be more freely removed from the inferior parts of the lung by expectoration. If this view be correct, there must be a much more copious expectoration of tuberculous matter, even in the earliest stage of phthisis, than has been generally noticed. Inclining to a somewhat different view of the nature of phthisical disease, I would regard its prevalence in the less mobile parts of the lung as a consequence of the greater proclivity of these parts to vascular obstruction and to the lower degrees of irritation arising from the incomplete expansion and contraction of the vesicular structure, and from the consequent imperfection in the process of aeration of the blood.

* The indications of percussion are sometimes deceptive, from the combination of a partial emphysema with the tubercular or miliary induration. This dilated state of the air-cells in the vicinity of indurated vesicles and bronchi, which is explained in the view which I have given of the pathological causes of emphysema, may counterbalance all effect produced by the indurations. This circumstance, which was first pointed out to me by my friend, Dr. Edwin Harrison, must increase the number of causes, in which even the physical signs are negative. But by this emphysema, the respiratory sound in the part will be diminished to a degree inconsistent with the healthy resonance on percussion, and the usual presence of a subcrepitant or submucous rhonchus would lead to a suspicion that something more than emphysema is present.

tuberculous deposits, will be distinctly perceived.* There is sometimes such an accumulation of tubercles also in the bronchial glands, and about the root of the lung, as to cause a dull sound on mediate percussion between the scapulæ.

The stethoscopic signs are more delicate, but, perhaps, more equivocal tests than those of percussion. When the tubercular induration exists in a degree even less than that required to change the percussionary resonance, the respiratory murmur will be less distinct than usual; or it may present somewhat of a whiffing or bronchial character; and a diffused bronchophony, or unnatural fremitus on the exercise of the voice, will be heard in the corresponding points of the chest. It is when they are more manifest on one side than on the other, that these signs are the most certain, and existing in points where the respiratory murmur is naturally quite vesicular and free from bronchophony; as in those parts of the infraclavian and acromial regions which are close to the head of the humerus. Towards the sternum, in the interscapular region, and in the axilla, these signs indicate the probable existence of tubercles, *only when there is a distinct difference between the two sides of the chest;* for the distribution of the bronchi in these parts is often such as naturally to produce similar phenomena. When the tubercular induration in the upper parts of the lung is considerable, it has the effect of conducting the sounds of the heart with great distinctness to the upper regions of the chest. This fact was, I believe, first noticed by Dr. Townsend. I have found it a useful confirmation of other signs; but the more complete consolidation of hepatization in these parts produces it in a still greater degree.

Such are the direct signs of tubercles and the granular in-

* These remarks are equally applicable to the investigation of pulmonary apoplexy, and circumscribed peripneumony or emphysema. They likewise suggest a source of errors which I have observed to be very common among auscultators.

durations; but usually there co-exists some inflammation of the bronchial mucous membrane, which adds to, and, perhaps, somewhat obscures these signs, by producing different catarrhal rhonchi; generally the sibilant and the sub-mucous. The pressure of solid matter on the air-vesicles, will also sometimes occasion a subcrepitant rhonchus. The long continuance of such a sign in the apex of a lung, especially if joined with a deficient resonance on percussion, would warrant a strong suspicion of the existence of tubercles.

It is to the concomitant bronchitis that we must ascribe the expectoration of the early stage of consumption. It is usually scanty, the cough being dry; but sometimes it consists of a thin glary liquid, which, when abundant, generally indicates numerous miliary granulations. A universal mucous rhonchus, with some dulness on percussion, and short breath, is a common attendant on one of the worst forms of the first stage of phthisis; and whenever these symptoms arise, in a subject disposed to tubercular disease, without being referrible to a common cold, there is reason to fear that the lungs are extensively invaded with granulations or tubercles, and that these will run a rapid course.

Hæmoptysis is a frequent but not constant precursor of phthisis, and it seems, in some cases, to be caused by the presence of miliary indurations and tubercles obstructing the circulation in the lung, and exciting a violent straining cough; and in others, in the form of pulmonary apoplexy, to precede, and probably occasion, the developement of the tubercles themselves.

I think, on the whole, that in the greater number of instances, the physical signs of this stage of tubercles are such as to give strong, but not conclusive, evidence, of an incipient phthisis; and cases, that are perfectly free from them may, accordingly, be regarded so much the more favourably. The practitioner should, however, be very guarded in pronouncing

on the nature of the disease at this period: he should study attentively the history and general symptoms,* and wait for the results of repeated examinations; when, if tubercles are really present, their signs will generally become more evident daily, and at last of a nature quite unequivocal.

It is when the tubercles, having passed into the softened state, become evacuated through the bronchi, and leave a cavity communicating with them, that the most characteristic signs of phthisis manifest themselves. In the cavity thus formed, the sound of the air passing in and out through the liquid that it still contains, is the first sign, and constitutes what is called *gargouillement, the gurgling or cavernous rhonchus.* It may be considered as an exaggeration of the mucous rhonchus, and it so nearly resembles that produced in the trachea and large bronchial ramifications, that the symptom must be considered doubtful, when heard only near the sternum, in the axilla, or in the upper part of the interscapular region; as it may here be produced in these air-vessels. The cavernous rhonchus will vary a good deal, according to the form and size of the cavity and the quantity and spissitude of its liquid contents. This variety may be easily conceived without description, on a consideration of the physical nature of the phenomenon. When this cavernous rhonchus is heard over an extended space, there are probably several cavities communicating with each other, and all containing a considerable quantity of liquid. When the subject is thin, and the cavity superficial, percussion in its vicinity sometimes produces a sound something like that of a cracked cup or jar, struck with the knuckle. This is only a modification of the

* To enter into an account of the general symptoms of phthisis, would require far more space than can be spared in this little work; and I can with confidence refer the reader to Dr. Clark's valuable article in the Cyclopedia, for the fullest and most accurate history that has ever been given of this disease.

gurgling, rendered somewhat metallic by the tinkling echo of the neighbouring bronchial tubes.*

As the liquid contents of a cavity are evacuated by expectoration, the cavernous rhonchus passes into what is called the *cavernous respiration.* The sound of this is very characteristic, and represents to the mind exactly the passage of air in a cavity. It has not the diffused sound of vesicular respiration, is more sonorous and circumscribed than tracheal, and in different examples may be very perfectly imitated by blowing into shells of different sizes. As cavernous respiration differs according to the size of the cavity in which it is produced, so the variety of sound may be taken as a means of judging of its size; the rule generally being that the deeper and hollower the sound of cavernous respiration, the larger is the cavity.*

There are, however, other sources of variety which it may be useful to notice. Thus the sound may be like the blowing of a bellows, or contracted almost to a whistle, according as the communication with the bronchi is free or constricted. If, with a contracted bronchial orifice, the cavity be of a large size, particularly if lined with a rigid false membrane, the respiration will become *amphoric,* or like the sound produced on blowing into a phial; partaking of the character and ex-

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* This sound may frequently be produced in the dead body by percussion under the clavicles; but it does not here always indicate a cavity, as its seat is usually in the trachea and larger bronchi.

† Dr. Spittal seems to object to this statement, and remarks that the size of the aperture is another cause of variety, just as the tone of blowing into an empty phial can be lowered by partially closing its mouth. I must, on my part, object that this case is not quite a parallel one; the sound here is the result of two simultaneous currents of air, one entering, and the other leaving the bottle, and the alteration of the tone, by the size of the aperture, arises from the degree of condensation, caused by the entering current, (as in closed panpipes.) In pulmonary cavities, on the other hand, unless their walls be very rigid, the air passes to and fro alternately, as the cavity is compressed or dilated by the respiratory movements. The size of the communication with the bronchi may affect the sound in the manner described in the next paragraph.

planation of that before described as occurring in pneumothorax.

The thickness of the parietes of the cavity do not, so much as might be expected, modify the sound of cavernous respiration. It may sometimes be heard distinctly through healthy tissue more than half an inch in thickness, and seems then more distant. When the cavity is more immediately contiguous to the parietes of the chest, the sound appears so near, that the auditory impression is like that of blowing into the ear itself. Sometimes it comes only in successive puffs, apparently interrupted by something moveable suddenly interposed; and this, according to Laennec, occurs when a very thin curtain of pulmonary tissue, still spongy, forms the parietes of the cavity immediately opposed, but not adhering, to the pleura costalis. The physical cause of intermittent puffing I presume to be the filling and emptying of this loose portion of lung during the respiratory movements.

The cough, which may be considered an exaggeration of respiration, gives the same varieties of character as this latter, and sometimes may produce the signs, when ordinary respiration does not effect the entrance of air into the cavern. When the cavern has only recently communicated with the bronchi, or when it is again nearly filled with sputa, the air enters it and produces the cavernous rhonchus, only in the fuller inspiration that accompanies cough. In other cases this respiratory movement often renders the signs of the existence of a cavern, more unequivocal and decisive. In a cavern that is vast in size, and pretty simple in form, and having only a narrow orifice communicating with the bronchi, the respiration may be insufficient to have such free access to it as would give the sign of amphoric resonance; but the cough would not fail to make its existence known, by the tinkling echo, (tinnitus metallicus,) that, as in pneumothorax, would accompany it.

Another most distinct and certain sign of a cavity in the

lungs is furnished by the voice. We shall best understand this by referring to what we have formerly said on the production of the voice. That reverberation which exists in the trachea and bronchial tubes of larger size, (we then observed,) is so broken down and destroyed in the finer complication of the cellular parenchyma, that either the voice is not propagated at all through it, or only in a dull diffused fremitus. But if a cavity be formed in this parenchyma, and a prolongation of the bronchial *sounding board* thus produced, the voice will then be heard in the corresponding part of the chest, in a tone and intensity more or less perfect, according as the cavity is adapted to receive and transmit the vocal resonance from the bronchi.*

When the cavity is of moderate size and regular form, empty or nearly so, and in free communication with a large bronchial tube, and either it is very near the surface of the lung in contact with the thoracic parietes, or the intervening tissue is rendered a good conductor by condensation, the voice is transmitted in the most perfect and unmodified manner, and seems to be produced in that spot of the chest, seemingly distinct from the oral voice. This is *perfect pectoriloquy*. If heard with the stethoscope, (which for this purpose is best adapted with the stopper in) the sound of the voice seems to come through the tube, and enters the observer's ear louder than that which, coming from the patient's mouth, strikes the other ear; but the verbal utterance is never so distinct.†

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* I have before explained that this phenomenon depends on the air in the bronchial tubes, and cavities communicating with them, constituting vibrating systems with the voice, in greater or less perfection.

† How often does the voice thus heard make known a melancholy truth the speaker never dreamed of! More than once has it occurred to me that the very words which, in that delusive confidence with which this malady enshrouds its victims, ridiculed my examination of the chest, roundly saying that nothing ailed them there, have belied their meaning, and coming from the breast, have told a far different tale!

When heard to the degree just described, in parts where there is naturally little or no resonance of the voice, it proves (equally with cavernous respiration) beyond doubt the existence of a cavern communicating with the bronchi. All the circumstances above mentioned not often occurring together, perfect pectoriloquy is of much rarer occurrence than the other signs of cavities.

It is less certain when occurring near the sternum, in the axillæ, and between the scapulæ; but if the resonance seems defined, passes up the tube of the stethoscope, and is heard to this degree on one side only, there can be little doubt even in these cases: for the natural bronchial resonance of these parts scarcely ever passes the tube, is generally diffused, or can be traced in a line along the course of the bronchus, and is rarely very unequal on the two sides. That part of the acromian region in the angle formed by the junction of the clavicle and coracoid process of the scapula, may admit of examination; for if the stethoscope is held nearly perpendicularly on this spot, it is out of reach of the laryngophony heard in other parts of this region. It may be held in mind that natural bronchophony exists most in young or thin subjects, with a sharp voice, and is generally slight in those who are stout made and have deep voices: but the same circumstances are also favourable to the distinct development of pectoriloquy.

Imperfect pectoriloquy is that kind which does not seem to enter the stethoscope, but only to resound at the end. This standard of imperfection will not, however, always denote the uncertainty of the sign; for what is, according to this definition, imperfect pectoriloquy, if it occur where it cannot be confounded with natural resonance, particularly if confined to one side, may be deemed a pretty certain indication of the existence of a cavity. It cannot be trusted to when heard in the sternal half of the infraclavian and mammary regions, the axillæ and interscapular spaces. Neither is it always possible to make this distinction of pectoriloquy into *perfect* and

imperfect, although in the extreme degrees it is sufficiently apparent; for sometimes it seems only partially to enter the tube, some words being loud and near, and others in the same sentence more distant: a very bass voice scarcely ever seems to traverse the tube completely.

The smallness of the size of a cavity, its imperfect communication with the bronchi, or its distance from the surface, may render pectoriloquy of the imperfect or doubtful kind; hence these conditions being changed, (as often happens in the course of the disease) the pectoriloquy will again become perfect, and *vice versâ*. So likewise doubtful signs of a cavity may be taken as certain, if they arise suddenly in a spot where such had never before been observed; particularly if this happen after coughing and expectoration.

The vocal resonance is sometimes modified in pectoriloquy. This not unfrequently happens when there is a little liquid in the cavity, which being occasionally raised in bubbles, interrupts the sound and gives it somewhat of a saltatory and slightly tinkling character. The vocal resonance is also sometimes alternated with puffs of cavernous respiration; for it may happen, if the communication with the bronchi is small, that a little increased force in the passage of the air through the cavern, may momentarily prevent the transmission of the vocal vibrations into it. Cavities of irregular form, with partial septa and soft loose parietes, do not produce so perfect a pectoriloquy as those of simple form and smoothly lined. Those of moderate size perhaps are the best adapted to produce the symptom; but Laennec relates an example where a cavity not larger than a plum stone produced a distinct pectoriloquy. When the cavity is very large, the communication with the bronchi is often not sufficient to transmit the full vibrations of the voice to the large volume of air in the cavity. In that case, a tinkling echo accompanies the voice and the cough, as in pneumothorax; and this echo

will be more metallic and resounding, in proportion as the cavity is ample, and its parietes smooth and tense. Percussion, as well as the history of the case, will generally suffice to distinguish this case from pneumothorax: the sound is never so clear, and when mediate percussion is used, it may often detect irregularities from the varied density of the subjacent parts, which do not appear in the elastic drum-like sound of pneumothorax.

The pectoriloquy, produced in a string or row of small excavations, frequently presents much of the character of bronchophony; the tone of the voice being rendered more sharp, and somewhat cracked. It may be sometimes difficult to distinguish between this, and bronchophony produced by a partial inflammation: the history may generally determine whether an ordinary pneumonia has taken place; but if an inflammation of the intercurrent kind has been engrafted on a chronic catarrh, or other affection of the lungs, the symptoms of the latter may have masked its progress, and it may thus have escaped observation. However, in this case there is generally some indication, either in the expectoration, the remains or return of the crepitant rhonchus, or the cessation of the bronchophony, that will be sufficient to distinguish the inflammation. An intercurrent circumscribed inflammation frequently occurs in lungs affected with tubercles in different stages; and in a greater extent is not uncommonly the immediate cause of death. The checking of such inflammations at their commencement is a principal object in the treatment of phthisis; for, besides the immediately fatal effect that they sometimes determine, we have seen that they also tend to accelerate the progress of the tubercular disease. Hence the expediency of frequently using the stethoscope in phthisical cases, to ensure the detection of the inflammation at a time when a few leeches or a counter-irritant may be sufficient to check it, without wasting by depletions the little pittance of

strength that might still hold on existence a short period longer.

I formerly mentioned, when speaking of chronic bronchitis, that the signs of an extensive dilatation of the bronchi resemble those of a tuberculous cavity in phthisis. Where a bronchus is dilated at a point to the size of a hazel nut, or even of a plum stone, it is plain that it is physically capable of producing all the signs of a tuberculous cavity of the same size; that is, a coarse gurgling rhonchus, if it contain liquid; and cavernous respiration and pectoriloquy, if it be empty. The case of such a dilatation is not very common, but it does occur; and as the other symptoms resemble those of phthisis, it may be mistaken for this disease. In time, however, a distinction may be made by the signs of progress which the tubercular disease generally presents; the cavity becomes more extended, and causes signs of *hollow*, more remarkable and extensive than dilated bronchi could produce; and not unfrequently cavities are formed in other places. Dilated bronchi do not often affect the sonoriety of the chest on percussion, to the degree that the engorgement and infiltration about the tubercular cavities do; nor can they produce the other physical signs of large cavities. The most common situations of dilated bronchi are, in the scapular, mammary, and lateral regions: the subclavian and acromian are the more usual seats of phthisical signs.

There is generally some condensation of tissue in the neighbourhood of tubercular cavities; owing either to the deposition of other tubercles in a crude state, or to the effect of chronic inflammation. Hence it seldom happens that the chest recovers its natural resonance on percussion on the corresponding points, even after the contents of a cavity have been evacuated. When the cavity is large and superficial, the chest may sound well in some places over it, but its resonance is irregular, and less elastic, and if the other side is still

in a healthy state, a remarkable but indescribable difference may be both heard and felt by the percussor. Sometimes the sound is almost absolutely dull, owing to the flaccid walls of a cavity acting as a damper on the sonorous vibrations; but the feel thus given to the fingers striking, is very different from that of percussion over a solidified lung. It is perceptible that the walls of the chest yield, and not unfrequently a sort of gurgling or fluctuation may be felt under the fingers.

I have mentioned that fistulous cavities have a tendency to increase in size; and this may be effected either by the softening and evacuation of neighbouring tubercles, or by simple ulceration of the adjoining tissue. Thus the symptoms will, by the extension of the disease, become more and more evident, in too many cases, and soon put the diagnosis beyond a vainly-hoping doubt.

If the cavity be near the surface of the lung, and there be no adhesion of the pleura at the spot, there is a chance of a perforation of the pleura taking place, and producing a pneumothorax. This accident is commonly manifested by the sudden occurrence of the general signs of acute pleurisy, caused by the irritating effect of the tubercular matter and air upon the pleura. I have already described pneumothorax produced in this manner; and refer to what is said in the section on this subject for an account of the physical signs. They are often very remarkable. I have heard, in a case of this kind, an amphoric respiration, so loud that it could be distinctly perceived without applying the ear or the stethoscope to the chest, and, I think, must have attracted observation, if I had known nothing of auscultation.

The evacuation of a softened tubercle into the pleura, without communication with the bronchi, is of more rare occurrence. It generally produces a pleurisy, which is remarkable for the suddenness of its attack. The effusion is sometimes accompanied with a disengagement of gas in the pleura. If

the phthisical disease is not far advanced, the effusion may be absorbed, and adhesion may be formed between the pleuræ; but the shock of acute attack is often too great for the wasted tottering frame to bear, and the patient falls under it.

The character of the sputa has been more consulted in consumptive diseases, than in any other; but as the distinctions insisted on have been founded on the erroneous notion that the expectoration of pus is characteristic of the second stage of phthisis, the earlier opinions on this subject are fallacious and unprofitable.

The characters of the sputa in the second stage of phthisis, or during the softening and evacuation of the tubercles, would be much more distinctive, were they not almost always mixed up with the mucous and muco-purulent secretion of a chronic bronchitis, which more or less prevails at this period. Hence the signs as relating to the tuberculous disease, must be considered in a corresponding degree ambiguous. Attentive examination will often discover, in the mucus expectorated, fine whitish streaks, which consist of the softened tubercle; more rarely there are little yellowish white masses, like grains of boiled rice, which are portions of crude tubercle. As the softening proceeds, and the cavities are enlarged, the sputa become less frothy, sink in water, and are principally composed of greenish white masses of irregular shape and outline, sometimes tinged in parts of a dirty red or brownish colour, occasionally streaked with blood. These flatten at the bottom of the vessel, like a piece of money, whence they have sometimes been called nummulary sputa. In some rare instances, small portions of the pulmonary tissue itself have been detected with the preceding. When the disease is farther advanced, the expectoration assumes a brown, dirty green, or grey colour, each sputum being frequently surrounded with an areola of a bloody tinge.

It is exceedingly difficult to say what degree of weight should

be attached to these several appearances, even where they are unequivocally seen. It might be supposed that the presence of the whitish streaks, or of the little white masses, would be conclusive as being themselves tuberculous matter; but appearances of the same kind may proceed from other sources. The minute bronchial ramifications, in chronic inflammation, sometimes secrete a purulent liquid, which may produce the same streaky aspect; and vermicular concretions and filaments of yellowish white lymph, formed in the same way, may be mistaken for little fragments of tubercle. The little rice-like bodies, which were considered by Baglivi and Bayle, and even by Hippocrates, as indications of phthisis, are moreover closely simulated by the sebaceous concretions formed in the tonsils; and, according to Andral, by similar productions from the follicles in other parts of the pulmonary mucous membrane. The white matter from the tonsils may, however, be always distinguished, as Laennec has pointed out, by their fœtid odour, and by their greasing paper when heated. Globular yellowish-white masses, like irregular balls of flock or wool, apparently consisting of pus held in shape by a little tenacious mucus, have been noticed by several writers, as peculiar to phthisis. A precisely similar appearance is, however, sometimes presented by the sputa in chronic bronchitis. The dirty brown or greenish matter, flattening and becoming nummulary when separate, and when together forming a smooth sluggish *purilage*, which appears later in the disease, and takes its origin from the tubercular excavations, is much more certainly characteristic of phthisis. To sum up, we may say that an occasional examination of the sputa, by far the greater part of which proceeds from an accompanying bronchitis, rather than from the tubercular disease itself, can only enable us to distinguish phthisis in the very rare case of tuberculous matter, or portions of the tissue of the lung being present; but with Dr. Forbes and M. Andral, I think, that

by a careful daily inspection of the expectoration, we shall not fail to find, in the successive and progressive changes which it presents, the means of forming a pretty accurate diagnosis, which, if confirmed by the general and physical signs, will seldom leave a shadow of doubt.

After the contents of a tuberculous cavity have been evacuated, there is generally lymph thrown out, which, if not removed by the ulceration of the adjoining tissue, becomes in time converted into a membranous lining of the excavation, at first serous, secreting a serous or sero-purulent matter, but afterwards becoming denser, of fibrous or fibro-cartilaginous texture, and yielding only a scanty secretion.

The formation of this membrane is the effort of nature towards the cure of phthisis; and the researches of Laennec have given satisfactory proofs that this effort is not always unsuccessful. The process is simple. The fibro-cartilaginous membrane gradually contracts, until it at length nearly obliterates the cavity, and becoming smaller and more dense, draws in the pulmonary tissue, and at length forms little more than a kind of cicatrix, around which the puckerings of the texture are very remarkable. The signs of such change are the diminution and gradual cessation of the symptoms of a cavity: and if the cavity was large, its cicatrization will produce some contraction of the chest on that side.

Many, very many, are the causes that prevent the success of this process of nature. Often, if the lungs are not already too extensively pervaded by tubercles to leave enough pulmonary tissue after their evacuation and the cicatrization of the cavities, to support life, the constitutional cause engenders more; so that, while some cavities are healing, other tubercles are generated, which may be fatal either by their present abridgment of the pulmonary function, or, by the exhaustion produced by the ulcers entailed by their suppuration. Add the number of contingent causes that may prove fatal

before this curative effort can take effect; hæmoptysis, inflammation, suffocation by sudden bursting of a vomica, perforation of the pleura, bodily exhaustion, constitutional complication—these, and many others coming in the deadly train—and we shall see how diminutive is the chance of recovery from phthisis.

The case that should afford us hope is that in which the pulmonary organic disease seems to be limited, the function little embarrassed, the body not much reduced, and neither harrassed by a complication of complaints, nor tainted by hereditary diathesis. Even others of worse aspect *may* turn out favourably; experience has proved it; and although in them our anticipations cannot be brightened by hope, neither may they be totally darkened by despair. And thus, if the study of physical signs shall often appal us by discovering the dreadful enemy that holds an object of our care, it has also established the consolatory fact, that his grasp is not universally relentless.

Section III.—*Encephaloid Disease, Melanosis, &c.*

There is little need that I should detain the reader by a detailed notice of some organic diseases of the lungs, of rare occurrence and obscure nature; for whatever is known of them will suggest to him, now accustomed to the acoustics of the chest, the physical signs that are likely to accompany them. The lungs may become solidified in various ways by the deposition in them of morbid matters similar to those which invade other parts of the body. Thus melanosis, encephaloid, or medullary sarcoma, fungus hæmatodes and scirrhus, are occasionally developed in the pulmonary tissue; and they may occur in a circumscribed form, or diffused through a considerable extent of texture. They would then produce the same physical signs as tuberculous disease of analogous extent, and could be distinguished from this only by the general

symptoms, and the absence of the constitutional indications of tubercle.

These diseases usually prove fatal, either by encroachment on the function of the lungs, or from being simultaneously developed in other organs (especially in the liver), before the process of softening and ulceration can be effected. To this, nevertheless, they certainly tend; for I have seen instances, and others are on record, of ulcerous cavities formed in melanose and encephaloid solidifications of the lung; and the expectoration, in one case of a black and red, and in the other of a streaky whitish and sanguinolent purilaginous matter, led to a suspicion of the nature of the diseases, before death. In such cases the stethoscope would indicate the gurgling and other signs of a cavern in the lung.

The general signs of these affections are more commonly those of obstructed circulation, dropsy, &c., with oppressed breathing, than those of emaciation, common in tuberculous disease. The difference in the duration and progress of these several morbid productions sufficiently explain the variety in the symptoms; and I am disposed to think that these differences proceed in great measure from the mechanical condition and degree of vitality in which these matters are deposited from the blood, as I have endeavoured to point out analogous circumstances in the nature and changes of tubercle.

The albuminous matter of encephaloid disease is obviously deposited in an organizable form, but which yet falls short of the vitality of ordinary false membrane. The globules of which it is composed unite together, and vascular ramifications are formed through them; but they have no cohesive power to restrain the further effusion from the vessels: hence the tendency to growth in tumours formed of this matter. When it occurs in an infiltrated form in the tissue of the lung, it sometimes presents an appearance intermediate between that of tuberculous and that of hepatized solidification, and unless

there are portions of the diseased production occurring separately, it might be viewed as one or other of these affections. A predominance of loose vascular structure, with patches of effused blood, give to the encephaloid matter the characters which have obtained for it the name *fungus hæmatodes.*

The matter of melanosis or black tubercle may occur infiltrated in a natural structure, or in distinct tumours or deposits of an irregular cellular organization. The black matter appears to be a modification of the colouring matter of the blood, in which carbon is in excess, or even in a free state. The organized texture of melanose tubercles and tumours, presents great variety in its degrees of perfection, sometimes approaching to the most vital products of inflammation, in others approaching to the inorganic structure of scrofulous tubercle. The modification of the colouring matter of the blood, seems therefore to be the essential pathological condition of this disease, as an altered or deficient vitality of the fibrinous globules is of tuberculous affections.

The black matter which is found in the lungs of the older inhabitants of large towns, is a carbonaceous matter, and beyond doubt is derived from the soot inhaled in the air, as first supposed by Dr. Pearson. Its deposition and permanency in the tissue of the lungs, is a proof, not (as Majendie maintained in the analogous case of the carbonaceous matter of tattoed skins, and of the insoluble oxide in persons coloured by the internal use of nitrate of silver) that there are no textural reparation and absorption, but that this absorption cannot act on insoluble solid matter. I believe that this black matter finds access to the pulmonary texture principally through abrasions, softenings, or other lesions of the bronchial mucous membrane, slight injuries of this kind being common accompaniments of an ordinary cold and cough. This coaly dust does not appear generally to produce any injury to the function of the lung, but getting into any corners out of the

immediate sweep of the circulation, such as in the angles of the lobules, on the sides of large vessels, in cicatrices of old lesions, and in the bronchial glands, it remains slowly accumulating until death, or until it is carried off, in expectoration, by some pulmonary disease. But there are some curious instances on record, in which this accumulation has taken place so rapidly and extensively as to infringe on the function of the lung, producing œdema and a black consolidation of the tissue, which tends to ulceration and the formation of cavities. A case has been described by my lamented friend, Dr. J. C. Gregory,* in which the habitual inhalation of the dust of a coal mine had produced a disease of this kind, accompanied with the cachectic anasarcous state consequent on the obstruction to the pulmonary function. Other cases are related by Dr. W. Thomson,† in which a similar affection could be traced to continued employment by the light of lamps which give out much sooty smoke. In these instances an additional cause of pulmonary disease seems to be required to produce the black infiltration in the manner which I have mentioned; and this cause was presented either in prior bronchial complaints, or in the occupation of the individuals as stonemasons, which of itself frequently produces severe lesions of the bronchial membrane, and even of the pulmonary tissue. In Dr. Gregory's case the coal dust was the mechanical irritant as well as the colouring matter.

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* *Edinb. Med. and Surg. Journal*, No. 109.

† In a paper read to the Medical Section of the British Association, Edinburgh, 1834. See also a paper by Mr. Graham of Glasgow, on Charcoal in the Lungs. *Edinb. Med. & Surg. Journal*, No. 121.

Section IV.—*Diseases of the Bronchial Glands.*

It is very common to find the bronchial glands exhibiting traces of disease even in cases where no symptoms of it had been indicated during life. The presence of the black matter, like that in the lungs, can scarcely be called a disease, since scarcely any adult is entirely free from it. But besides this, the glands are sometimes enlarged, their unstained parts being of a reddish colour; and as they have been found in this state when the adjoining parts of the lungs have been inflamed, there is reason to believe the tumefaction to be caused by inflammation. Laennec says that he has met with abscess in them in a very few instances.

They are frequently the seat of tuberculous deposit; and I have seen instances, both in children and in the adult, of its formation in them exclusively, no other part of the body shewing any trace of tubercles. When thus tuberculous, they sometimes attain the size of a pullet's egg, and by pressure on the trachea or bronchi, or on the blood-vessels, induce dyspnœa and obstructed circulation. Dr. Carswell considers this to be not uncommon in children; and he would ascribe to it the dyspnœa occurring in them, without signs of other disease, when the chest continues to be sonorous on percussion, and the respiration every where audible though weak. Tuberculous matter in the bronchial glands is liable to the softening, and to the drying and cretaceous conversion which we have noticed to be its course in the lungs. The softened matter may be evacuated through the bronchi, leaving a fistulous cavity; and M. Guersent describes this as not an uncommon case in children. Dr. Carswell notices an instance of a child, in which this softening and ulceration proved fatal, by opening a branch of the pulmonary artery.

The calcareous matter found in the bronchial glands, does

not appear to be always the dry remains of tuberculous deposits; for I have seen calculous concretions in them which are obviously different from the gypsum-like matter of old tubercles, and without the enlargement which the scrofulous tubercle generally produces.

Encephaloid disease may attack the bronchial glands, from which it sometimes extends to the tissue of the lung. A remarkable case of this kind lately occurred at St. George's Hospital. The disease constituted a tumour in the mediastinum, which involved and partially obstructed both the blood vessels and trachea; and nearly the whole of the left lung was solidified by an infiltration of the same matter. The mixture of black matter in the lung, and the presence of two cavities, gave it a resemblance to tuberculous disease, from which it would scarcely have been distinguished but for the presence of the reddish white organized matter in the root of the lung, and which extended into, and was gradually lost in, the solid grey matter of the lobes. The symptoms in this case were partial œdema, and lividity of the face and neck, and other signs of obstructed circulation and respiration.

The physical signs of considerable tumours of the bronchial glands, would be dulness on percussion in the upper portions of the interscapular regions, and on the spinous processes of the upper dorsal vertebræ. Signs of excavations there would be rendered doubtful by the bronchophony and, in thin subjects, pectoriloquy, naturally existing in these spots.

PART III.

AUSCULTATION OF THE HEART.

In both the former editions of this work, I expressed a distrust with respect to our knowledge of the signs produced by the motions of the heart: and although the subject has engaged the attention of many ingenious reasoners and enterprising experimenters, I must confess that it appears to me to be in several respects still involved in uncertainty. In giving to these topics, therefore, a consideration somewhat fuller in this than in the former editions, I do not hope to make physical signs illustrate pathology, in diseases of the heart, as I have endeavoured to do in other affections of the chest; but it may be useful to separate and appreciate the points that really are certain and useful, from those that are doubtful or ill-defined, both with a view to make our present knowledge safely available to the student, and to guide future inquirers in further investigations. The uncertainty which involves the physical signs of diseases of the heart, depends not only on our ignorance of the relations of many lesions and morbid motions, as causes of sound, but also on the doubt which still hangs over the physical origin of the natural sounds. The valuable researches of Dr. Hope have done much towards the elucidation of both these subjects; and with the advantage of the light which he has obtained for us, there is reason to hope that a few well-devised physiological experiments, and an extended series of cautiously examined clinical cases,

will furnish us with the desired knowledge, and render the diagnosis of diseases of the heart more clear and satisfactory. I hope to be able, before the conclusion of this work, to attempt the experimental investigation to which I allude; but the clinical observations, to be satisfactory, must be a work of considerable time and patient attention.

CHAPTER I.

AUSCULTATION OF THE HEART IN HEALTH.

On applying the ear to the region of the heart in a healthy person, a sound is heard at each pulsation, followed by an interval of silence. This sound is double; consisting of a dull slow sound, immediately followed by a short quick one. The first sound is produced by the systole of the ventricles, and is synchronous with the pulse of arteries near the heart. The second or short one accompanies the ventricular diastole. Laennec rates the relative duration of these, in each ordinary pulsation, to be as follows: the first sound, two-fourths; the second sound, one-fourth, or a little more: the interval of silence, one-fourth, or a little less. These sounds are naturally most distinct in the space between the cartilages of the fourth and seventh ribs of the left side, and on the lower part of the sternum; the former part corresponding with the left, and the latter with the right side of the heart. Beyond this space the sounds are rarely distinct, in persons of good proportion and middling stoutness. In thin subjects, they are heard over the whole front of the chest, and sometimes reach to the left dorsal, and rarely to the right dorsal regions. I have very commonly found them audible through the vertebræ between the scapulæ.

Simultaneously with the first, or systolic sound, an impulse or shock is communicated to the stethoscope. It is most perceptible at and between the cartilages of the fifth and sixth ribs, where it may be felt by the hand; but the stethoscope commonly renders it sensible in lean persons over the whole præcordia; and if the sternum is short, in the epigastrium also.

There is in healthy persons a very marked difference in the strength of the impulse, according as the ribs rise and fall in respiration. When the chest is fully expanded, the margins of the lungs overlap the pericardium, and by their soft yielding substance in some degree intercept the propagation of the impulse to the parietes. After expiration, on the other hand, the apex and a portion of the body of the heart come in immediate contact with the walls of the chest, and the pulsations may be felt more distinctly and over a greater extent. A considerable thickness of fat or œdema will more or less intercept the impulse; and so also may considerable emphysema of the adjoining lobes of the lung, and effusions of air or liquid within the pericardium. Considerable variety in the force of the impulse may occur from various extraneous causes acting on a healthy heart. Thus the pressure of tumours behind it, flatulent distension of the stomach, great enlargements of the liver or spleen, contraction of the chest from pleurisy, deformity from rickets, and similar causes, which have the effect of pushing the heart into closer contact with the anterior walls of the chest, will make its impulse against them stronger. Again, extensive effusions of air or liquid, in the left pleura, may displace the heart, so that its impulse can only be felt under, or even to the right of the sternum, or in the epigastrium. Their proper physical signs will sufficiently distinguish such causes; and these displacements of the heart, as long as its sounds are healthy, are to be considered in reference rather to these other affections than to the pathology of the heart.

The action of the heart is naturally accelerated by exercise, stimulating drinks, heat, &c., and this quicker action is attended with an increased impulse and with louder sounds. Febrile and inflammatory diseases have the same effect; but it is important to notice, that in a healthy heart these causes of excitement do not perceptibly alter the rhythm or order of the motions, and the sounds and impulse still bear a general relation to the strength and frequency of the pulse. In diseases of the heart, on the other hand, whether functional or organic, these relations are variously altered, and the sounds and impulse present great diversities not only in proportion to each other, but also in their specific characters. It is useful, therefore, for those unaccustomed to the auscultation of the heart, to familiarize themselves with the varied phenomena which the different circumstances above-named may produce in the healthy heart. So also where the sounds of the heart are obscure, we may render them distinct by making the patient walk briskly for a few minutes. It is to be remarked that the increased loudness and strength of the pulsations is in these cases accompanied with an increased quickness; but in many cases of disease of the heart, the sound becomes louder or duller at the ordinary rate of pulsation. There are varieties, among different individuals in health, as to the extent of the audible pulsations; and I believe that the remark made by Laennec on this point, may be modified to this effect,—that the extent of pulsation of the heart is in proportion to the size of the ventricles, the thinness of their walls, and the abruptness of their contractions.

CHAPTER II.

AN INQUIRY RESPECTING THE MOTIONS AND SOUNDS OF THE HEART.

In the last edition of this work, I introduced, in the form of an appendix, a summary sketch of the various notions which have been advanced respecting the ordinary motions and sounds of the heart; and by comparing them with well-established physiological or pathological facts, I was enabled to set aside as untenable the greater number of these views, and to state with some precision the weak or uncertain points of those which still deserve consideration. As this sketch will still serve to illustrate the argumentative history of the subject, I introduce it in a note below;* and it will remain for me in this chapter to consider those views between which there have not been hitherto facts sufficiently conclusive to decide.

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* It is of considerable utility in the examination of a controverted point, to review fairly the various opinions respecting it, and by collating them with available facts, to determine the comparative probability of these views: if this had been done with regard to the present subject, much useless speculation might have been saved, and some animal life spared; for any attentive reader of the periodic medical literature, must have perceived that the same opinions have been broached, refuted, and revived by successive writers, and the same experiments performed and reiterated, in apparent ignorance of preceding enquiries. Others, besides the authors quoted, may have supported the following views on the subject in question, but it is only the views which I wish to deal with, and I name the writers to shew that the arguments which each has advanced have been carefully studied.

1. M. Laennec. *a*. 1st sound, impulse, and pulse, caused by the ventricular systole. *b*. 2nd sound by the systole of the auricles.—*Remarks.* *a*. Generally admitted, and proved by various facts and experiments. *b*. Disproved by the fact noticed by Harvey and Haller, and confirmed by modern experiments, that the auricular contraction immediately precedes that of the ventricles: also by this fact, that both sounds sometimes continue after the auricles have ceased to contract.*

* Dr. Hope's Experiments on Asses. See his Work, p. 36.

From the statements below it may be considered as demonstrated by former experiments:—

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2. Mr. Turner.* 2nd sound produced by the falling back of the heart on the pericardium after the systole of the ventricles.—*Remark.* Disproved by the fact that the sound continues when the heart pulsates out of the pericardium.

3. Dr. Corrigan.† *a.* Impulse and 1st sound caused by the rush of blood into the ventricles during the auricular systole. *b.* 2nd sound by the ventricular systole, which he considers to be instantaneous.—*Remarks.* *a.* Disproved by the clearly ascertained facts that the 1st sound and impulse accompany the systole of the ventricles when the auricles have ceased to contract. *b.* Disproved clearly in large animals by the ventricular systole, (which is not instantaneous) and the pulse of arteries near the heart, evidently preceding the 2nd sound;‡ and further disproved by several pathological phenomena.

4. Dr. David Williams.§ 2d sound caused by the flapping open of the auriculo-ventricular valves against the sides of the ventricles; these valves he supposes to be opened by the musculi papillares.—*Remark.* This is contrary to the received opinion of anatomists with respect to the functions of the auricular valves and musculi papillares, and there is no collateral argument to maintain so gratuitous an assumption.

5. M. Pigeaux.‖ *a.* 1st sound produced by the blood rushing into the ventricles at the moment of their diastole. *b.* 2nd sound by the collision of the blood against the walls of the aorta and pulmonary artery. *c.* The ventricles contract in a moment of silence before the 2nd sound. *d.* The intensity of the sounds proportioned to the force by which the blood is impelled.—*Remarks.* *a.* Opposed by the facts stated against 3 *a*; opposed also by many pathological facts, such as the occurrence of a murmur with the 1st sound, in case of diseased semi-lunar valves. *b.* Disproved by the fact that the 2nd sound occurs distinctly *after* the pulse in the carotids, and therefore after that in the larger arteries. *c.* Opposed by the observation that the 1st sound and ventricular systole occur together and correspond in duration. *d.* This is opposed by the morbid phenomena of dilatation of the ventricles, which always increases the first sound, and of hypertrophy, which diminishes both sounds.

6. M. Majendie.¶ 1st sound and 1st impulse produced by the ventricular systole impelling the apex with a shock against the walls of the chest between the fifth and sixth ribs; the 2nd sound and 2nd impulse by the blood, in refilling the ventricles at their diastole, forcing their parietes with a shock against the sternum. —*Remark.* Completely disproved by the fact that both sounds continue when the heart does not touch the parietes of the chest.**

* Med. Chir. Trans. Edin. vol. iii. † Trans. of King's & Queen's Coll. of Phys. Ireland.

‡ Dr. Hope's Experiments, p. 31 of his work; and those of Mr. Carlile, Dublin Journal of Medical Science, vol. iv.

§ Edin. Med. & Surg. Journal, Oct. 1829.

‖ Arch. Générales de Médecine, Juillet et Novembre, 1832.

¶ In Lectures delivered at the College of France, in 1834, reported in the Lancet, Feb. 1835.

** See the Experiments of Dr. Hope, (p. 30 et seq. of his work) of M. Bouillaud (Journ. Hebdom.) and my own, as described further on in the text. Many other facts might be stated as conclusive against this "last new view," but the above-named one seems to me quite sufficient.

1st. That the auricles contract first, producing no sound.
2nd. That the auricular contraction is immediately followed

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7. M. Rouanet.* *a.* 1st sound caused by the closing of the mitral and tricuspid valves against the auriculo-ventricular orifices during the ventricular systole. *b.* 2nd sound by the reaction of the blood in the arteries on the semilunar valves at the moment of the ventricular diastole.

8. Mr. H. Carlile.† *a.* 1st sound produced by the rush of blood into the arteries during the ventricular systole. *b.* 2nd sound by the reaction on the semilunar valves as stated in *b.* 7.

9. Dr. Hope. *a.* 1st sound and impulse, caused by the ventricular systole. *b.* 2nd sound and *back stroke*, or 2nd impulse, by the ventricular diastole. The natural as well as morbid sounds produced by the motions of the contained fluid.

Before we sift the questionable points in these three last views, it will be proper to review the principal grounds on which we adopt their description of the sounds and motions, in defiance of many preceding authorities. Having been present at some of Dr. Hope's experiments on the ass, I had ample opportunity of convincing myself that the sounds were connected with the motions of the ventricles only. When the pericardium was laid open, and the large heart exposed, vigorously pulsating; the eye watching it, the hand grasping it, and the stethoscope applied to it, gave perfectly corresponding impressions; insomuch, that on substituting touch for hearing, it was difficult to banish the impression that one still *heard* the double sound which was so exactly represented in quality and duration by the motions of the ventricles, as felt and seen; and on combining touch and hearing, by applying the hand and the stethoscope at the same time, these impressions, which corresponded in nature and duration, were found also to be perfectly simultaneous. The apex of the heart was observed and felt to strike against the ribs at each systole, and thus was explained the impulse. The motions of the auricles, when regular, preceded the ventricular motions and sounds; they were slight and undulatory, increasing from the sinus to the appendix, where they terminated suddenly, and were immediately followed by the ventricular systole. They afterwards became irregular, sometimes failing and sometimes occurring twice slightly during the period of ventricular repose, and in one experiment entirely ceased some minutes before the movements and sounds of the ventricles. In no instance were they attended with any perceptible sound. This account is confirmed by the experiments of Mr. Carlile, which satisfactorily explain the succession of the motions of the auricles and ventricles; but they were performed on animals too small to illustrate the sounds. He very justly shews that the pulse cannot be simultaneous in all the arteries at once, but must be successive, transmitted in a wave from the heart to the end of these elastic tubes.

Although it seems fairly established that the first, or dull sound, is produced by the systole of the ventricles; and the second, or quick one, by their diastole, it is by no means clearly explained in what way these actions generate these

* Journ. Hebdom. No. 97; also Mr. Bryan, Lancet, Sept. 1833, and M. Bouillaud, Journ. Hebdomad. 1834.

† Dublin Journal of Medical Science, vol. iv. The essay was likewise read at the Cambridge Meeting of the British Association.

by the ventricular systole, which is accompanied by the first or dull sound. This systole, by straightening the anterior

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sounds. The following causes have been severally assigned as physically capable of generating the first sounds during the systole of the ventricles. 1. The collision of the particles of fluid in the ventricles. (Dr. Hope, Dr. Spittal.) 2. The rush of blood into the great arteries. (Mr. Carlile.)—3. The closing of the mitral and tricuspid valves. (M. Rouanet, Mr. Bryan.)—4. The muscular contraction itself.

1. The first of these explanations is ingeniously proposed by Dr. Hope, but he advances no facts in direct proof of the hypothesis. In a number of experiments which I have made on the generation of sound, I have found liquids, of all bodies, the most difficult to excite to sonorous vibration; and although they readily transmit vibrations already produced in solids, it requires a combination of circumstances to make them originate sound. This is consistent with the explanation which I have given of the production of sound, (p. 5, *et seq.*) for impulses which throw solids into sonorous vibrations, are expended in liquids in causing a displacement of their particles. On making an experiment with a gum elastic bottle, by filling it with water, and then forcibly compressing it under water by the end of the stethoscope, (avoiding the use of the hand, for that produces its own muscular sound) I have failed in procuring any sound at all approaching to that of the heart's contraction. The blood yields readily to the contracting ventricle, and there being no obstacle to the escape of blood from it, further than the weight of the arterial column, which the normal action of the heart can quietly and steadily overcome, it passes into the arteries without vibration. But if there be an obstacle to the current of the blood from the ventricle, whether that obstacle be a narrowing or a projection in the orifice, the current will act on it just as the bow does on the string of a violin; a sound will be excited, and thus are produced valvular murmurs. Again, if instead of the orifices being narrowed, the heart contracts with unnatural briskness, expelling its contents with convulsive energy, the natural outlets then become relatively narrow, and are thrown into vibrations; this is the *rationale* of the bellows murmur which accompanies the jerking pulse of pericarditis and the irritation of inanition. But the difference of these sounds, and of the circumstances that excite them, from those of the normal action of the heart, makes me hesitate to refer the latter to the same principle; and the fact that the morbid are often superadded to the natural sounds, also inclines me to think that they have a distinct cause.

2. The second explanation of the first sound, the rush of blood into the larger arteries, is perhaps less liable to the acoustic objection before urged, than the preceding opinion, for the blood has acquired an impulse when it enters the arteries, and if its course there is not free, it might readily produce a sound. But in their natural state, the arteries give passage to the blood as smoothly as the heart parts with it, and it would prove an imperfection in nature were it otherwise. Moreover, if this explanation were true, the large arteries rather than the heart would be the principal seat of the sound; and the sound should be increased by an hypertrophied heart, with a strong pulse, and diminished by a dilated heart and a weak pulse; yet the reverse of these is presented in nature.

3. The closing of the auricular valves. The principal objection to this as the

convexity of the ventricles, brings their apex into forcible contact with the ribs, and thus is produced the impulse. This

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only cause of the first sound, is, that it must be instantaneous, and confined to the first part of the ventricular systole, whereas we know that the first sound is prolonged during the whole period of this action.

4. Although Laennec referred the first sound to the systole of the ventricles, he did not attempt to define the physical cause of its production. In the former edition of this work, I ventured to class it among the muscular sounds which Dr. Wollaston* first noticed to occur in all cases of rapid muscular contraction. This sound may be exemplified by applying the fleshy part of the thumb to the stethoscope or naked ear, and bending and straightening the thumb. It is louder in muscles that are thin, and in a state of considerable tension; and it is remarkable that it does not cease with the apparent movement, but continues as long as the muscle remains contracted and tense; it then takes on an intermitting character, like the noise of the rolling of a carriage over rough pavement, whence Dr. Wollaston was led to infer that muscular action is not perfectly continued, but consists of a series of minute contractions and relaxations. A good example of it may be obtained on applying the stethoscope to the neck of a person who holds his head back towards the opposite side, and then throws the platysma myoides into contraction. It still appears to me, that the most simple and satisfactory way of accounting for the first or systolic sound of the heart, is to refer it to this class of sounds. Their physical production seems to depend on the sudden tightness or tension into which the fibres of muscles are thrown when they contract; and the self-acting power of these fibres constitutes them the motors as well as the subjects of sonorous vibrations. Here we have to remark the extreme facility with which the motions of solids produce sounds, compared with those of fluids; for it is almost impossible to touch, stretch, bend, or compress solids, without throwing them into sonorous vibrations. In this case we have a series of cords or fibres brought by a zig-zag tension, into a state of rigidity capable of sonorous vibrations, and the impulses developing these vibrations are communicated by the oscillations of those contracting fibres themselves; these oscillations in a greatly magnified degree, are apparent in the quivering seen and felt in the muscles of a horse drawing with unusual effort. The varieties observed in the contraction of the heart seem to me to be perfectly explicable on this principle. The sound begins the moment the fibres arrive at a state of tightening or tension; it continues until the contraction is completed and the blood expelled from the ventricle, and ceases the instant of the diastole. To perceive more readily the effect of hypertrophy, and of distillation, let us attend to the sounds produced by the tension of linen or canvass, (for muscle is, mechanically speaking, equally a web of fibres,) and we shall find that in proportion as we thicken the substance, we obscure the sound which is produced on briskly stretching it; but when we use thin and simple webs, the sound becomes proportionally loud and clear. I shall not pursue the illustration of this explanation further, for I introduce it here only interrogatively, as deserving a place among other views, on the claims of which, future observation and experiment must decide. I must only remark,

* Croonian Lecture, Phil. Trans. 1810.

systole, by throwing an additional quantity of blood into the arteries, causes the arterial pulse, which in arteries near the

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that M. Pigeaux is in error when he maintains that muscular sounds cannot be produced under water: I find them more distinct and free from adventitious sounds of the surface, and I have been able to intimate the sounds of the heart very exactly by muscular movements of the hand under water. I will conclude with the question, if the first sound of the heart is produced by another cause, what becomes of the muscular sound in this case of rapid muscular contraction?

We now come to the subject of the second sound, which, although certainly occurring at the moment of the diastole of the ventricles, has received several different explanations as to its physical cause. The only two which appear tenable in the present state of our knowledge are—1. The reaction of the arterial columns of blood against the semilunar valves. 2. The impulse of the blood from the auricles refilling the ventricle at its diastole.

1. The first of these bears a very inviting aspect, for the 2nd sound is just of that abrupt flapping character that might be supposed to result from the action of a thin valve. According to this view, the arteries, more than the heart, should be the seat of this sound. The tense column which throws these valves into play, should receive their shock more forcibly than the heart, which at that moment has become flaccid, and ill adapted to transmit sound or impulse (backstroke) through the whole of its substance. There are some cases of disease which seem to militate against this view, but as the history of these cases was not studied with especial reference to this point, their accuracy is not, perhaps, clear enough to warrant our arguing upon them, I therefore still do not consider this view as disproved, and it should claim attention in future investigations.

2. This is Dr. Hope's explanation of the second sound: when the diastole takes place, the blood, impelled by a number of concurrent circumstances, shoots with instantaneous velocity from the auricles into the ventricles; and the reaction of the ventricular walls on its particles, when their course is abruptly arrested by the completion of the diastole, is, he conceives, the cause of the loud, brief, and clear sound. The concurrent circumstances which impel the blood into the ventricles at the moment of their diastole, are the distention of the auricles in which the blood has been accumulating during the ventricular contraction; the weight of the ventricles collapsing on the auricles thus distended; the width of the auriculo-ventricular orifices; and lastly, the sucking power of the ventricle in its diastole. With respect to this last, Dr. Hope does not assume that the ventricles have an actively dilating power further than what proceeds from the physical elasticity of their parietes, but such a power has been ascribed to them by Bichat, Pechlin, Carson, and others, and even by Laennec; and although opposed to what we at present know of animal dynamics, it would be rash to absolutely deny the possibility of its existence. I would suggest that the injection of the coronary arteries, which occurs the instant the systolic action ceases, may somewhat contribute to the dilatation of the ventricles. Whatever be the cause, the diastole in large animals is sufficient to force open the hand of a person grasping the ventricles, and it is therefore not surprising that this should have been ascribed to an actively dilating power. It is in favour of Dr. Hope's explanation of the

heart is synchronous with the ventricular systole, but in those more distant succeeds it at an interval occupied by the transmission of the wave through the blood along the elastic tubes from the heart.

3rd. That the ventricular systole is immediately followed by the diastole, which is accompanied by the 2nd or short sound.

4th. That there is then an interval of rest, at the conclusion of which the auricles contract and the series of motions is repeated as before.

But it was still uncertain in what way the ventricular systole produced the first sound, or how the diastole developed the second; and various opinions were broached by different writers on these subjects. Thus the first sound was ascribed by Mr. Carlile to the rush of blood into the great arteries; by M. Rouanet and others to the closing of the auriculo-ventricular valves; by Dr. Hope to the collision of the particles of fluid in the ventricles; and, by myself, to the muscular contraction itself.

The 2nd or short sound, accompanying the diastole of the ventricles, was ascribed—by Dr. Hope, to the impulse of the blood from the auricles refilling the ventricles;—by M. Rouanet, Mr. Carlile, M. Bouillaud and others, to the reaction of the arterial columns of blood against the semilunar valves.

I concluded the appendix before quoted, by recommending Dr. Hope to investigate experimentally these points, which were there shown to be doubtful; but his engagements, and other circumstances, having prevented him from doing so, I

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second sound, that it does not falsify Laennec's signs of disease of the auricular valves; and although for acoustic reasons before stated, I would have placed the seat of the sound in the parietes of the ventricles, rendered momentarily tense by the sudden influx of blood, rather than in the motions of the fluid, I have until lately inclined to this explanation of the cause of the second sound. It needed, however, as Dr. Copland observes, further confirmation; and I would add, that the whole subject of the sounds of the heart required further research.

lately undertook the task. I made a point of ensuring Dr. Hope's presence and testimony at the experiments, and I conferred with him and several other gentlemen as to the best mode of performing them.*

Through the kind assistance of Sir Benjamin Brodie, in providing me with some of the Woorara poison, the results were more satisfactory, whilst the animals were put to less suffering, than in any prior experiments. This poison, better than any other, suspends the action of the nervous system without affecting the irritability of the part, and it proves fatal by destroying the animal sensibility on which the act of respiration depends. If the respiration be then maintained artificially, the heart will continue to beat, and the circulation will proceed for a considerable period; until, in fact, the various organs become cooled and otherwise injured, for want of the continuance of other processes, which are in some way maintained by the nervous influence. Thus in the following experiments the heart continued to act vigorously for more than an hour after the extinction of animal life; and might have done so longer, had it been necessary; but all the results were clearly ascertained and confirmed by those present within that period.

Experiment I.

About twenty grains of Woorara, moistened with water, having been inserted in an incision in the haunch of a donkey two months old, the animal died in fifteen minutes. Artificial respiration was immediately established, and the chest being opened, the pericardium was slit down and the heart exposed. Its pulsations were regular and vigorous, the auricles con-

* I was present at an experiment attempted by Dr. Hope, in November last, at Mr. Field's; and he then planned modes of suspending the action of the valves, similar to some of those afterwards adopted in my experiments. The animal was killed by a blow on the head, and this so much impaired the heart's action, that no results were obtained.

tracting immediately before the ventricles. The double sound was distinctly isochronous with the systole and diastole of the ventricles. The following points were then observed and noted, after repeated examinations by several present.*

Observation 1. The 1st sound was equally audible on all parts of the ventricles.

Obs. 2. The 2nd sound was most distinct near the roots of the great arteries, being audible there in the weaker pulsations, when it could not be heard by the stethoscope applied to other parts of the ventricles.

Obs. 3. Pressure on the arterial roots by the fingers, or by the stethoscope, invariably stopped the 2nd sound. Slight pressure caused a whizzing, or bellows murmur, with the 1st sound.

Obs. 4. On pushing the auricles, by the end of a finger, into each auriculo-ventricular opening, the ventricular contractions became weak and irregular; but the 1st sound, although weak, was still heard alone.

Obs. 5. At each systole, the sudden tension or tightening of the ventricles was felt, by the finger applied to their body, as an abrupt shock, with which the 1st sound exactly coincided.†

Obs. 6. The left auricle was cut open, and the mitral valve partially destroyed; the blood issued in jets at each ventricu-

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* The following gentlemen were present; and I here beg to offer to them, and to those who assisted at the other experiments, my thanks for their kind co-operation and testimony.—Dr. Arnott, Author of the Elements of Physics, &c.; Mr. Babington, Surgeon to St. George's Hospital, &c.; Mr Good, Surgeon to St. George's and St. James's Dispensary; Dr. Hope, Assistant Physician to St. George's Hospital, &c.; Mr. Henry Johnson, Demonstrator of Anatomy, &c.; Dr. T. Peregrine, House Surgeon at St. George's Hospital; Mr. G. Smith, Lecturer on Anatomy, &c.; Mr. Tatum, Lecturer on Anatomy, &c.

To Mr. Tatum, Mr. Henry Johnson, and Mr. H. James Johnson, I am especially obliged, not only for their able assistance, but also for the use of their splendid new dissecting room, and its commodious appurtenances, in Kinnerton-street, where these experiments were performed.

† This Observation was, I think, suggested by Dr. Hope.

lar systole; yet the 1st sound still accompanied the systole. The 2nd sound was not heard after this incision.*

Obs. 7. The right auricle was also freely laid open; still the 1st sound continued.

Obs. 8. I pushed my finger through the mitral orifice into the left ventricle, and pressed on the right, so as to prevent the influx of blood into either ventricle: the ventricles continued to contract strongly, (especially when irritated by the nail of the finger in the left) and the 1st sound was still distinct, but not so clear as when the ventricles contracted on their blood.

Obs. 9. The same phenomena were observed when both the arteries were severed from the heart.

Until the auricles were cut open, (as mentioned in 6 and 7) the 2nd sound was audible in all the strong pulses of the heart, but it was not heard after, although upwards of 30 pulses, most of them vigorous, took place. Ten or twelve strong contractions occurred after the introduction of the finger, as mentioned in Obs. 8.

This experiment lasted an hour and twenty minutes from the commencement of artificial respiration.

Experiment II.

About fifteen grains of Woorara (powdered, and made into a paste with water) were introduced into a wound under the haunch of a young ass, about six weeks old. The animal died in about thirty-five minutes. Artificial respiration was then immediately established, and the chest opened by cutting through the cartilages of the ribs, to the left of the sternum, and along the upper margin of one of the ribs near the shoulder, and then breaking back three or four ribs, so as to expose

* The results after 6 were witnessed by Dr. Hope, Mr. H. Johnson, and myself, the other gentlemen having left. The observations were noted during or immediately after the experiments; generally by Dr. Hope or myself.

the contents of the left side of the chest. The following points were noted, as observed by several present.*

Obs. 1. Before the pericardium was opened, the 1st and 2nd sounds were very distinctly heard, although the heart touched no part of the parietes of the chest.

Obs. 2. Both sounds were distinctly heard through a lobe of the lung interposed between the heart and the stethoscope.

Obs. 3. The pericardium being completely slit open, the 2nd sound was observed to be, decidedly, most distinct at the origin of the pulmonary artery and aorta, where it was louder than the 1st sound, and had perfectly its natural short, clear, flapping character. With the stethoscope applied on the body of the ventricles, the 2nd sound was heard less distinctly, and seemed more obtuse and distant.

Obs. 4. When the stethoscope was applied to the aorta about three inches from its origin, the 2nd sound (without the 1st) was heard following the systole of the ventricles as felt by the observer's finger.†

Obs. 5. The aorta and pulmonary artery being for a few seconds compressed between the finger and thumb, the 1st was accompanied with a bellows murmur, and the second sound ceased, during the continuance of the compression. This experiment was repeated several times by Dr. Hope and myself.

Obs. 6. A common dissecting hook was passed into the pulmonary artery, and was made to draw back and thus prevent the closure of the semilunar valves: the 2nd sound was evidently weakened, and a hissing murmur accompanied it.

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* Mr. Bushel, Lecturer on Anatomy, &c.; Mr. Good, Surgeon to St. George's Dispensary, &c.; Dr. Hope; Mr. H. Johnson, Surgeon, &c.; Mr. Keate, Surgeon to their Majesties, &c.; Dr. Macleod, Physician to St. George's Hospital, &c; Dr. Page; Mr. Partridge, Junior Professor of Anatomy at King's College, &c.; Mr. Malton and Mr. Seagram, Pupils at St. George's Hospital; Mr. Willesford, Surgeon, &c.

† This Observation was suggested by Mr. Keate, and Obs. 2 by Dr. Hope.

A shoemaker's curved awl was then passed into the aorta, so as to act in the same way on the aortic valves: *the 2nd sound now entirely ceased, and was replaced by a hissing.*

Obs. 7. The hook and the awl were withdrawn; *the 2nd sound returned and the hissing ceased.* This and the preceding experiment were repeated, and observed by Dr. Hope, Mr. H. Johnson, Mr. Malton, and myself.

Obs. 8. Experiment 6 was repeated, with the same result, and whilst Dr. Hope listened, I withdrew the awl from the aorta. He immediately said, "Now I hear the second sound." I then removed the hook from the pulmonary artery; Dr. H. said, "Now the second sound is stronger, and the murmur has ceased."

Obs. 9. The pulmonary artery was cut open, and the finger introduced into the right ventricle;—the heart continued to contract irregularly, and the 1st sound alone was obscurely audible.

Obs. 10. Slight contractions took place after the ventricles were laid open, and the columnæ carneæ were seen to contract simultaneously with the fibres of the ventricles.

These observations lasted during an hour and ten minutes after the commencement of artificial respiration; and until the opening of the artery in Obs. 9, the contractions of the heart were generally regular and vigorous.*

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* Two other asses were subjected to experiment, but from accidental circumstances, no conclusive results were obtained from them. One was in so weakly a state before the poison was applied, that when the chest was opened, the heart pulsated very feebly, and without any sound. A curious circumstance was noted in this case: when the ventricles had almost ceased to move, the auricles went on contracting regularly and fully, but not simultaneously, for the movement of the right preceded that of the left by about the same interval as the auricular movements generally precede the contraction of the ventricles; and it appeared that these movements of the auricles were sufficient to maintain to a certain degree the circulation, for arteries, when wounded, yielded florid blood, but not in jets.

In the other experiment, the substance of the ventricles was accidentally wounded in opening the chest; this occasioned much hæmorrhage from the coronary artery, and weakened the heart's action so much, that the second sound was

The deductions from these results are simple and obvious; and they not only decide between the views before stated, but they appear to me to demonstrate most satisfactorily the true seats and causes of the sounds of the heart.

That the first sound is not caused by the rush of blood into the great arteries, (as supposed by Mr. Carlile) is proved by Obs. 4, 6, 7, 8 and 9, of Exper. I.; and Obs. 9, of Exper. II.; in which the first sound continued, although little, and and in the latter cases no blood, could have been thrown into the arteries. A further proof against this view may be seen in Exper. II. Obs. 4, from which it appears that the first sound is much less audible in the large arteries than in the heart.

That the 1st sound is not dependent on the closing of the auriculo-ventricular valves, (as imagined by M. Rouanet and others) is evident from Exper. I. Obs. 4, 6, 7, 8, 9, in which the closure of these valves was partially or completely prevented, yet the 1st sound still continued.

That the first sound is not produced by the collision of the particles of fluid in the ventricles, (as formerly conceived by Dr. Hope) appears from Obs. 4, 8 and 9, of Exper. I.; and Obs. 9, of Exper. II.; in which the sound was produced, although there was no blood in the ventricles.

That the 1st sound is produced by the muscular contraction itself, may be considered as proved by Obs. 8 and 9 of Exp. I.; in which every other possible source of sound was excluded, and the 1st sound still accompanied the systolic action of the ventricles.*

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not heard. This result was generally remarked, that whenever the ventricular systole was weak, and the arterial tension was not kept up by it, the second sound ceased. These unsuccessful trials were useful to us, in familiarising us with the anatomy, and in preparing for the others.

* This view of the cause of the 1st sound, was first published by me in the first edition of this work, in 1828, and I am not aware that it has been entertained by any other writer. M. Majendie is in error when he ascribes a similar opinion to

That the 2nd sound is produced by the reaction of the arterial columns of blood tightening the semilunar valves at the ventricular diastole, is clearly proved, not only by the situation of these valves being the especial seat of the sound, (Exp. I. Obs. 2; and Exp. II. Obs. 3, 4) but also by the numerous observations in which the cessation or reproduction of the sound was effected by the suspension or restoration of the action of these valves. (Exp. I. Obs. 3; Exp. II. Obs. 5, 6, 7, 8.)

It being thus proved that the 1st sound is essentially produced by the tightening of the muscular parietes of the ventricles, and the 2nd sound by the subsequent sudden tension of the semilunar valves, it is easy to perceive how various circumstances may increase or diminish the sounds, as they augment or impair the degree or abruptness of this tightening or tension in these parts. Thus the mass of blood in the heart increases the clearness of the 1st sound, by affording an object, around which the fibres effectually tighten; whilst the auricular valves, by preventing the reflux of this blood, increase its resistance, and thus add to the tension necessary for its expulsion. Probably, in common pulsations, the ventricles do not attain the degree of tension which is sonorous until the closing of the auricular valves; this closure, as the commencement of the resistance, brings at once to its acmé the muscular tension, which continues until the contents of the ventricles are sufficiently expelled. This accounts for the sudden or flapping commencement often perceptible in the 1st sound, and it suggests how the due action of the auricular valves generally contributes to its clearness. The degree and abruptness of the systolic tension was well seen in Obs. 5,

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Laennec, who, on the contrary, only associated the abnormal murmurs with the muscular sound produced by a fancied spasm of the heart and arteries; but neither in his works, nor in his lectures, did he give any opinion as to the physical cause of the healthy sounds of the heart.

of Exp. I.; and the external feel and view of the systole gave us the impression of its being a motion in itself sufficient to produce sound.* The auricular valves, the cordæ tendineæ, the columnæ carneæ, and internal fibres of the ventricles, if they attain the same degree of tension as the exterior of the ventricles, may have an equal share in the production of the 1st sound; but I am disposed to think, that what we hear proceeds chiefly from the contracting tenseness of the external walls of the heart, both because they are nearer to the ear, and because in Exp. I, Obs. 8, the contraction of the left ventricle upon my finger within it, was by no means so abrupt or strong as that of the exterior, as felt by the other hand, and still heard through the stethoscope.

The termination of the systole of the ventricles is abrupt, being immediately followed by the diastole: and it is obvious that the first effect of this must be, to occasion the sudden closing of the semi-lunar valves, pressed now only on their concave side by the force of the arterial column of blood. Hence the 2nd sound immediately succeeds the 1st, or rather appears to terminate it, by its abrupt, clear *flap*, which in the healthy heart differs as much in character from the 1st sound, as the simple valves by which it is produced differ from the thicker muscular web of the ventricles, the tightening of which causes the 1st sound. The mobile state of these valves, the bulk of blood propelled by the ventricular systole into the arterial column, and the suddenness of the diastole by which this column is brought to press fully backwards on the valves, are the circumstances which give clearness and loudness to the 2nd sound; and it may be seen, in the foregoing experimental observations, how various causes interfering with these, im-

* The results of these experiments so completely confirm the views that I stated in the last edition of this work with respect to muscular action as a cause of sound, that I need here only refer to my former remarks as quoted in the note, p. 167.

paired or suspended the 2nd sound. Exp. I, Obs. 3, 4, 6, &c., Exp. II, Obs. 5, 6, 7, 8, 9.

The whole of these observations abound in facts which concur with many former experiments in entirely overthrowing the extraordinary opinions of M. Majendie on the sounds of the heart, which I have noticed in a former page. For the satisfaction of some of those present, whose doubts had been raised by the positive tone of the assertions of so distinguished a physiologist, and by the plausibility of some of his explanations, Obs. 1 and 2 of Experiment II were made, and all the gentlemen present agreed that the same sounds that had been heard before the chest was opened, were most distinctly produced after that operation, when the heart struck against nothing in its motions. After this, it is quite unnecessary to go through the details of the ingenious superstructure which M. Majendie has erected on a foundation thus proved to be erroneous. It is surprising that such an eminent observer should permit himself to be so blinded by a favourite notion, as to strain conclusions in its favour, and tax his ingenuity for its support, in total disregard of simple and decisive facts. On reading attentively the various arguments and cases which M. Majendie brings forward, (as reported in the Lancet, Feb. 1835, from his Lectures) I find there scarcely any phenomena clearly described which will not admit of a more simple and satisfactory explanation from the views which have been established by the preceding experiments. Certain of his assertions, such as that water or air in the chest, entirely suspend the sounds of the heart, will be viewed by experienced auscultators rather as proofs of an imperfection in the observer's hearing, than as arguments in support of his opinions.

Although it is proved by the preceding observations that the impulse of the heart against the chest does not produce its natural sounds, yet I am disposed to admit, that in violent action of the organ, its more sudden and abrupt strokes

against the chest do cause a sound, which constitutes the loud termination of the 1st sound in these cases, and which seems nearer the ear, and more like a knock, than what is heard in the ordinary action of the heart. In common pulsations, the apex of the organ is drawn upwards and forwards at each systole, and sliding obliquely on the smooth pericardium, does not impel against the ribs with sufficient abruptness to cause sound. But in quicker and more violent pulses, the abruptness of the motion, and the force of the blow against the side of the chest, are such, as can scarcely fail to produce sound.

Another circumstance which seems to have misled M. Majendie, is the fact that the sounds of the heart are louder when the chest is entire, than when the organ is exposed. This, instead of proving that the sounds are necessarily produced on the thoracic walls, merely exemplifies the general fact, that the sounds of a sonorous solid are increased by being confined in a close cavity with elastic walls, which augment the sound after the manner of a sounding board.

I trust that M. Majendie will see, in the preceding comments, no other spirit than the same love of truth, and the same distrust of hypothetical "fables" and of false inferences, which he upholds as his guides; and as his opportunities and powers of observation may enable him to do much for the better knowledge of the pathological branch of this subject, I hope that he will soon abandon the wrong scent on which he has hitherto pursued the investigation.

CHAPTER III.

AUSCULTATION OF THE HEART IN DISEASE.

Disease in the heart may be indicated by several modifications of the physical phenomena described in the preceding chapters, and these modifications may respect the position, extent, and character of the pulsations, and the nature and order of the sounds that accompany them. The circumstances producing these changes are generally more varied in nature than those which modify the physical signs of the organs of respiration; and this fact, together with the more complicated structure of the heart, renders its auscultatory signs more ambiguous and more difficult to interpret. The labours of Corvisart, Laennec, Bertin, Andral, Louis, Latham, Elliotson, Hope, and others, have furnished us with much valuable information, which makes the diagnosis of diseases of the heart much easier and more certain than it formerly was; but there are still several affections which often present no distinctive signs, and there are some signs the physical causes of which are not entirely known. In complicated cases of organic disease, also, the action and reaction of the different parts of the mechanism of the circulation on each other, often present phenomena of a very complex character, which require much careful examination and study to analyse and to trace them to their true causes. A minute consideration of all the symptoms, both physical and general, can alone guide us to a correct diagnosis in such cases; but as such a mode of treating the subject would require far more space than can be conveniently assigned to it in this treatise, I must refer for details to other recent works on Diseases of the Heart, especially to that of

Dr. Hope, and to his contributions to the Cyclopædia of Practical Medicine. I shall here only briefly state the general principles of the semeiology of diseased states of the heart, hoping that with the present advantage of a more precise knowledge of the physiology of the organ, a few more years' study will make these principles applicable to all cases, and thus render the diagnostic pathology of the heart simple and satisfactory.

Dilatation of the Heart.

Any circumstances which render the heart incapable of sufficiently expelling its contents, and thus occasion a distension of its parietes, if sufficiently prolonged, eventually cause their permanent dilatation. Thus an obstruction to the passage of the blood through the arteries, will cause a preternatural fulness of the ventricle from which they proceed, and where the irritability of the heart is not such as to excite the muscular fibres to greatly increased action to expel the load, the ventricle will be simply dilated, with a corresponding thinning of its parietes. Again, the cause may sometimes be in the fibres of the heart itself, when, from weakness or deficient irritability, they do not contract sufficiently to expel the due quantity of blood, which accordingly acts as a constantly distending force, and causes an increase of the cavity, with an attenuation of its walls; and where the affection is considerable, and extends to both ventricles, the heart acquires a rounded shape.

Now in this state, as the degree of contraction is diminished, and the apex is therefore less forcibly drawn against the ribs, the impulse felt in the præcordia will be slighter and brisker, as well as lower, than usual. The sound of the ventricular contraction, on the other hand, although shorter in duration, will be louder and clearer than usual, inasmuch as the tight-

ening web is thinner, and more simple;* and it will be heard over a larger extent, inasmuch as the surface of the heart is increased by the dilatation. The physical signs of simple dilatation of the ventricles are therefore, (as described by Laennec) a diminished impulse, and a clear and short first sound, which is heard over an extent greater than would be expected from the weakness of the impulse. In considerable dilatation, the first sound resembles in shortness and flapping character the second; and as Dr. Hope remarks, it is sometimes to be distinguished from the latter only by its synchronism with the pulse of the carotid.† The pulse, in dilatation of the ventricles is necessarily weak; and if the disease is considerable, there may be dropsy or other cachectic states resulting from inefficiency in the circulatory apparatus. This is more especially the case with dilatation of the left ventricle, the physical signs of which will be most apparent to the left of the sternum, between the fifth and seventh ribs. Dilatation of the right ventricle not unfrequently accompanies chronic diseases of the lungs, which impede the passage of the blood from it. Its signs are chiefly perceptible under the sternum. It is commonly accompanied by a pulsating swelling of the jugulars.

Hypertrophy of the Heart.

This affection may well be placed in opposition to the preceding, for although it also often arises from an obstruction to the circulation, its character and signs are the converse of those of dilatation. Any circumstances which for a considerable time excite the heart to a constantly increased action, determine an augmentation of its substance, and a thickening

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* See note, p. 167. Hence attenuation of the walls of a ventricle without dilatation, will increase the clearness of the sound, but not its extent.

† When the 1st sound is as clear as the 2nd, and if it is at the same time perceptible on the right side of the back, the dilatation is extreme.—*Laennec.*

of its muscular parietes.* Now the same obstructions to the circulation which cause dilatation of the heart, such as diseases of the valves, &c., give rise to hypertrophy in those whose heart is more irritable and active; for the obstacle to the passage of the blood excites it, in these subjects, to increased action. So also, as diminished irritability alone may lead to dilatation of the heart, preter-natural irritability alone may occasion its hypertrophy, by making it contract with undue energy in its natural task. This is exemplified in the hypertrophy resulting from pericarditis, which is sometimes attended with a diminution of the cavities of the ventricle, but still causes the symptoms of hypertrophy. Excessive and long-continued exertions of the whole body, especially during its growth, seem to be capable of giving rise to hypertrophy. It is not clear whether this is the immediate effect of the increased labour given to the heart, or whether it may not be the result of an increased irritability, caused by the over-straining of the organ in its extraordinary task.

The increased thickness of muscular substance gives an hypertrophied heart more strength of contraction, but the greater complexity of its fibres prevents that simplicity and unity of motion, with which a thinner muscular lamina contracts. Hence the impulse is stronger, and of greater extent, but less abrupt, and of longer duration. In fact, in extensive hypertrophy, it consists, not only in the striking of the apex

* The manner in which the heart becomes hypertrophied, in consequence of increased action, depends, I conceive, in part on the immediate relations of the function of the organ with that supply of blood which nourishes it. Violent action of the heart is far from increasing the substance of other parts of the body; and, in truth, this action does not proportionally augment the distribution of the blood through them, but is principally expended in the arteries near the heart. The coronary arteries especially receive this increased impulsion of blood, which therefore passes most rapidly through them; and this free supply of the pabulum of irritability and energy maintains and feeds the heart in its inordinate exertions, while the distant parts suffer from a relatively small supply of blood, and from consequent weakness.

at the acmé of each systole, but also in the pushing of the body of the heart against the ribs, as it swells in each contraction. This description will lead us to perceive also, that the first sound must be more prolonged and duller than natural. The weakness of the sound, as compared with the strength of the impulse, is most remarkable; but the reason of this weakness is sufficiently apparent, not only in the more prolonged character of the contraction, but also in the effect which large bundles of tightening fibres must have in interfering with, and neutralising each other's vibrations. The illustration which is given of this matter, in the sudden tightening of a piece of cloth or canvass of different degrees of thickness, has been already referred to ;* and it very well exemplifies the point before us. Thus, when fine cotton or linen-cloth is abruptly tightened, it gives a clear short note; but when a piece of thick blanket or sackcloth is substituted, the sound emitted is muffled and dull. The 2nd sound, although ill-conducted through the increased substance of the ventricles, will still be heard distinctly under the sternum and below the clavicles, where, as Laennec observes, it is often unusually clear. Laennec considered it possible to distinguish which ventricle is hypertrophied, by the position in which the impulse is most felt, whether at the sternum or to the left of it. This criterion, together with the greater clearness of the sound on the other side, where the impulse is weak, holds good in most ordinary cases; but if the hypertrophy be excessive, and extend to the apex, and especially if it be accompanied by a thinning of the walls of the opposite ventricle, the apex will be pushed towards the weak side, and will produce the impulse there more than on the side of the hypertrophy. This circumstance, which was

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* See note, p. 167.

noticed by Laennec, is thought by Dr. Spittal* to detract much from the above-named criterion; and it may lead us to be guided, rather by other signs, and by the position of the weaker sound, than by that of the impulse. The impulse in great hypertrophy produces a *heaving* of the ribs, which sensibly raises the head of the auscultator, and then suddenly sinks back. This instantaneous collapse following the heaving impulse, Dr. Hope calls the back stroke, and he considers it very characteristic of extensive hypertrophy of the ventricles, especially if conjoined with dilatation. Hypertrophy of the left ventricle causes a hard, and generally full pulse, throbbing and fulness in the head, increased by exertion, and other effects of a too forcible determination of blood. Hence it may produce vertigo, paralysis, apoplexy, and other results of this pathological condition. Hypertrophy of the right ventricle is more rare, and is commonly caused by the flow of blood, from it being impeded by diseases in the lungs, or valvular obstructions on the left side of the heart. It is also a common accompaniment of those malformations in which the ventricles communicate, and the weight of the aortic circulation is partly thrown on the right ventricle. In its turn it may produce in the lungs the various effects of too full a supply of blood, such as pulmonary apoplexy, congestion, pituitous catarrh, asthma, hydrothorax, &c.

Hypertrophy and Dilatation combined, occur much more frequently than the simple affections. This combination of disease results from the joint operation of the causes described as occasioning dilatation and hypertrophy. Thus an obstruction to the circulation, such as a disease of some of the valves

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* Edin. Med. and Surg. Journ. 1834. Tumours behind the heart, which force it against the parietes of the chest, may render its impulse like that of hypertrophy. I have known an aneurism of the descending aorta produce this effect with a heart the parietes of which were natural.

of the heart, causes an accumulation of blood in one or both of its cavities, they are excited to increased efforts to expel the load, and thus in time become hypertrophied; but being still unable sufficiently to effect its expulsion, the mass of blood acts as a distending force, and produces a dilatation of the cavity. The greatest enlargement of which the heart is capable, is produced by this conjoint affection in both ventricles, and sometimes amounts to more than three times the ordinary size.

The physical signs of the double affection are in great measure a combination of those of hypertrophy with those of dilatation; but the degree of prevalence of either of these affections will greatly modify the result. Thus, in hypertrophy with slight dilatation, there is a strong heaving impulse, with an abrupt collapse, or back stroke, and a prolonged, diffused, but not clear sound. In dilatation with slight hypertrophy, the sound is loud, commencing abruptly, and heard over a large space, whilst the impulse is unnaturally great only when the heart is excited, as in palpitation, when it produces hard abrupt circumscribed blows, without heaving. The explanation of these signs will be sufficiently clear from a consideration of the illustrations which have been given of the simple affections. The heart, by being thus thickened and dilated, may attain a size sufficient to produce a dulness more extensive than usual, on percussion on the sternum, and below the fifth left rib, which scarcely ever takes place in simple dilatation or hypertrophy; and M. Bertin remarks, that in extreme cases the heart, arrested in its protrusion downwards by the diaphragm, may assume a more horizontal position, with its apex more to the left, and displace the lung upwards to above the fourth rib.* Dr. Hope adds, that adhesions to the pericardium may retain a heart thus enlarged higher than it

* Traité des Maladies du Cœur.

would be placed by its weight; and that the organ thus impacted between the spine and the front walls of the chest, sometimes causes an unnatural prominence of the præcordial region.* The effects of hypertrophy and dilatation of the heart will vary according to the prevalence of either affection. Palpitation and other disorder of the circulation, will accompany extensive degrees of the disease; and they will be violent and heaving when the hypertrophy predominates, but noisy, fluttering, and accompanied by a feeling of faintness, when dilatation is the chief disease. It is usually attended with more dyspnœa than the simple affections; and it is generally more distressing in its effects, and tends more rapidly to a fatal termination.

Pericarditis.

Inflammation attacking the pericardial coverings of the heart, is often a very formidable disease, and sometimes very obscure in its character. It may manifest itself by pain or heat in the region of the heart, increased by pressure in the intercostal spaces; dyspnœa, or a feeling of oppression; a frequent, hard, jerking pulse, afterwards becoming small, irregular, and ultimately intermittent; and as the disease proceeds towards a fatal termination, great anxiety of countenance, fits of palpitation and fainting, anasarca, &c. occasionally present themselves. But, as Laennec observes, each or all of these symptoms may be absent, and it has occurred to the most distinguished physicians to discover the existence of pericarditis (especially where it has been masked by other inflammations) only by examination after death. The effect of the inflammation is to cause the effusion of serum and coagulable lymph; and the extent of this effusion is probably, as

* Cycl. of Pract. Med., Art. Hypertrophy of the Heart.

Dr. Hope suggests, the principal cause of the variety of the symptoms; the sense of oppression, the irregularity of the pulse, and the syncope, being occasioned by the liquid effusion, by pressure, impeding the heart's action.

The jerking impulse and arterial pulse are by no means peculiar to pericarditis, as they proceed only from increased irritability, and occur frequently in various kinds of nervous irritation; but they are useful signs to draw the attention of the practitioner to the heart during rheumatism, in which this organ is apt to be affected. A more characteristic stethoscopic sign of the earlier stage of pericarditis, is that first distinctly described by M. Collin, and afterwards by MM. Reynaud, Honoré, and others. It resembles the creaking produced in a new saddle in riding; and the similitude is not only in the nature of the sound, but also in its rhythm, the motions of the heart sometimes presenting the same changes in the sound, that, in the other case, result from the paces of the horse. I have been able to verify this sign in three cases, by examination after death. It no doubt proceeds from the friction of the heart, either less lubricated than usual, or covered with an uneven film of lymph, against the pericardium, in the same way as a similar sound is occasionally produced in incipient pleurisy, (see pp. 89 & 96) and, as it has been lately asserted, in peritonitis also. It is not, however, always present, and, as in pleurisy, the effusion of serum, or the formation of adhesions, must suspend it.

As a bellows-murmur also accompanies the systole of the ventricles, which from increased irritability contract with spasmodic abruptness, the creaking sound of friction may be confounded with this; and it has probably passed as a modification of the bellows-murmur with Dr. Hope and others, who have not recognised it. It may, however, be distinguished by its appearing to be superficial, and generally accompanying the whole of the heart's motions. It is scarcely to be heard

at the upper part of the sternum, or in the carotids, where, on the other hand, the bellows-murmur is very evident. As it is produced only in pericarditis, the leather-creaking sound is a valuable sign; and the more so, as it generally occurs at a period when the inflammation and its products are recent, and are more within reach of remedial measures, than when the disease is more advanced, and characterised by other signs. When this sign is absent, the discovery of pericarditis must often rest on negative evidence, in there being dyspnœa and fever, without the signs of any other disease of the chest.

The signs of effusion are, an irregular fluctuating impulse, some of the strokes of which are soft and double, whilst others are occasionally hard and abrupt, as if the heart suddenly reached the ribs through the liquid. The sounds are generally audible, though weak; the 1st sound being attended with a bellows-murmur. If the effusion is extensive, the pulsations are intermittent, feeble, and scarcely audible, and the 2nd sound is absent, as in all cases of very weak pulsations, the quantity of blood thrown into the arteries being insufficient to tighten the semilunar valves to the degree which produces sound. If the effusion is extensive, the whole præcordial region will sound dull on percussion. The disposition of the fluid, in pericarditis, to accumulate, depends on much the same causes as in pleurisy; and the chapter on that subject may be consulted also on the solid products of inflamed serous membranes, as the remarks given there, are generally applicable to effusions of lymph and adhesions of the pericardium. These derive their peculiarities from the motion to which they are continually subjected, which prevents the lymph effused from forming a smooth surface. Thus, if the lymph is plastic and highly organizable, it is drawn, by the contraction of the ventricles, into transverse rugæ or vermicular ridges, if there be liquid effusion enough to keep the surfaces asunder; but if this be absent, the lymph is drawn by the repeated contact

into a honeycomb shape, like the lining of the second stomach of graminivorous animals, or in some cases into arborescent shapes, like those produced on separating two flat greasy surfaces. Where the lymph is of lower vitality, it adheres less closely to the surface that exudes it, and presents a shaggy or villous appearance.

Adhesions of the pericardium are a common result of pericarditis, when the surfaces come into contact. They may be either partial or general. When partial, they vary greatly in their effects on the heart's action, sometimes deranging it greatly, restraining the freedom of its movements, and occasioning severe palpitations and dyspnœa on exertion, which soon cause hypertrophy, and prove fatal with dropsy, &c.; in other cases, where the adhesions are loose, and occurring near the base of the heart, they embarrass very little its action. It is therefore only in the former case that the jogging or tumbling motion, described by Dr. Hope as characteristic of adhesion, is present; and in a large number of cases, the signs of adhesion of the pericardium are very equivocal. The pericardium may be entirely adherent to the heart, without producing any prominent or distinctive symptoms; and although such a state must no doubt embarrass the free action of the heart, I have repeatedly seen instances of its being found after death in persons who had not lately presented any signs of disorder in the organ. Where the heart is considerably enlarged, and closely adherent by its upper parts, it will pulsate with increased force, but not lower down than natural, as in simple enlargement. But if the adhesion is loose or lower down, this result will not be observed. A bellows-murmur very commonly attends the first sound in adhesions of the pericardium; and if there be no constriction of the valvular orifices, it must be considered to arise from the unnatural abruptness with which the heart, fretted in its movements by the restraint of the adhesion, contracts on the

blood, and causes it to pass with a sonorous friction through the natural orifices.

Softening of the Heart is probably sometimes caused by inflammation, but in many cases it has been found where no signs of inflammation have existed in any form, and it is therefore rather to be regarded as a modification of nutrition, perhaps the result of a cachectic state of the fluids. In carditis, or inflammation of the muscular tissue manifesting itself during life, by increased action, and sometimes pain of the heart, dyspnœa, a very quick, contracted, and hard pulse, the heart has been found in a softened state after death, the ventricles being collapsed and flabby, and being easily lacerated when squeezed or pulled. In cases not preceded by inflammatory symptoms, a greater degree of softness has been observed in the muscular substance of the heart, which is often paler than usual, and sometimes presents a yellowish tint. Laennec considered that this softening rendered the sounds of the heart more obtuse than natural; and this, the result of his experience, we should be more ready to receive, as it accords well with the view lately given of the cause of the sounds; a soft flabby muscle being insusceptible of the degree of tension capable of producing a loud clear sound. There is, however, such a variety in the sounds of a healthy heart, that the mere obtuseness of sound would only give us a suspicion of the existence of softening in those who evince signs of peculiar weakness in the organ, sometimes with attacks of angina and palpitation, during which the sounds are often much clearer. Laennec thought that a partial softening was sometimes produced in the hours of imperfect circulation which precede a lingering death, and I have lately seen a case which was found after a death of this kind.

Diseases of the Valves of the Heart.

The several valves of the heart are occasionally altered in structure in a variety of ways, and if the alteration much impedes their function, a degree of obstruction to the circulation will result. This, by causing dilatation or hypertrophy, according to the degree of irritability in the organ, may become one of the most formidable kinds of lesion incident to the heart. Valvular disease, with hypertrophy, dilatation, or both, produces that terrible array of symptoms by which extensive organic disease of the heart has been long characterized. These are generally, dyspnœa and palpitation, at first only on extraordinary exertion, or mental emotion, but in time becoming aggravated into paroxysms of the worst forms of asthma and orthopnœa, cough, sometimes very severe, with copious frothy serous expectoration, various signs of weak or impeded circulation, such as cold extremities, pains and fatigue in the limbs, and in the advanced stages, lividity of the lips, cheeks, nails, &c. congestions of blood in the liver, spleen, lungs, and other organs, and dropsy in its various forms. The local symptoms are generally pretty prominent, especially when, as is usually the case, the heart becomes enlarged; but the physical signs of dilatation and of hypertrophy are sometimes obscured by the irregularities into which the current of blood is thrown by the valvular obstruction, and by the unusual sounds which sometimes result. When the character and position of these sounds is pretty distinct, they may still guide us to form a right judgment of the case; but where they are obscure, and only doubtfully referrible to particular parts of the heart, the physical signs lose that simplicity and distinctness of character which give them superiority, and in the present state of our knowledge, they will not assist us in our diagnosis of the exact nature of the case, better than the general symptoms. Before we can assign

their due weight to the unusual sounds just adverted to, it will be necessary to investigate the principles on which they are produced.

Besides the two sounds naturally produced in the heart, the 1st by the systole of the ventricles, and the 2nd at their diastole by the flapping of the semilunar valves, there sometimes occur others of a peculiar kind. Such are, the *bellows-murmur*, the *filing*, *rasping*, and *sawing sounds*, and the *purring tremor*. These adventitious sounds Laennec hypothetically attributed to a spasmodic or convulsive action in the heart or arteries. In the first edition of this work, I gave the following opinion: "I am disposed to think, that were we better acquainted with the laws of the production of sound, we might find that it may be excited by the motion of liquids, as well as by that of air, in or against solids of a particular form; and that we might find a more satisfactory explanation of the phenomena in question in the moving mass of blood being thrown into sonorous vibration by some modification in its course. Such a modification might be produced by thickening or irregularity in one of the valves of the heart, or by spasmodic action of some of the columnæ carneæ, by any obstacle in the calibre of an artery, &c.; and these causes might, as in the analogous case of air, render the passage of the blood sonorous, instead of, as it usually is, silent."* M. Bertin had also approached the same view, when he ascribed the bellows sound to the increased friction of the fluid in passing through the contracted orifices of the heart;† and in fact the original opinion of Laennec seems to have been similar. More recently, Dr. Spittal and Dr. Hope have adopted

* The acoustic principle, which is glimpsed at in this opinion, has lately been experimentally established by M. Cagniard Latour, who has succeeded in producing not only sounds, but musical notes, by the motion of liquids in tubes and through apertures. The kind of bellows sound, called musical, will therefore receive a ready explanation.

† Traités des Maladies du Cœur, 1824.

this view, and they have endeavoured to explain, in accordance with it, the various modes in which the bellows and grating murmurs may be produced. It appears to me, that the principle which I had conjecturally advanced, has been fully established; and if we revert to the definition of sound given at page 4 of this work, and compare it with the several cases of the production of the sounds in question, we shall find them all fulfilling its conditions, in a certain *resistance given to the blood moving with a certain force.*

The resistance given to the current of blood, by excrescences, osseous deposits, or a rigid valve projecting into it, is too obvious to require comment, and the only condition required is a certain velocity in the current; if this be too low, there may be no sound, and accordingly we sometimes find that a bellows or rasping murmur, resulting from these causes, will cease when the circulation becomes very languid. An orifice contracted by the adhesion or thickening of valves, developes sound by the vibrating resistance which the edges of the aperture give to the current forced through it. In the same way when through imperfect closure of a valve, the blood regurgitates into the cavity from which it has been expelled, the unnatural chink or opening not being shaped for the free passage of liquid in this reversed direction, a whizzing or blowing accompanies the constricted reflux. This was exemplified in Obs. 6 & 7 of Experiment II. Again, the natural orifices of the ventricles may occasion such resistance to the escape of blood, as to become a cause of sound, and this may happen under two conditions. One is when the heart contracts with unnatural briskness, as in inordinate nervous excitement, in reaction after extensive depletions, and from inflammation or peculiar irritability of the organ itself; the orifices then become *relatively* narrow, and, although unaffected by the current in its natural force, are thrown into vibrations by the convulsive abruptness with which the blood

is forced through them. The other condition is when the cavity of the ventricles, instead of being as usual elongated, and terminating in the arteries somewhat in a funnel shape, are dilated and rounded, so that they do not discharge their contents through the orifices with the usual facility. This cause of murmur, from simple dilatation of the ventricles, was first pointed out by Dr. Hope; and it may be more exactly traced to the decussation and therefore resisted motion of the different parts of the current, as expelled from a cavity of a rounded form. The bellows-murmur produced by pressure on an artery, noticed by Laennec and others, and exemplified in Obs. 3 of Exp. I, and Obs. 5 of Exp. II, readily receives its explanation on this principle of resisted motion. The varieties in the various morbid sounds, which have been called bellows or blowing, filing, rasping, and sawing, from their resemblance to these familiar processes, may sometimes be traced to the nature of the resistance given to the current; but, in other cases, they depend only on the force with which the current passes, the former of these varieties being changed into the latter by accelerated motion of the blood, and the reverse being effected by repose, blood-letting, and other agents that tranquillize the circulation.

The *purring tremor*, which is a remarkable vibration sometimes felt in the region of the heart and along the arteries, and which Laennec compared to the sensation communicated to the hand placed on the back of a cat purring, may be considered as a further degree of the same phenomena, but it includes another acoustic principle, that, namely, of *vibrating systems*. When a cause of vibration exists in the circulation, whether that cause be an ossified valve, inordinate abruptness in the systole of the ventricles, or any other, a greater degree of it will not only occasion a tremor in the spot where it is produced, but will throw the whole blood in the arterial tubes into vibration, exactly as during the strong notes of the har-

monica the whole water contained in them is seen in crispations, and is known to reciprocate as well as to conduct the sound. The vibrations in the arteries may be too gross to produce sound, and they are then felt only as a mechanical thrill, like that of the jarring pulse of irritative reaction after hæmorrhage.

From this consideration of the principles of the production of the abnormal sounds of the heart, the reader will be prepared to conclude, that, arising as they do from such a variety of mechanical causes, they can indicate particular lesions only when their position and collateral signs concur in referring them to some one of these causes. I believe that in many instances this additional evidence may be obtained; but in others, the opinion formed of the nature of the disease can often amount to little more than a suspicion. The generally permanent character of the sounds of fixed valvular or other mechanical lesions, will in some degree separate them from those resulting from temporary irritation; but it must be borne in mind that the former, in certain states, will not produce sounds, and that therefore *their* signs may be but temporary, or may not exist at all.

As it is not the object of this work to embrace minute anatomical descriptions of the results of disease, except in so far as they are useful to illustrate their general pathological effects during life, I shall not detail the varieties of valvular disease which are described in the works of Corvisart, Burns, Laennec, Bertin, Hope, and others; but a slight glance at their general effects on the apparatus of circulation, will be enough to give some insight into their relation to diagnostic signs.

Diseases of the valves may be conveniently divided into two kinds: those which more or less impede the current of blood in its proper direction, either by rigidity or thickening of a part of the valve, by a deposition or excrescence on it, or

by the contraction of its orifice; such lesions may be termed *obstructive.* The other kind comprehends those which, in a shortened, relaxed, broken, or perforated state of a valve, render its closure more or less ineffectual, and permit reflux of blood, in the wrong direction, to take place through it. These for brevity may be called *regurgitant* lesions. Some lesions may be both obstructive and regurgitant; and they must interfere the more with the circulation, as they impair both the opening and the shutting capacities of the valve.

Aortic valves. Obstructive disease in these, may cause a murmur with the 1st sound, and if the obstruction be considerable, the murmur will be so loud and prolonged, that the 1st sound is scarcely audible. According to Dr. Hope, it is heard best about the middle of the sternum, where it seems superficial and hissing; but I have not observed this in all instances; for in persons with a deep chest, and lungs well developed and spongy in front, the sound is often loudest about the cartilages of the fifth and sixth left ribs. In these cases, I have generally found it audible, and sometimes very loud, also at the top of the sternum, and even in the carotids, which has been enough to distinguish its cause from regurgitant disease of the auricular valves. The pulse is generally weak, especially when the obstructive disease of the aortic valves is considerable, and the murmur prolonged. The 2nd sound is also usually weak on the left side, not only because the disease may directly prevent the free flapping of the valves, but also because the aorta is not freely filled with that weight of blood which throws the valves into abrupt tension. Still in moderate degrees of obstructive disease, especially when the action of the heart is increased, the 2nd sound and the strength of the pulse may be but little impaired. Obstructive disease of the aortic valves generally causes enlargement of the heart.

Regurgitant disease of the aortic valves, causes a murmur

instead of the 2nd sound. (This was illustrated in Exp. II. Obs. 6, 7, and 8.) It will be best heard about the middle of the sternum, and is generally short and shifting, and may be distinguished by contrast with the 2nd sound in the pulmonary valves, which still remains audible to the right of the sternum. If the regurgitation be considerable, which is a rare case, the pulse of distant arteries must be weak, whatever be the force of the heart's action. Regurgitant disease of the aortic valves will commonly occasion dilatation with hypertrophy of the left ventricle.

Mitral Valve. Obstructive disease of this valve, commonly consists in an adhesion together, or ossification, or rigidity of some of its parts; or in a thickening and contraction of the fibrous ring at its base. It may cause a murmur with the diastole of the ventricle, and therefore, at the time of the 2nd sound; for although the ventricle in itself produces no sound, yet, when the orifice, by which it becomes re-filled is contracted, the current being partially resisted in passing through, may become sonorous. This will therefore leave the result much as Laennec represented it, inasmuch as there is a current from the auricles to the ventricles during the diastole of the latter, although this current is not produced as he supposed, by the contraction of the auricles. But the results of my late experiments must modify the statements of both M. Laennec, and Dr. Hope, in this respect, that the contraction of the mitral orifice with its impeded current and attendant murmur, will not necessarily supplant the 2nd sound, inasmuch as this sound is seated in the semilunar valves, the action of which may still be perfect. The murmur, when loud, may doubtless drown the sound; and further, there may be connected circumstances which may prevent the full action of the semilunar valves; as for instance, when the quantity of blood supplied from the contracted auricular opening, and through the ventricle, be insufficient to make the

valves flap distinctly at the end of the systole. The same thing is more likely to occur in regurgitant disease of the mitral valve, because in this, much blood is lost backwards, instead of adding at each pulse, to the tension of the arteries.

Regurgitant disease of the mitral valve may arise from causes like those that obstruct the orifice; but a shortened, elongated, or lacerated state of the laminæ of the valve or of the chordæ tendineæ, or an enlargement of the orifice by great dilatation of the ventricle, may also constitute it; and I think with Dr. Hope, that these last forms of lesion are sometimes overlooked in anatomical examinations.* This lesion may obviously produce a murmur with the 1st sound. This sound is said by Dr. Hope to be most distinct at the left margin of the sternum, between the third and fourth ribs; but I have generally heard it best more to the left, as about the nipple or a little below it; and its position here, and that it is not heard in the arteries, have been to me the best indices of regurgitant disease of the mitral valve.† Considerable obstructive disease of the mitral valve, will render the pulse small, but if there be no regurgitation, it may still

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* I lately had a case under my care, in which, although a bellows-murmur had for months constantly accompanied the first sound before death, the heart showed no disease but a thinness and shortness of the membranous portions of the mitral valve, which was remarkable only on attentive observation, and on comparing them with the size of the orifice. The number of the chordæ tendineæ was fewer than usual, but their muscular pillars were inordinately developed, as if from increased efforts to close the orifice. The patient had long shown signs of disease of the heart, and during the fever of which she died, violent palpitation and its consequences were the most prominent symptoms. The pericardium was also uncommonly thin.

† Various circumstances, such as enlargements or adhesions of the heart, solid or liquid effusions in the lungs or pleura, and other causes, may derange these signs, and I am far from giving them as a certain guide. There are very few rules that can be laid down as absolute, in the diagnosis of diseases of the heart, for until all the elements of their signs be known, it is impossible to estimate all their combinations or the consequences of their derangement; and I would again repeat, let these difficulties warn us against precipitate opinions, and excite us to a more systematic and diligent investigation.

be hard, which can rarely be the case when the ventricle empties itself freely both ways; and in this case the pulse is often irregular and intermittent. Either form of lesion generally causes hypertrophy of the left ventricle, with dilatation of the auricle; and in case of extreme obstruction of the orifice, there is sometimes contraction of the cavity of the ventricle.

Diseases of the valves are by far more common on the left than on the right side of the heart; and this probably depends on the greater liability to being strained, and the more fibrous structure of the left valves, circumstances that dispose them to inflammation and consequent change of structure in case of violence and of rheumatic affections.

Semilunar Pulmonary Valves. Lesions in these are perhaps more rare than in any other valves, scarcely occurring except in malformations of the heart. Obstructive disease might cause a murmur, which Dr. Hope says would be heard best at the middle of the sternum, and is to be distinguished from disease of the aortic valves in being more superficial and whizzing. A more easy distinction would probably be found in its being inaudible over the great arteries, by its not affecting the pulse, and by its causing more marked signs of venous congestion and disease of the right side of the heart.*

Tricuspid Valve. Lesions of this valve, although rarer than those in the left valves of the heart, are more common than in the pulmonary valves. They seldom attain the degree of ossification, but generally consist in thickening, cartilaginous deposit, or gluing together of the segments, or in a contracted or dilated state of the orifice. They may cause a deep blowing or filing murmur, which is most distinct under the sternum at its juncture with the fourth rib, regurgitant disease occasioning it with

† If there be also malformation leading to direct passage of blood from the right to the left cavities of the heart, there will probably be that blueness of skin which characterises such congenital errors of structure. See "*Malformations of the Heart*," by the Author, in the *Cycl. of Pract. Medicine*.

the 1st sound, and obstructive disease with the 2nd. If the regurgitation is considerable, it will occasion a pulse in that part of the jugular veins which is close to the clavicles; and this sign occurs especially in considerable dilatation of the right ventricle, in which the tricuspid valves either do not entirely close the orifice, or are so much relaxed and distended at each systole, as to give an impulse backwards to the column of blood in the veins.

When any of the preceding lesions are combined, and their signs are complicated, the obscurity of the case will be generally increased, for unless the locality or the character of the morbid sounds be distinct, the stronger and more prominent one may cover or disguise the other. When the sounds are different, one being filing or grating, and the other blowing, the difficulty is less, and the position and nature of each affection may be often pretty exactly indicated. Rasping or sawing sounds are rarely produced by mere contractions, or by soft depositions, unless for a short time during increased action of the heart; so that when such murmurs are permanent, it may generally be concluded that there is osseous deposit or rigidity in or about some of the valves. Hypertrophy and dilatation often render the signs of valvular disease more evident; and they do so, both by augmenting the force by which the murmurs are produced, and by rendering more distinct the place and order of the two natural sounds and motions, which must always serve as guides to the position and cause of abnormal sounds which may accompany them.

Nervous Palpitation.

This is a very common affection, and like other nervous irritations, may be excited by sympathy with the various other organs, such as the stomach and intestines, the brain, the uterus, &c. and especially by mental emotions. Sedentary

and enervating habits predispose to it, by exalting the sensibility of the nervous system to that state which Cullen called *mobility*. Temporary impressions on the circulation, such as the pressure of a distended stomach on the blood vessels, a constrained posture, a sudden change of temperature, may also, in those whose heart is irritable, cause a fit of palpitation, which in these cases is merely a form of irritative reaction.* The same reaction is shown more permanently in the palpitation which succeeds great loss of blood, or inanition from other causes, whether from evacuations, abstinence, or defect in the sanguific process, as in the various forms of anœmia and chlorosis, which sometimes curiously simulate organic disease of the heart. In all these cases of nervous palpitation, the sound of the heart's pulses is loud, but it does not seem diffused, as in dilatation; the impulse is strong and jerking, but it is not attended with heaving, as in hypertrophy; the bellows or filing murmur which may be present in both heart and arteries, will not be constant, as in valvular disease; and the subsidence and total removal of all these symptoms, when irritation is allayed, and when the pulse regains its natural standard, will sufficiently distinguish nervous palpitation from organic affections of the heart. Nervous palpitation does unquestionably often occur in those whose heart is really unsound, and adds greatly, both to the sufferings and danger of the patient; and one of the strongest impressions which my experience in the treatment of organic diseases of the heart has created, is, that to prevent and counteract states of irritation, both of fulness and defective excretion on the one hand, and of inanition or imperfect sanguification on the other, constitutes the greatest and most important objects of our practice in these cases.

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* See my Essay on Irritation, in the *Cycl. of Pract. Med.*

Aneurisms of the Aorta.

This is one of the diseases of which Laennec said that he had found no pathognomonic sign; and although, by careful examination and study of all the symptoms, considerable degrees of it may be generally discovered, I think with him that we have no constantly certain method of distinguishing it. It may occasionally produce the various several signs of obstructed circulation which are presented by organic lesions of the heart; and as it frequently happens that the heart is at the same time more or less diseased, the aorta may still escape from suspicion. When the aneurismal tumour, pressing on the adjacent internal parts, causes pain under the sternum, or in the throat, extending to the arm, sometimes with numbness, difficulty of swallowing or of breathing, and when the patient experiences an unnatural beating in the upper part of the chest, the disease may yet not distinguish itself from tumours of other kinds which may produce these symptoms. But when an impulse is felt under or a little to the right of the sternum, greater than that felt in the region of the heart, and especially if the impulse be accompanied by a single loud sound, whether this sound be grating or not, there can be little doubt of there being aneurism or considerable dilatation of the aorta. Laennec says that if these phenomena be found constant after repeated examinations, the diagnosis may be considered certain, for it is extremely rare to feel the impulse of the heart beyond its region, even in cases of the most marked hypertrophy. If the tumour can be felt externally, the touch will generally enable us to remove the doubt of another possible case, whether the tumour pulsates in itself, as an aneurism, or being solid, propagates the natural pulsations of the artery under it. It sometimes happens, however, that fibrinous deposits within, after a time made aneurisms feel hard externally.

The passage of the blood through an aneurismal sac at each systole of the heart, is always accompanied with a sound; but this sound varies considerably in different cases. When it is simple, abrupt, loud and rasping, it is most characteristic, and such a sound heard at the top of the sternum or above it, may be considered as distinctive of aneurism. But it is often double, and appears so much like the two sounds of the heart, that it gives the impression of its being the heart immediately under the stethoscope. Dr. Hope asserts that aneurismal sounds may always be distinguished, even when double, by the 1st sound decreasing, and the 2nd sound becoming more distinct, progressively, as the stethoscope is moved from the top of the sternum towards the region of the heart. My own experience does not accord with this statement, for in all the cases of aneurism that I have seen, in which the sound was double, the 2nd as well as the 1st was much stronger over the tumour than in the region of the heart, and Dr. Ferguson cites a fact of the same kind.* I have often observed, that where there is no disease, the 2nd sound is quite as distinct at the top of the sternum as in the region of the heart, and in hypertrophy of the ventricles, it is often much more so. The reason of this and my previous observation, may be readily understood, on the view of the 2nd sound which my experiments have led me to adopt; the tightening valves propagate their sound through a tense arterial column more readily than through the heart, which at that moment has become flaccid, and less adapted to transmit a sound or impulse through it.† Below the top of the sternum, the intervening pulmonary tissue prevents the free passage of the sound, but if this tissue be condensed by disease, or if the aorta be dilated so as to push it aside, the 2nd or valvular sound will then become

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* *Edin. Med. and Surg. Journ.* Jan. 1835.

† See, in illustration of this, Obs. 4 of Exp. II.

more evident there than in the region of the heart. But if the tension of the arterial column be inconsiderable, it may not transmit sound so well as the heart, and in this case, whether in health or disease, the 2nd sound may be only heard in the region of the heart.

On the whole, therefore, although there may be cases in which an impulse, and a single, abrupt, loud sound, with or without rasping, may characterise aneurism of the higher portion of the aorta, or of the innominata, yet many cases occur in which these characters are not present, and in which the signs are therefore more or less equivocal.

Simple dilatation of the aorta takes place most frequently at its arch, which is most exposed to the distending influence of the heart's action. It seems to originate in a disease in the arterial coats, in consequence of which the middle tunic loses a portion of that elasticity which enables it to counterbalance the distending force. Deposits of various kinds, atheromatous, cartilaginous and osseous, often take place at the same time, aud sometimes protrude through the inner coat, which is often also corrugated, and thus the interior surface may be rugged and uneven. In this case, a rasping or sawing murmur in the arteries may accompany the 1st sound, and the only way in which this is to be distinguished from diseased aortic valves, is in the 2nd sound still remaining distinct; and this criterion will fail, if the disease of the valves is only obstructive, and does not prevent their flapping. The sign of dilatation proposed by Dr. Hope, a progressively increasing loudness of the murmur, on exploring from the heart to the top of the sternum, fails, for the reasons before named; and Dr. Ferguson* relates a case in point, in which disease of the aortic valves produced a rasping murmur, loudest at the top of the sternum. Although, then, in individual

* Loc. Cit.

cases, various circumstances may be taken into account, so as to form a pretty correct opinion, we are still without any of those simple and certain diagnostic marks that may be obtained in many other diseases of the chest.

Aneurism of the descending aorta may sometimes be recognised by a short rasping sound, heard at some part of the spine, and not heard in front ; but aneurisms do not always produce this sound, and their pulsations, until they become considerable, may be easily confounded with those of the heart. In the abdomen, they may be more readily felt and distinguished.

EXPLANATION OF THE PLATES.

PLATE I.—*Construction of the Stethoscope.*

I HAVE already exposed the general principles of the construction of the stethoscope; this plate will enable us to come to particulars. We have said that the office of this instrument is triple:—1, As a solid conductor, to convey sounds along its fibres; 2, As a tube to contain a column of air, through which sounds are conducted; 3, As a perforated cylinder hollowed at one end, to concentrate, in the central canal, sounds produced over some extent of surface. Now the perfection of a stethoscope will depend on its being so constructed as to fulfil best all three parts of its office.

First, as a solid conductor. The wood of which it is composed should be of straight and rigid longitudinal fibres, not too dense, and uninterrupted by knots or inequalities; pine, cedar, or cherry wood answer very well. The ends of these instruments should be so formed as to bring the ends of the fibres in close contact with the parietes of the chest, from which the sounds come, and with the ear, to which they are transmitted.

The central canal, which prepares the instrument for the second part of its office, does not materially impair it as a solid conductor. This canal should be perfectly straight, with walls as even and smooth as they can be made, so as to offer no obstacle to the parallel vibrations, and to reflect onwards the oblique ones. It is of essential importance in using the instrnment, that it be so applied to the chest and ear, that this central canal shall have no communication with the external air, otherwise the vibrations would be lost outwardly, instead of entering the auditory meatus. The end applied to the chest should, therefore, be made slightly concave, the better to secure its exact aud perpendicular apposition to it. The form of the other end must be adapted to the ear of the auscultator. If his ear be flat, it may be made flat or very slightly concave; but if the tragus and antitragus are prominent, it must be made concave in a proportionate degree,

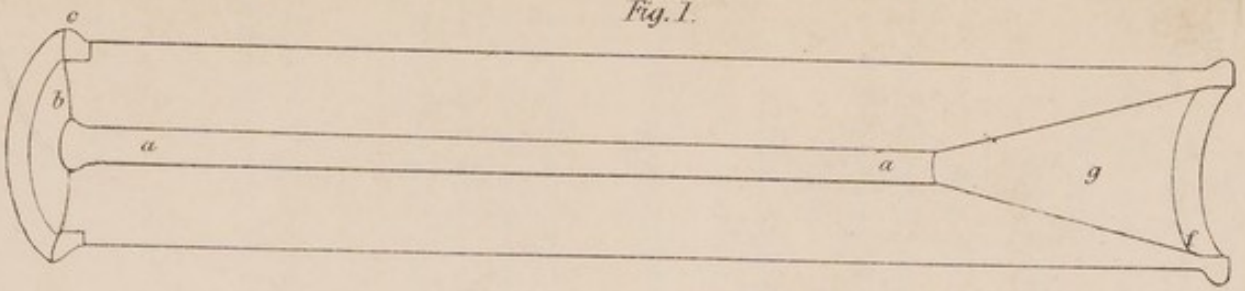
otherwise they may be pushed in, and close the meatus. The end fits better to most ears, by being enlarged a few lines in diameter by a ferule, or ring, (*c*, Fig. 1) either turned in the wood, or made of ivory or a hard wood, and glued on. The ring impedes, rather than assists, the adaptation of the instrument to the ears of those whose temporal zygoma is very prominent; and, in other cases, the breadth and concavity of the ear end of the stethoscope must be proportioned to the size and form of the ear of each auscultator. He should not be hasty in choosing his instrument, but when he has found one exactly to fit his ear, he will obtain a more perfect tact by confining himself to it, than by using a variety.

We have lastly to consider the excavation, which enables the instrument to concentrate diffused sounds; and I have ascertained that the simplest and most effectual kind is a conical excavation, the sides of which subtend an angle of not more than 25°; (*g*, Fig. 1 & 2,) and this form of cavity is now generally adopted. If the angle be greater, it will partake more of the objectionable property of the parabolic curve, which can direct the vibrations aright only by repeated reflections.

The perforated stopper or plug, *e* (fig. 1) by fitting exactly into the cavity, *g*, reconverts the instrument into the perforated cylinder. It ought to fill the cavity completely, and to hold tightly in it by the flute joint *ff*. (fig. 1) or by the tube *h*, (fig. 2.) Fig. 1, represents a longitudinal section of the instrument in all its proportions, one half the real size.

Such I believe to be the best construction to fit the stethoscope for its several purposes. A more portable instrument may be made like fig. 2, which I think is an improvement on that of M. Piorry, inasmuch as the principles which I have exposed are not sacrificed to portability, and it is free from the deteriorating and troublesome adjustments of ivory joints and screws. The ear end, B, may be either turned solid in the wood, or it may be made in a separate piece of ebony, box, or ivory, to slip on the end, D, without screwing. I have found in a ear-piece of this kind, made of caoutchouc, with a little collar of wood, the advantage of admitting a slight degree of motion between the instrument and the ear without displacement, which recommends it for the examination of the chests of children

Fig. 1.



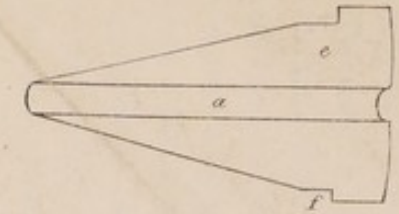


Fig. 2.

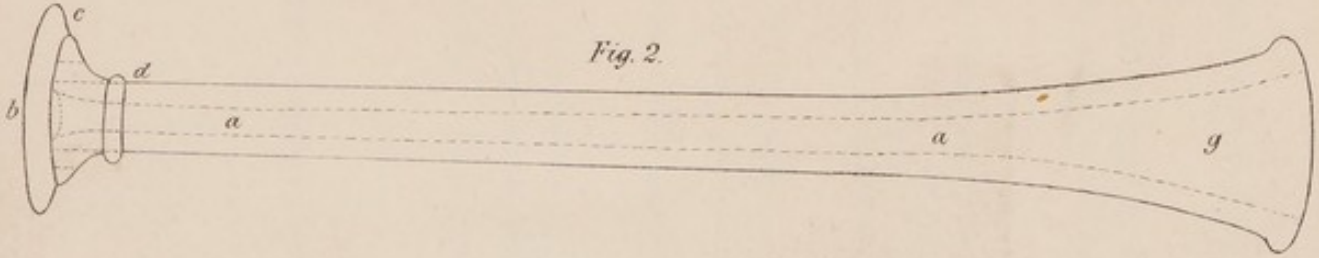


Fig. 3.

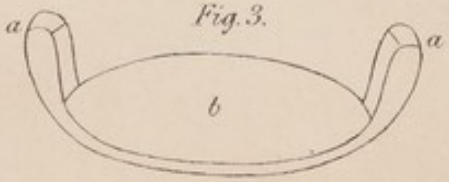


Fig. 4.

Johnson, Litho. 12, Newcastle St. Strand.

and others who cannot be kept quiet; but this advantage is obtained at the expense of a loss of intensity in the sounds. As a solid conductor for the sounds of the heart, the portable stethoscope, (fig. 2.) is perhaps somewhat inferior in power to the cylinder, (fig. 1.) but the difference is not great; and it must be allowed, that much more depends on the attention and method of the hearer, than on the form of the instrument. I must at the same time remark, that there is enough advantage in a well made stethoscope to make it desirable that the student should procure such a one; and I still recommend Grumbridge, turner, Poland-street, as the best maker that I have met with. A supply of his instruments is kept at Mr. Churchill's, 16, Princes-street, Soho.

Fig. 1.—The Cylindrical Stethoscope.

aaa. Central canal.
b. Ear end, slightly excavated and enlarged by the ring *c*.
e. Stopper, fitting accurately the cavity *g*, and holding firmly by the flute joint *f f*.

Fig. 2.—The Portable Stethoscope.

aaa. Central canal.
b. Ear end, made to slip on the end *d*, as shewn by the dotted lines.
e. The Stopper, fitting accurately the hollow *g*, and holding in the canal *a*, by a horn or ivory tube, *h*.

These figures represent the instruments of half their proper dimensions.

Figures 3 and 4 represent the forms and size of pleximeters that I have found most useful. *a a* Are the parts by which they are held. The plate *b*, should be made very thin, and covered on the upper side with soft leather. See p. 19.

Plate II, gives the position of the thoracic regions, and is explained by the figures in Table I.

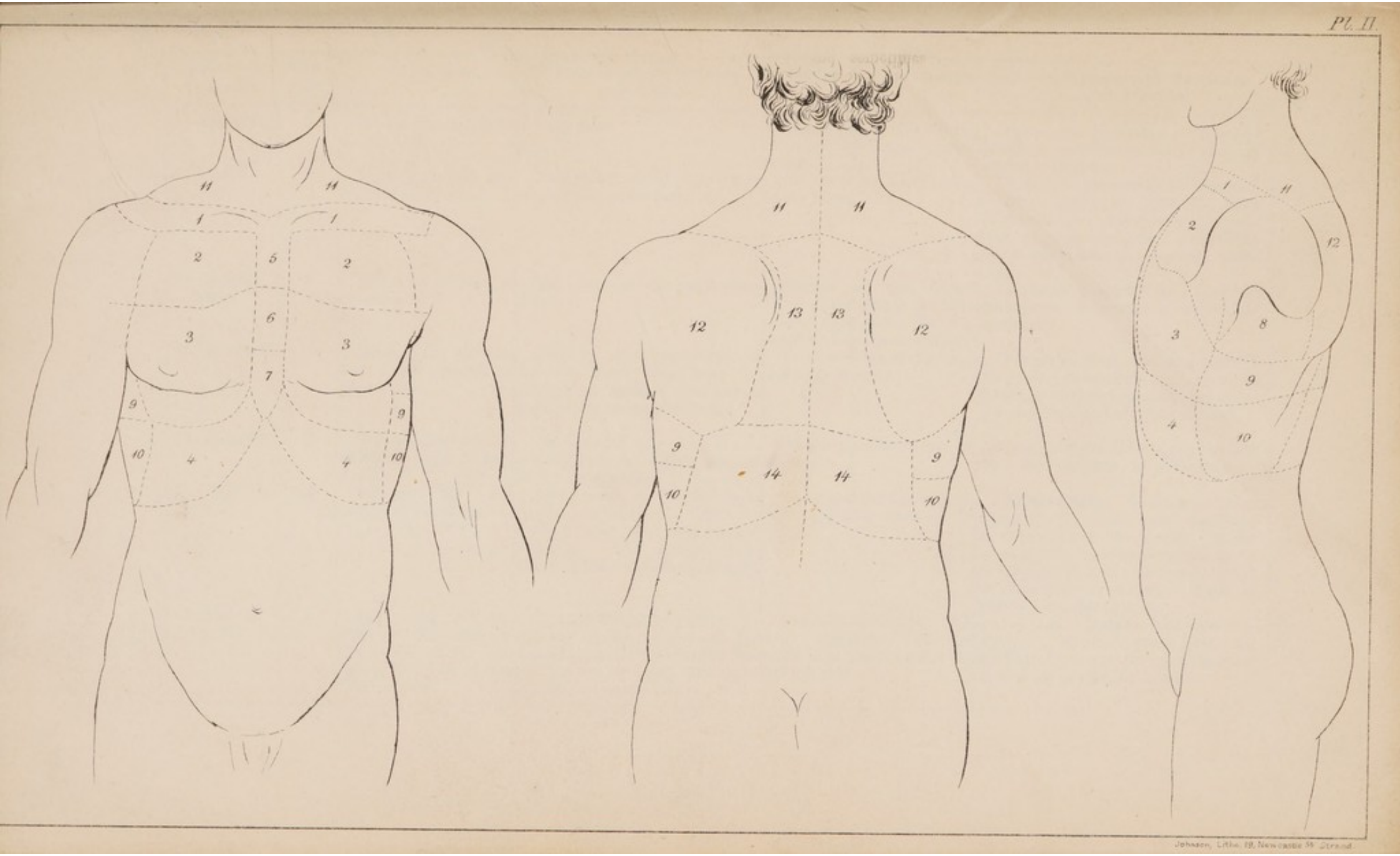
TABULAR VIEW

OF THE

THORACIC REGIONS, IN RELATION TO THE SIGNS OF AUSCULTATION, &c.

TABLE I. *(See Plate II.)*

Regions. Plate II	Number.	Situation.	Natural Sound on Percussion.	Interior Corresponding Parts.	Signs most commonly produced there by disease.
1. Clavicular (subclavian of Laennec).	2	Clavicles.	Very clear towards the sternum: clear in the middle; dull close to the humerus.	Apices of the lungs.	Dulness on percussion in phthisis; generally most on one side.
2. Infraclavian (anterior superior of Laennec)	2	Between the clavicles and the 4th ribs.	Very clear.	Superior lobes of the lungs; large bronchi near the sternum.	Irregular dulness on percussion, diffuse bronchophony, impaired respiration, and afterwards cavernous rhonchus and pectoriloquy, in phthisis. Various rhonchi in catarrhs.
3. Mammary	2	Between the 4th and 8th ribs.	Very clear; particularly by mediate percussion. In women a clear sound can be obtained through the mammæ only by mediate percussion.	Middle lobes of the lungs; large bronchi in the upper part, near the sternum; the heart, generally covered by the lungs, in the lower part of the left region.	Rhonchi in catarrh; more rarely phthisical symptoms. On the left side, dulness on percussion in hydropericardium and enlargement of the heart; increased impulse in hypertrophy, and increased sound of pulsation in dilatation of the heart; constant bellows or rasp sound in valvular disease.
4. Infra. mammary.	2	Between the 8th ribs and the margin of the cartilages of the false ribs.	Dull on the right side: on the left irregularly dull, or unnaturally resonant.	The liver on the right, and the stomach on the left side, covered only on the upper part by the thin margin of the anterior inferior lobes of the lungs.	Crepitant rhonchus in incipient pneumonia. Extinction of respiration in advancing pleurisy. Dry crepitation in interlobular emphysema.
5. Superior sternal.	1	Upper part of the sternum.	Very clear.	Large bronchi.	Bronchial rhonchi in catarrh. Only half the sternum dull on percussion in hepatizations, the whole dull in extensive liquid effusion, of one side.
6. Middle sternal.	1	Middle part of the sternum.	Very clear.	Margins of the middle lobes of the lungs.	
7. Inferior sternal.	1	Lower part of the sternum and ensiform cartilage.	In the upper part clear; rather less so in fat persons. Below, sometimes more dull; sometimes tympanitic.	Above, margins of the lungs; below the heart, liver, and sometimes the stomach.	Signs of diseases of the right side of the heart; dulness on percussion in effusion or fat, in the pericardium, enlarged heart, &c.



Johnson, Litho. 69, Newcastle St Strand.

Regions. Plate II.	Number.	Situation.	Natural Sound on Percussion.	Interior Corresponding Parts.	Signs most commonly produced there by disease.
8. Axillary.	2	In the axillæ above the 4th ribs.	Very clear.	Upper part of the lateral lobes of the lungs. Large bronchi.	Dulness on percussion, cavernous rhonchus, pectoriloquy, &c. in phthisis. Catarrhal rhonchi.
9. Lateral.	2	Between the 4th and 8th ribs at the sides.	Very clear; unnaturally so, in emphysema of the lung.	Middle of the lateral lobes of the lungs.	Dulness on percussion in advanced pleurisy: and on the right side, from enlarged liver. Ægophony in advancing pleurisy: crepitant rhonchus, and bronchophony in advancing pneumonia.
10. Inferior lateral.	2	Below the 8th ribs, at the sides.	The same as the infra-mammary.	Margin of the lateral lobes of the lungs; the liver on the right side; the stomach and spleen on the left.	Crepitant rhonchus in incipient pneumonia. Extinction of respiration in pleurisy.
11. Acromial.	2	Between the clavicles and upper margin of the scapulæ.	Dull by direct percussion. A tolerably clear sound may be elicited by mediate percussion, particularly near the clavicles.	Superior lobes of the lung, and large bronchi.	Dulness on percussion in extensive tubercular accumulation; cavernous rhonchus and respiration, and pectoriloquy in phthisis. Catarrhal rhonchi.
12. Scapular.	2	The scapulæ, and the muscular ridge below them.	The pectoral resonance can be elicited from this region only by mediate percussion.	Middle posterior lobes of the lungs.	Catarrhal signs. Ægophony in pleurisy. Bronchophony in pneumonia.
13. Interscapular.	2	Between the inner margin of the scapulæ.	Pretty clear by mediate percussion, or when the arms are crossed, and the head bowed forwards. The spinous processes of the vertebræ sound well.	The roots, and inner parts of the posterior lobes of the lungs.	Catarrhal signs. In the upper part, sound of respiration never destroyed in effusions into the pleura. In the lower portion sometimes ægophony in pleurisy, crepitation and bronchophony in advancing pneumonia. Signs of diseased bronchial glands.
14. Inferior dorsal.	2	Below the inferior angle of the scapulæ and border of the serrati, to the level of the 12th vertebra.	Clear in the upper portion, by striking on the angles of the ribs, or by mediate percussion. Below, dull on the right, and tympanitic on the left side.	Base of the lungs. The liver encroaches on the right, and the stomach on the left side.	Crepitant rhonchus and bronchophony in incipient pneumonia and œdema; ægophony in pleurisy, and dulness on percussion in both.

TABULAR VIEW

OF THE

CHARACTERISTIC PHYSICAL SIGNS OF DISEASES OF THE LUNGS AND PLEURA.

TABLE II.

Disease.	Physical Signs in the Part Affected.			*Sputa.*
	Pectoral Sound on Percussion.	*Respiratory Sounds.*	*Vocal Resonance.*	
Acute Bronchitis.	Sometimes slightly diminished.	Weak in parts; accompanied by a rhonchus, first sonorous or sibilant, afterwards mucous. Towards the end, respiration sometimes inaudible in parts.	Natural.	Mucous; at first thin, afterwards viscid.
Chronic Bronchitis.	Slightly impaired, if the catarrh is extensive.	Weak in parts, or irregular, with mucous rhonchus.	Natural.	Mucous; sometimes purulent, sometimes streaked with blood.
Dilated Bronchi.	Sometimes partially impaired.	Bronchial, or a gurgling rhonchus.	Bronchophony or even pectoriloquy.	The same.
Pituitous Catarrh.	Partially impaired.	Weak, with sonorous, sibilant, and mucous rhonchi.	Natural.	Pituitous.
Dry Catarrh.	Natural.	More or less extinct in parts. Occasional sibilant, sonorous, and dry mucous rhonchi.	Natural.	A pearly thick mucus.
Spasmodic Asthma.	Sometimes impaired.	Usually weak, or even inaudible; but distinct and even puerile, immediately after holding the breath a while.	Natural.	Sometimes thin & copious at the end of each paroxysm
Peripneumony 1st stage.	A little impaired.	Weak, with crepitant rhonchus.	Natural.	Viscid, of a rusty hue.
2d stage.	Quite dull.	Bronchial, with crepitant rhonchus.	Bronchophony	Rusty & very viscid, or none.
3d stage.	Quite dull.	None; except sometimes a coarse mucous rhoncus.	None.	Sometimes purulent; brown and watery; often none.
Emphysema.	Unnaturally clear.	Diminished, and sometimes almost extinct. Cough accompanied by a sibilant rhonchus or crackling.	Natural.	A thick dirty grey mucus.
Œdema.	Dull, if the effusion is extensive.	Weak, with subcrepitant rhonchus.	Sometimes slight bronchophony.	Slightly viscid and colourless pituita.

TABLE II. *(Continued.)*

Disease.	Physical Signs in the Part Affected.			*Sputa.*
	Pectoral sound on percussion.	*Respiratory Sounds.*	*Vocal resonance.*	
Pulmonary Apoplexy.	Dull, if near the surface, & extensive.	Extinct in the apoplectic spots; a rhonchus, first subcrepitant, afterwards mucous, around them.	Occasionally bronchophony	Blood, or bloody mucus.
Pleurisy.	Quite dull.	First weak; afterwards extinct, except at the root of the lungs.	At first ægophony; afterwards none.	None: or catarrhal.
Pleuropneumonia.	Quite dull.	At first weak, with crepitant rhonchus; then bronchial, afterwards extinct.	Buzzing ægophony.	As in peripneumony.
Hydrothorax.	Quite dull.	Weak or extinct, according to the quantity of the effusion.	Ægophony, when the effusion is scanty.	Various.
Pneumothorax *a.* Simple.	Unnaturally clear.	Weak or extinct, according to the quantity of air effused.	Generally none; rarely metallic tinkling.	Various.
b. With fistulous communication with the bronchi, and liquid effusion.	Dull in the dependent parts; very clear above.	Ditto. Succussion of the chest produces sound of fluctuation. Respiration and cough, sometimes attended with amphoric resonance or metallic tinkling.	Metallic tinkling.	Phthisical.
Phthisis. *a.* Stage of grey induration, and crude tubercles.	Impaired, if the accumulation be extensive.	Weak, or somewhat bronchial, if the accumulation be extensive.	Diffused bronchophony, if the accumulation be extensive.	Sometimes catarrhal and scanty, sometimes pituitous, sometimes bloody.
b. Stage of evacuation of the softened tubercles.	Unequal.	Cavernous rhonchus, respiration, and cough.	Pectoriloquy, when the cavity is empty. Metallic resonance when the cavern is large.	Muco-purulent; purulent; tubercular; curdy matter; sanguinolent, grey or brownish grumous liquid.

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