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THE  
PHYSICAL EXAMINATION  
OF THE CHEST



THE  
PHYSICAL  
OF THE

THE  
PHYSICAL EXAMINATION  
OF THE CHEST

*IN HEALTH AND DISEASE*

BY

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CONSUMPTION AND DISEASES OF THE CHEST AT BROMPTON

WITH ILLUSTRATIONS ON WOOD

HENRY RENSHAW  
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## P R E F A C E.



IN this Manual I have endeavoured to embody prevalent doctrines with regard to the Physical Examination of the Chest so far as they are in accordance with practical experience, and to this end the works of many authorities have been consulted, a list of which will be found at the end of the book.

To the opinions of others I have added observations of my own, the result of many years' experience in the Out-patient and Pathological Departments of the Brompton Hospital, the number of patients that have come under my care being over twenty-two thousand.

It would encumber these few pages too much were I to quote, as I would fain do, all the authors from whom any fact has been gleaned, but it is necessary to acknowledge my indebtedness to the Works of Walshe, which are magistral in statement, accurate in detail, and full of evidence of a delicate, musical appreciation, which quality also characterises the observations of Flint, of New York, from whom I have learnt much.



The illustrations have been put on the wood with much care by Messrs. Evans, from my own drawings, the anatomical diagrams being based upon the works of Pirogoff, Sibson, Weil, and Frorieps. To Messrs. Churchill my thanks are due for leave to adapt two diagrams from Braune's Work. The illustration of Percussion is from the friendly pencil of Mr. Henry Moore.

Although I have taken pains to insure completeness and accuracy, errors will, of course, creep in, and I shall be grateful for any suggestions and emendations which will help to make this Manual trustworthy and useful.

SOUTH KENSINGTON,

*April, 1879.*

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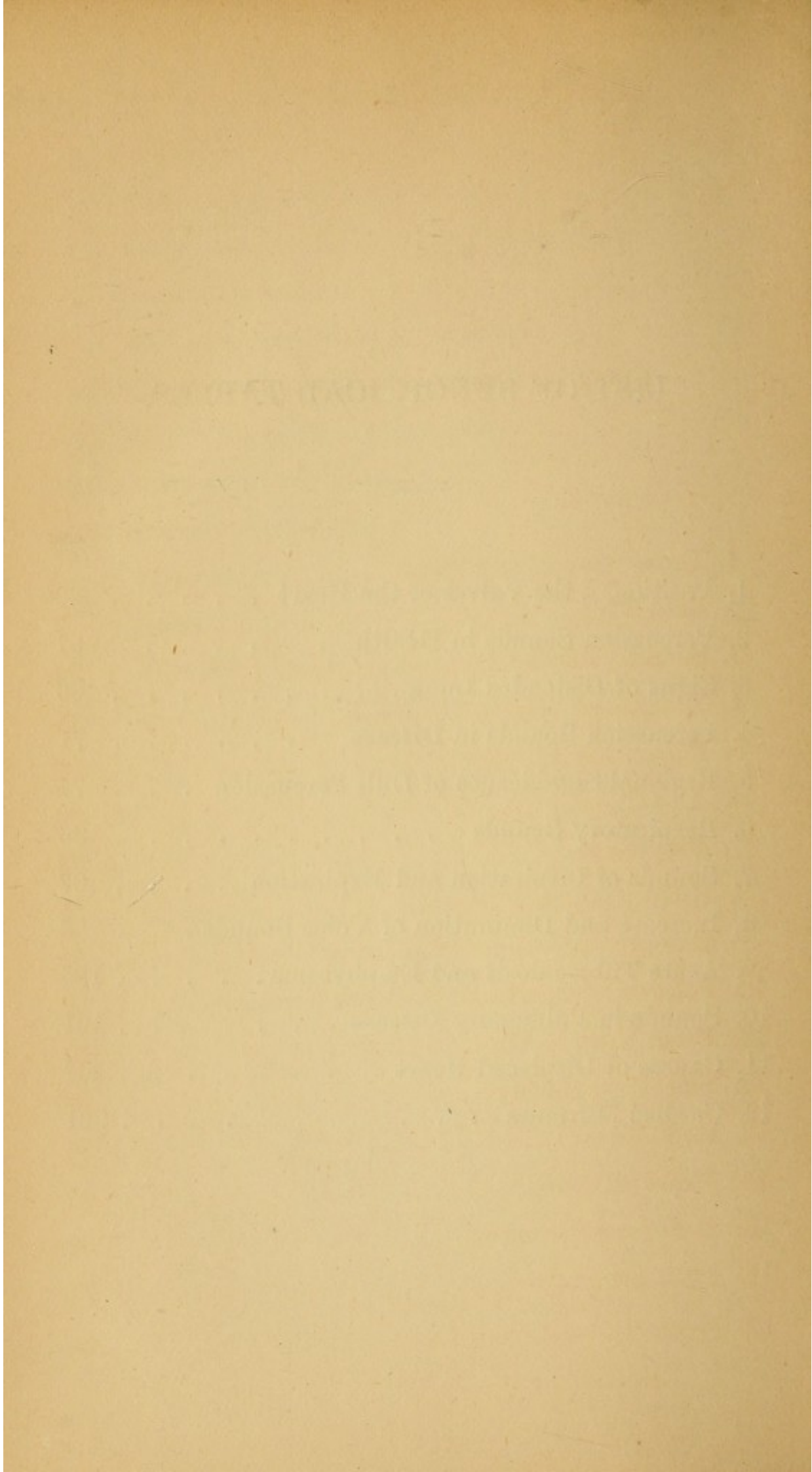
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THE  
PHYSICAL SIGNS  
OF  
DISEASES OF THE CHEST.

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CHAPTER I.

INTRODUCTORY REMARKS.

THE **physical examination** of the chest is made by the use of the three senses—sight, touch, and hearing—applied by special methods to the investigation of the condition of the chest and its contents.

The **object** of such examination is to ascertain the state of the thoracic organs—the lungs, heart, vessels, and other parts contained within the chest—so as to estimate the state of health of the individual under examination and the form of disease under which such a one may be suffering.

The phenomena which are observed by the methods of exploration used in an examination of the chest are called **Physical Signs**, a term applied conventionally to signify the indications of health and disease which are presented to the senses of the examiner, and distinct from **Symptoms**, which are chiefly derived from the answers of the patient given to questions which have reference to the history of the disease and the sensations caused by the disease.



**Physical Signs** are detected by the eye, hand, and ear of the examiner.

**Symptoms** are mainly gathered from details given by the patient.

The following are the methods used in the physical examination of the chest.

1. **Inspection** of the form and movements of the chest, with **Mensuration** and graphic representation; constituting examination by the eye.

2. **Palpation**, or the application of touch.

3. **Percussion**, or the test by striking.

4. **Auscultation** (including succussion), or the test by hearing.

The first of these methods appeals entirely to the eye; the second, and in some measure the third, concern the sense of touch; while the greater part of percussion and auscultation entirely depend upon the sense of hearing.

For the nice appreciation of physical signs, which require much training for their proper observation, constant cultivation of eye, ear, and touch is necessary; the difficulty of the art is great, and the student must always remember that the signs of disease are few compared with the manifold conditions which give rise to them; and it cannot be too often urged that reliance can rarely be placed on a single sign, but that the best results are to be obtained by grouping together all the physical signs and comparing them with the history and symptoms of the case.

In many instances it is found that the physical signs fall short of the amount of disease present, they may be modified by some transient condition; but the physician would no more be warranted in dispensing with the physical investigation of disease by all the means at his disposal than in depending solely upon physical signs for his opinion. Every method and appliance must be resorted to, but each should be estimated at its due value, and the student must neither be too sanguine as to the possibility of forming a just estimate of all forms of



disease by physical signs, nor cast down if he makes mistakes; but he may at the outset of his labours be warned that insidious and dangerous processes do not always reveal themselves to the eye, hand, or ear, and to avoid even egregious blunders much assiduity and caution are requisite. I have often observed that students, who after qualifying themselves by laborious and careful study for the practice of their profession, have applied themselves to the perfecting of their knowledge in the wards of a hospital set apart for pulmonary disease, start with confidence in their powers of diagnosis, but after some experience become disheartened from the number of their failures.

Some comfort may, however, be assured to them, for there is still a third stage to be reached, in which they attain a knowledge of what the art can do. If they are overwhelmed by failures they are in but a sorry plight as regards the profession they have chosen; but continued perseverance will ultimately bring them to that more satisfactory condition of knowing how far they can rely upon signs. Careful students learn more from failures than successes, and in these pages I shall endeavour to keep in view the difficulties of the subject, as well as those signs which lead to the triumphant detection of disease.

For the purpose of examination, the only instrument required is a stethoscope—a full description of which is given in another page: all others should be discarded, at least until some proficiency has been obtained in the use of the hands for percussion, and of the stethoscope. The use of many instruments is never advantageous, and the best kind of work is often done with the simplest tools.



## CHAPTER II.

## THE TOPOGRAPHICAL ANATOMY OF THE CHEST.

BEFORE proceeding to examine the chest the student ought to have an exact knowledge of the general anatomy of the thorax and its organs, and especially of the relative position of those organs with reference to the surface of the chest, so as to be able to say what organ or part of an organ underlies any particular point to which attention is directed.

For this purpose it is convenient to map out the surface of the chest into regions, and to each region are assigned those parts which are comprised beneath it.

The following are the divisions generally used:—A division into right and left is made by an imaginary plane passing through the middle of the sternum and the spine.

The **Right Side** contains—

The right lung, with its pleural covering, bronchial tubes, nerves, and bloodvessels.

The right auricle and part of the right ventricle of heart, the aortic and tricuspid valves.

The ascending aorta, the innominate artery, the right subclavian, and the large veins of the right side.

Below the diaphragm on the right side are the greater part of the liver and the gall-bladder, and the right kidney.

The **Left Side** contains—

The anterior mediastinum, the left lung and its coverings, bronchial tubes, nerves, and vessels.

Part of the right ventricle, the left auricle and ventricle, the pulmonary and mitral valves.



The transverse and descending portions of the aorta.  
 The left common carotid, and the left subclavian.  
 The left recurrent nerve winding round the arch of the aorta.

The left lobe of the liver, the stomach, spleen, pancreas, and left kidney are situated below the diaphragm on the left side.

The surface of the chest—front, back, and sides—is mapped out by an artificial arrangement of imaginary vertical and transverse lines, convenient for the purpose of registering physical signs generally; but for the exact location of a certain sign it will be found preferable to measure its position with reference to some anatomical part—the middle of the sternum, the clavicle, or a rib; and for this a small three-inch rule, which can be easily carried in the pocket, will be found very useful. The nipple is a good landmark in men, but its position varies slightly in individuals and under conditions of disease: it cannot serve for this purpose in women.

The anterior surface of the chest is thus divided:—

The **Midsternal** or **Mesosternal** line is an imaginary division passing down the centre of the sternum. On either side of this, forming the right and left lateral borders of the sternum, are the **Parasternal lines**.

The sternum is subdivided into an **upper** and **lower Sternal region**, opposite the lower border of the third rib. From the middle of the clavicle is drawn a vertical line, which passes through the nipple on each side to the lowest border of the ribs: this is the **Nipple line**. Between these lines, right and left, lies the front part of the chest, which is subdivided by transverse lines thus:—

The line of the clavicle.

A line level with the lower border of the third rib.

And another, level with the lower border of the sixth rib.

The regions which are thus formed are—

Above the clavicle—**Supra-clavicular**.

The line of the clavicle—**Clavicular**.



6 TOPOGRAPHICAL ANATOMY OF THE CHEST.

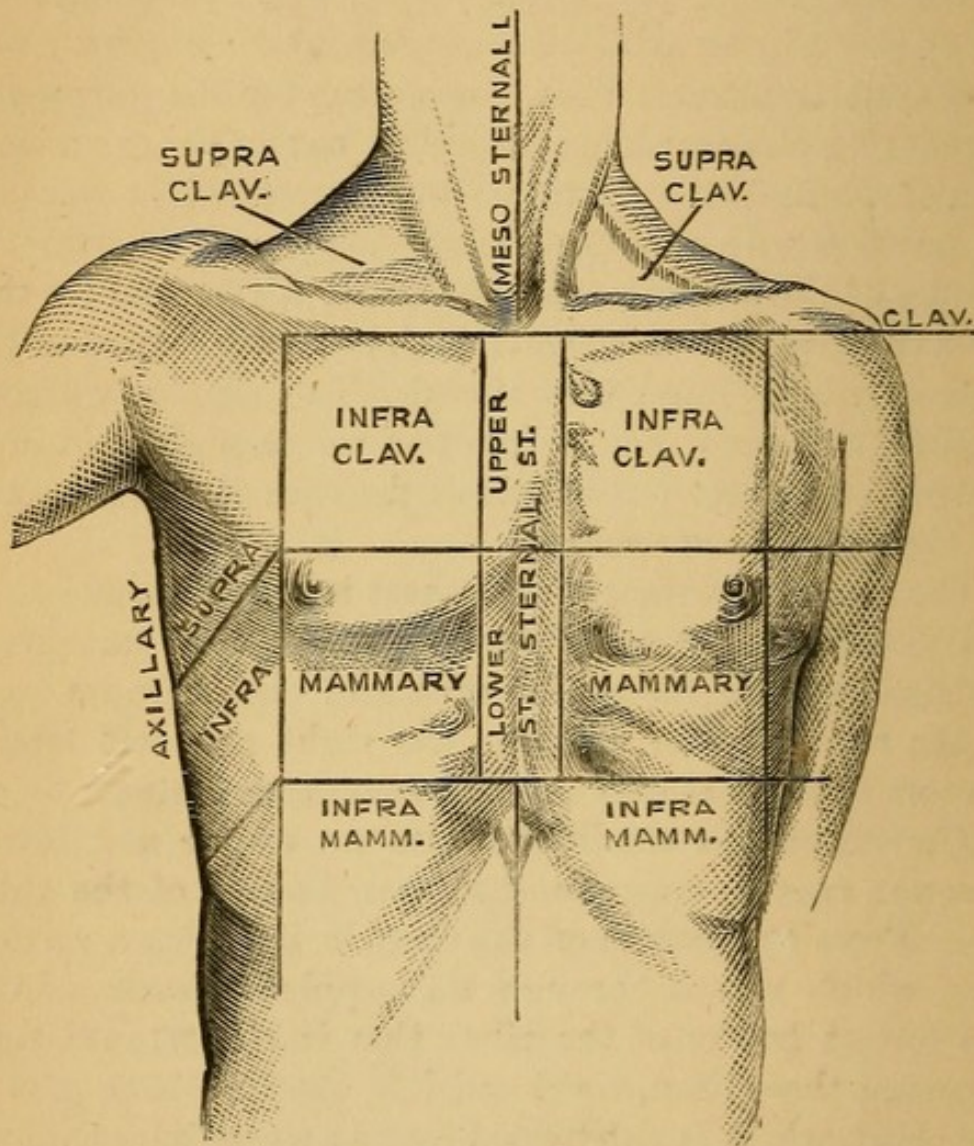
Below the clavicle—**Infra-clavicular.**

Between the third and sixth rib—**Mammary.**

Below the sixth rib—**Infra-mammary.**

The back is divided by the line of the spine and a line falling vertically from the head of the scapula. The subdivisions, which are very simple, are relative to the scapula.

FIG. 1.



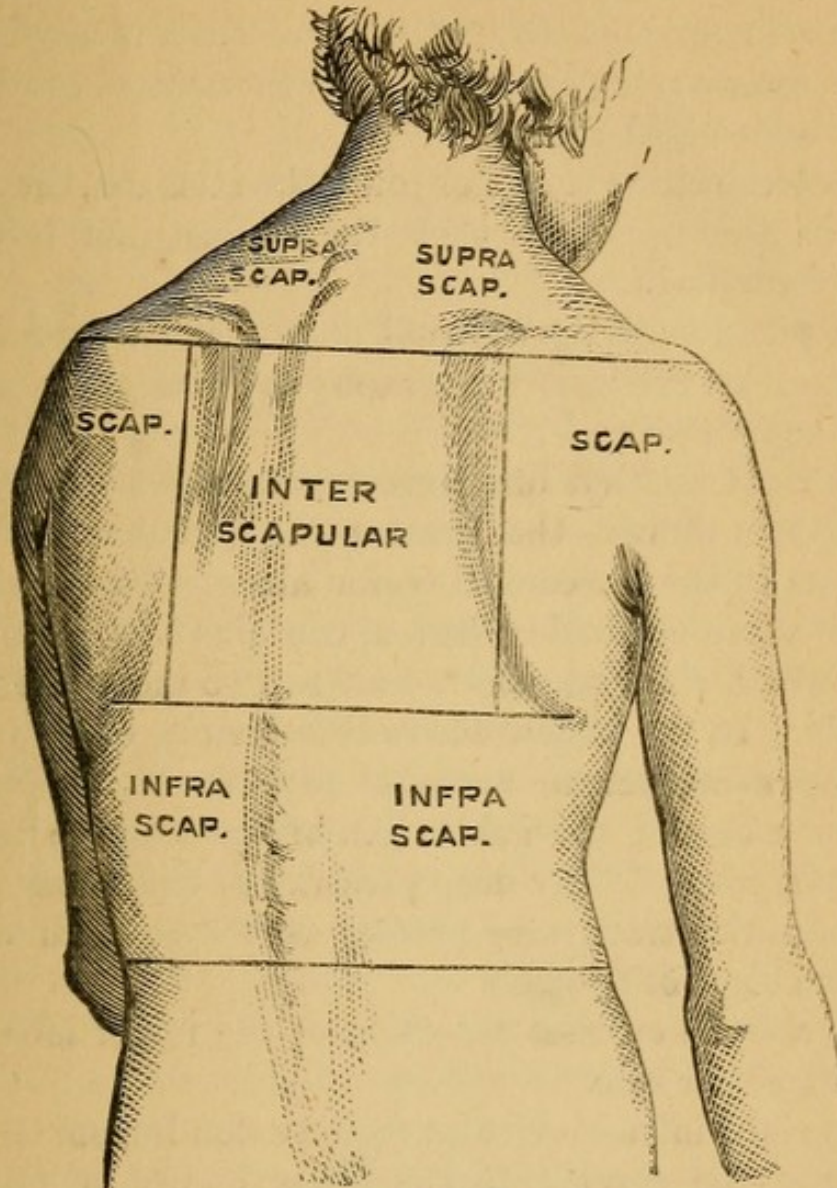
Front View of Chest.

That part of the back between the second and seventh ribs which is covered by the blade-bone is called the **Scapular region**, the part above being **Supra-scapular**. That part which is uncovered, and lies between the two shoulder-blades, is called the **Interscapular region**—right and left.



All that part which lies below the seventh rib is called the **infra-scapular** region.

FIG. 2.



Back View.

Between the line of the scapula behind and the nipple line in front lies the **Axillary region**—right and left—which is subdivided into an upper and lower by the continuation of the transverse line just above the nipple.

These divisions are not difficult, and are very easily mastered by studying the accompanying illustrations. Other regions sometimes mentioned in descriptions of thoracic disease are—

The **Præcordial region**, which overlies the heart, and



extends from the lower border of the second rib on either side of the sternum in front to the sixth rib, and is bounded on the right by a line a finger-breadth to the right of the **Right Parasternal line**, and on the left by a line a finger-breadth to the left of the left nipple line.

The **epigastric region**, which is the central third of the upper abdominal region.

The *scrobiculus cordis*, or pit of the stomach, the depression in the upper part of the epigastrium below the xiphoid cartilage.

The position of the internal organs and vessels in relation to the external topography of the chest may be given as follows:—

The right and left lungs occupy the right and left cavities of the thorax, the heart being situated under the sternum in the *præcordial* region already described.

The supra-clavicular region contains the apex of the lung, which rises one and a-half to two inches above the clavicle. In the hollow above the clavicle, which is called the **supra-clavicular fossa**, close to the sterno-mastoid, the pulsation of the subclavian artery may be felt and here compressed. By deep pressure at the upper part of the fossa the transverse process of the seventh cervical vertebra may be reached.

The clavicle overlies a portion of the upper lobe of the lung on either side.

The right infra-clavicular region extends from the lower border of the clavicle to the lower border of the third rib, and overlies the chief portion of the upper lobe of the right lung.

The right bronchus passes into the lung at the second intercostal space.

The left infra-clavicular region overlies a part of the upper lobe of the left lung.

The left bronchus passes into the left lung at the second intercostal space, a little lower than the right. Over this curves the arch of the aorta, just above the second rib, close to the border of the sternum.



In the second interspace is situated the left auricle, which is sometimes of considerable size in disease.

The right mammary region, between the third and sixth ribs, overlies the middle lobe of the right lung.

The right auricle is situated beneath the second and third interspace, close to the sternum.

The nipple in men is placed between the fourth and fifth ribs, usually in the fourth intercostal space, but sometimes in the fourth, sometimes in the fifth rib.

The **left mammary** region overlies a portion of the upper lobe of the left lung and the greater part of the heart—viz., the right and left ventricles.

The stomach extends to the fourth interspace.

The right infra-mammary region overlies the lower and a portion of the inferior and middle lobe towards the axilla.

The left infra-mammary region overlies a portion of the left lobe of the liver and the stomach, with part of the superior and inferior lobes of the lung.

In the right axillary region is the axillary portion of the right lung, and part of the liver.

In the left axillary region is the left lung, chiefly the inferior lobe, and the stomach.

**Behind.**—The lungs occupy all the region of the back, the bronchi, right and left, being situated in the right and left inter-scapular regions, between the fourth and fifth dorsal spines.

But it must be remembered that the aorta arches over the left bronchus, and passes down to the left of the spinal column in the left inter- and infra-scapular regions.

In the right infra-scapular region is the liver, extending to the level of the ninth rib; and in the left infra-scapular region is a small portion of the liver, the stomach, and the spleen; while below, on either side, is the region of the kidneys.

It is so desirable to know the exact relation of different parts, that the following account is given in alphabetical order for ready reference:—



**AORTA.**—Arch consists of an ascending, transverse, and descending portion.

**Ascending portion** (including the sinuses of Valsalva) starts from the level of the aortic valves situated in mid-sternum, on a level with the third cartilage in front, with the top of the body of the sixth dorsal vertebra behind.

It rises to a level with the lower margin of the second right cartilage, and behind to the upper border of the fifth dorsal vertebra. It has a length of two and a quarter inches. At its root it is covered in front by the appendix of the right auricle, and covers a little higher up the right pulmonary artery. The main pulmonary artery lies to its left, and the superior vena cava to its right.

**Transverse portion** lies directly in front of the trachea, the thoracic duct, and œsophagus; is situated behind the upper portion (manubrium) of the sternum, on a level with the second cartilage. The upper margin reaches the level of the upper border of this cartilage in front, and the third dorsal spine behind. From this border spring the innominate, the left carotid, and left subclavian arteries, all close together. The lower concave border arches over the left bronchus, being crossed in front by the left phrenic nerve, the left recurrent laryngeal nerve winding round it in a direction backwards and upwards.

**Descending portion** turns downwards behind the left bronchus near the fourth and fifth dorsal vertebræ, finally being fixed to the fifth and sixth vertebræ behind the root of the left lung, the œsophagus being on the right side.

**Descending thoracic aorta** lies in front of the left half of the spine from the sixth to the twelfth dorsal vertebræ.

It passes into the abdomen between the crura of the diaphragm downwards and slightly inclined to the right side, lying in front of the body of the first lumbar vertebra.

**ARTERIES.**—**Innominate** lies chiefly within the chest



behind the first bone of the sternum to the right. At its origin it lies in front of the trachea from a level with the upper margin of the second cartilage, and it subdivides into the right subclavian and right carotid just behind the angular separation between the sternal and clavicular ends of the sterno-mastoid muscle.

**Left carotid** springs from the transverse portion of the arch of the aorta to the left of the innominate, and passes upwards along the left of the trachea.

**Left subclavian** springs from the left end of the transverse part of the arch, and rises to a level with the margin of the first left rib.

**Pulmonary artery.**—The main vessel springs from the right ventricle of the heart behind the sternum, a little to the left of the middle line, on a level with the upper border of the third left cartilage or the second interspace.

It lies in front of and above the aorta. It rises to the under border of the arch of the aorta passing towards the left, and at the lower border of the second rib in front (the fourth dorsal spine behind) it divides into a right and left branch which supply the lung with blood for aëration.

The right pulmonary branch, the longer and larger of the two, runs transversely behind the ascending aorta into the root of the right lung, in front of the bronchus, behind the veins (V, A, B), the bronchus being above and the veins below (B, A, V).

The left pulmonary branch passes in front of the descending aorta, and enters the root of the left lung in front of the bronchus and behind the veins (V, A, B), above the bronchus and veins (A, B, V).

**AURICLES.**—The **right** auricle occupies a space behind the sternum between the lower border of the second right cartilage and the lower border of the fourth, from an inch beyond the right parasternal line to the middle of the left half of the sternum. The apex of the appendix is in the midsternal line at a level with the upper border of the third cartilage. Into this auricle open the venæ cavæ; the superior vena cava pours into it



all the blood circulating above the diaphragm; the inferior vena cava, guarded at its mouth by the Eustachian valve, returns all the blood from below the diaphragm.

The **left** auricle lies in front above the left ventricle, on the left of the pulmonary artery beneath the second left intercostal space, and extends from the lower margin of the second left cartilage to the lower border of the third cartilage; behind it curves round and downwards, lying between the fifth and the seventh dorsal spines.

Between the auricles is the auricular septum, which before birth is perforated by the foramen ovale. Into the posterior part of this auricle open the four pulmonary veins, two right and two left.

**Bronchial arteries** supply the lungs with nutrient blood. They spring from the aorta or from an intercostal artery, and are from one to three in number; they supply blood to the bronchial glands, and tubes and coats of the bloodvessels. Their capillaries ramify on the bronchial mucous membrane, and much of their blood is returned by the pulmonary veins to the left side of the heart.

**BRONCHUS.**—The right, horizontal and short (one inch), divides off from the trachea, and is distributed to the root of the lung on a level with the fourth dorsal spine, or between the fourth and fifth dorsal bodies behind and in front, opposite the second right cartilage. This tube is higher in position than the right pulmonary artery and vein.

The left, smaller and longer (two inches), passes obliquely downwards into the left lung, beneath the arch of the aorta, on a level with the fifth dorsal spine, one inch below the right; it crosses the œsophagus and descending aorta. In front it is level with the lower border of the second left cartilage. This tube is lower than the left pulmonary artery.

**THE DIAPHRAGM** may be considered as tripartite, having a right, middle, and left portion.

**The Right** rises as high as the middle of the fourth intercostal space a little below the nipple, the highest point being to the left of the nipple line. In the axillary



region it rises to the sixth and seventh ribs, behind it is level with the eighth rib.

By its contraction it expands the right lung and depresses the liver and right kidney.

**The Middle** portion forms a flat surface, inclining from the ninth or tenth vertebra, forwards and downwards to the lower end of the sternum. Upon it rests the heart, which is lowered and lengthened by its depression.

In front it is attached to the seventh cartilage of each side; between them a triangular gap is left behind the xiphoid cartilage.

**The Left** portion is narrower and lower than the right at its summit by three-quarters of an inch, at its attachment by a quarter of an inch. It does not rise above the fifth rib, and is found at a level with the fifth intercostal space, or the sixth rib; at the sternum, with the seventh intercostal space in the axilla, and with the ninth rib behind. The level varies in individuals. By its contraction it expands the left lung and lowers the stomach spleen and left kidney.

With tranquil inspiration the diaphragm descends half an inch; in forced inspiration from one to two inches. The diaphragm is supplied by the phrenic branches of the fourth and fifth cervical nerves and branches of the sympathetic.

RELATIVE HEIGHT OF RIGHT AND LEFT PORTION OF DIAPHRAGM.

<i>Region.</i>	<i>Front.</i>	<i>Side.</i>	<i>Back.</i>
Right Portion.	4th Intercostal.	6th Intercostal.	8th Intercostal.
Left Portion.	5th Rib.	7th Rib.	9th Rib.

**THE GALL-BLADDER** is situated below the edge of the right lobe of the liver, on the outside edge of the right rectus muscle near the ninth cartilage.

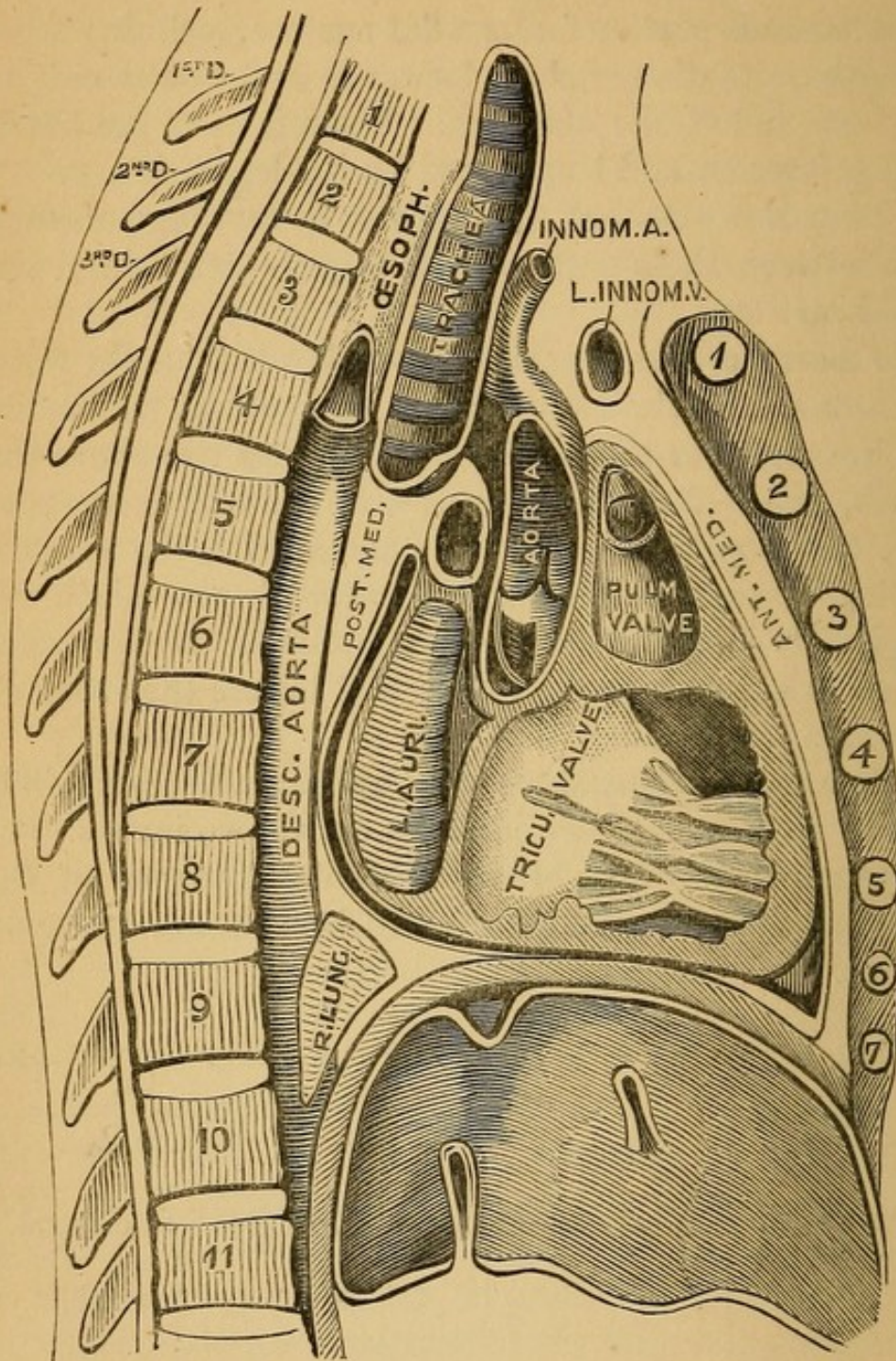
**HEART** occupies the præcordial region, and lies between the lower margin of the second rib and the upper



14 TOPOGRAPHICAL ANATOMY OF THE CHEST.

border of the sixth rib in front, and between the fourth and eighth dorsal spines.

FIG. 3.



Section through Sternum and Spine showing the position of Organs.  
(Based on the sections of Pirogoff.)

The base from the origin of the arteries is indicated by a line drawn through the sternum on a level with the



upper border of the third rib. The auricles extend above this, and are sometimes very large.

The **apex of the Heart** is situated beneath the fifth left intercostal space between the fifth and sixth left ribs in front and behind, on a level with but much to the left of the eighth dorsal spine.

The apex is generally one inch to the right of the left nipple line; one and a half inches below the left nipple; three and a half inches to left of middle sternal line. The heart, on the right side, is in relation with the third, fourth, and fifth right cartilages, and extends about an inch to the right of the **Right Parasternal Line**; on the left it is in relation with the third, fourth, fifth and sixth left cartilages, and extends to within rather less than an inch of the left nipple line.

The auricles, ventricles, and valves are specially described under their names.

A fuller description will be found on another page.

**KIDNEYS.**—These organs, right and left, are situated below the diaphragm, their hilus being opposite the first **lumbar** vertebra. Each kidney is about four inches long and two or three broad.

The right lies between the eleventh and twelfth dorsal spine and the line of the third lumbar spine.

The left is a little higher in position.

**LIVER** extends from the fourth intercostal space to the eleventh right rib, the highest point being a little to the inside and below the right nipple in front and behind from the eighth to the eleventh or twelfth dorsal spine. It comes into relation with the ribs behind, below the inferior margin of the right lung at the tenth dorsal spine.

It has two main lobes, the right and the left, of which the right is much the larger.

The lower edge of the **Liver** is sharp, and extends from the lower border of the right margin of the thorax upwards towards the apex of the heart, nearly to the left nipple line.

Below the xiphoid cartilage its edge is from half an inch



to two inches towards the umbilicus ; transverse diameter, twelve inches ; thickest part, three and a half inches.

In the right axillary region it is covered by the six lowest ribs.

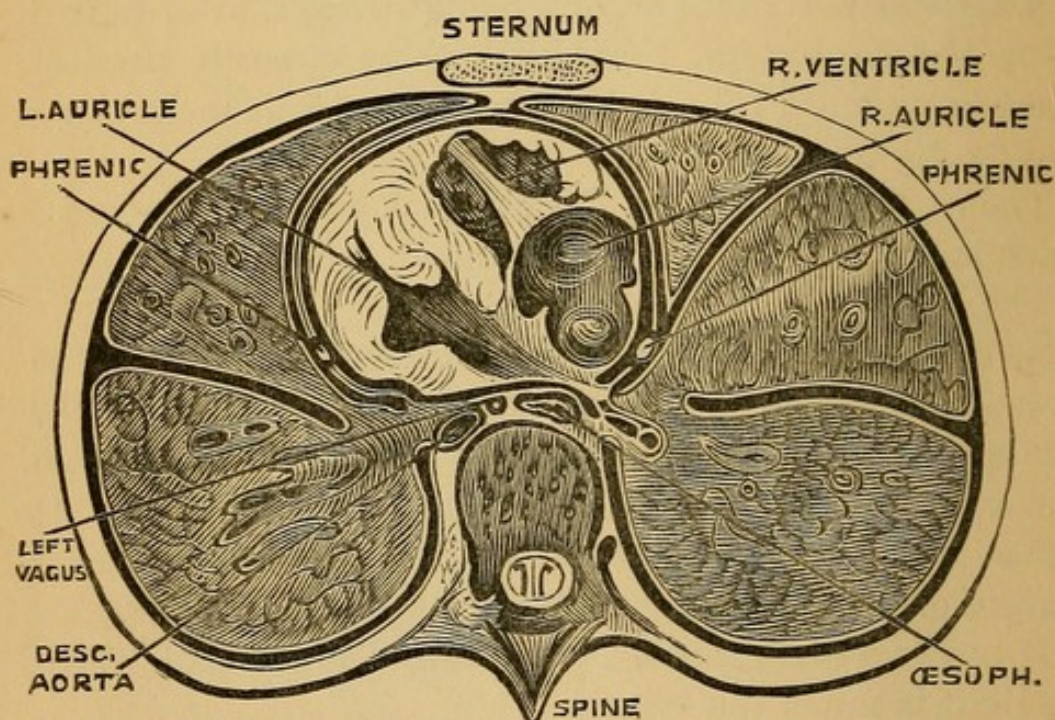
The left lobe hides the lesser curvature of the stomach, the pyloric and cardiac orifices.

**LUNGS.**—**Right lung** is divided into three lobes. **Upper lobe** extends from a point one and a half to two inches above the clavicle, opposite the lower edge of the seventh cervical spine to the upper margin of the fourth right cartilage.

**Middle lobe**, from the fourth right cartilage to the fifth or sixth intercostal space.

**Lower lobe** is bounded in the mammary region by the sixth rib, in the axillary region by the eighth rib, and behind it descends to a level with the tenth and eleventh dorsal spines.

FIG. 4.



Transverse Section showing portion of Heart and Lungs. (Braune.)

**LEFT LUNG.**—**Upper lobe** extends from a point one and a half to two inches above the clavicle, opposite the lower part of the seventh cervical spine, to behind the



sixth left cartilage, and ascends upwards through the axillary region towards the base of the spine of the scapula, and finally terminates opposite the third rib, three inches below the apex of the lobe.

Below the fourth rib on the left side this lobe forms a semilunar retraction, receding from the anterior surface of the heart; it curves round again towards the sternum over the apex of the heart in a peculiar kind of tongue.

**Inferior lobe** extends from the sixth left cartilage obliquely upwards and backwards to opposite the third left rib at its junction with the vertebræ. The lower margin of this lobe and the lung starts from behind the sixth left cartilage in front, and extends obliquely downwards and backwards, crossing in succession the seventh, eighth, ninth, tenth, and eleventh ribs. Behind, its lower margin reaches a level with the eleventh dorsal spine.

**Lymphatics** are superficial and deep.

The superficial or subpleural lymphatics form a network beneath the pleura.

The deep lymphatics arise from the walls of the air-cells and accompany the pulmonary artery and vein (perivascular lymphatics), and others from the walls of the bronchi accompanying the bronchial tubes (peribronchial lymphatics). All enter the bronchial glands at the root of the lung.

**MEDIASTINUM.**—**Anterior** is situated a little to the left of the mid-sternal line, and is bounded by the sternum in front and the pericardium behind. At its upper third it encloses the remains of the thymus gland; in its middle the two lungs come close together, while at the lower part the left lung recedes to leave the heart uncovered.

**Posterior** lies between the posterior surface of the pericardium in front and the spine behind.

It encloses the following parts:—

Œsophagus,	Pneumogastric nerves,
Vena azygos,	Descending aorta,
Thoracic duct.	



**Nerves** which supply the lung come from branches of the pneumogastric nerves, and the sympathetic from which the anterior and posterior pulmonary plexuses are formed.

**Nipple** in the fourth intercostal space, sometimes in the fourth, sometimes in the fifth rib, about four to five inches to the left of the mid-sternal line.

Its position varies, especially in disease.

**Esophagus**, or gullet, leads from the pharynx to the stomach, and lies between the fifth or sixth cervical vertebra, and the ninth dorsal vertebra, where it merges into the cardiac end of the stomach. Length, nine to ten inches.

**In the neck** it lies behind the trachea, the recurrent laryngeal line lying between the two on each side; **in the thorax** it is fronted by the lower part of the trachea and the left bronchus above, and by the posterior part of the pericardial surface.

The aorta lies to the left, the vena azygos to the right; the pneumogastric nerves being on either side.

**Pulmonary vessels.**—The functional supply of blood to the lungs is through the pulmonary arteries, which subdivide (as they accompany the bronchial tubes) and terminate in capillaries, which form a fine network on the inner surface of the air-sacs. They do not anastomose, and the blood is returned by the pulmonary veins, which also receive the greater portion of the blood supplied by the bronchial arteries.

**Scapula** lies upon the back part of the thorax, between the level of the second and seventh ribs. The spine of the scapula in the usual upright position is nearly horizontal and level with the clavicle. It is just over the septum, between the upper and lower lobes.

The base of the spine is level with the third dorsal spine; the lower angle is level with the seventh or eighth dorsal spine, and moves between the seventh and ninth ribs.

The two spines are  $5\frac{1}{2}$  inches apart when at rest,  
                                   9 inches when stretched,  
 $8\frac{1}{2}$  inches in the ordinary position  
                                   for percussion.



The lower angles are 6 inches apart at rest,  
 13 inches when stretched,  
 10 inches in the ordinary position  
 for percussion.

**Spleen** lies between the axillary lines on the left side, beneath the ninth, tenth, and eleventh ribs.

Its upper edge is level with the ninth dorsal spine,  
 „ lower „ „ eleventh dorsal spine.

**Stomach** varies much in size, sometimes contracted and empty, sometimes full and distended.

Five-sixths of the viscus lies to the left of the mid-sternal line; one-sixth to the right.

The **anterior** surface is in relation with the anterior abdominal wall, the diaphragm and the under-surface of the liver, chiefly on the left side.

The **posterior** surface is in relation with the diaphragm, pancreas, transverse duodenum, spleen, left suprarenal capsule, and kidney.

The cardiac orifice is to be found at the seventh left costal cartilage.

The pylorus lies generally three finger-breadths below the ensiform cartilage, to the right of the middle line; but this varies very much at times, and I have seen it in the pubic region.

**Thoracic duct** starts from the first lumbar vertebra, and passing through the diaphragm to the right of the aorta, ascends upwards behind the arch of the aorta and to the left of the œsophagus, opening into the internal jugular or subclavian vein on the left side.

**Trachea** lies in the middle of the neck, and extends from the level of the sixth cervical vertebra to the level of the fourth or fifth dorsal vertebra behind, and the second rib behind the manubrium of the sternum in front. It is four to five inches long. On its right lies the innominate artery, on its left the left common carotid; while behind it lies the œsophagus.

**VALVES—Cardiac.**—Aortic valves consist of three semilunar or sigmoid valves, one of which is posterior and



two anterior. Perhaps the best method of naming them is derived from their position as regards the inter-ventricular septum: one being septal, the others anterior and posterior respectively. They are situated behind and below the pulmonary valves, and in front of the two auriculo-ventricular openings. They lie behind the sternum near the middle, and on a level with the lower border of the third rib in front, and the body of the sixth dorsal vertebra.

**Mitral** extends from the third left cartilage, and lies behind the left half of the sternum at this level, and between the sixth and eighth dorsal spines behind.

**Pulmonary** may, like the aorta, be considered as having septal, anterior and posterior segments; lies in front of and above the aortic valves, and to the left behind the left border of the sternum, encroaching upon the second interspace.

**Tricuspid** lies behind the lower half of the sternum, in front of and to the right of the mitral valve, from the sternum close to the third left cartilage, to the fifth right cartilage.

All these valves are so close together that they may be all covered by a threepenny-piece (half-inch). While a shirt button (quarter-inch) would include portions of all but the tricuspid.

<i>Valve.</i>	<i>Position.</i>	<i>Average Size in Inches. (Health.)</i>	<i>Extreme Size. (Disease)</i>
Aortic.	Mid-sternum. Lower border of third cartilage.	3·1	4·0
Pulmonary.	Upper border of third left cartilage, or Second intercostal space.	3·5	4·8
Mitral.	Third left cartilage.	4·0	5·3
Tricuspid.	Between third left and fifth right cartilage.	4·7	5·6



**VEINS—Pulmonary.**—From each lung two proceed from the junction of branches arising from the pulmonary capillaries. In the root of the lung they are anterior to the bronchus and artery, and finally terminate in the back of the left auricle. They anastomose freely in the lung and have no valves.

**Cardiac** spring from the coronary capillaries of the heart and terminate in the coronary venous sinuses, which open into the right auricle between the inferior vena cava and the tricuspid valve.

**Superior or descending** vena cava receives the blood from the head, neck, upper limbs, and walls of chest. It opens into the right auricle at upper posterior part. Just above the pericardium it receives the azygos vein.

The **azygos vein** receives the blood from the back of the chest. At the lower part of the chest it is divided into a right and left vein, the right being connected with the right lumbar vein, and the left with those of the left side. This vein has valves and forms the continuity between the superior and inferior system of veins.

**Inferior or Ascending** vena cava carries the blood from the lower limbs, the abdominal walls and organs. It opens into the lower posterior part of the auricle, and has no valves, except the Eustachian valve at its orifice.



## CHAPTER III.

## THE MOVEMENTS AND ACTION OF RESPIRATION.

A SHORT account of the movements of the chest and the alterations of the thoracic organs by the action of respiration is necessary before proceeding to an inspection of the chest.

The amount of breathing surface in the lungs is so far above the ordinary requirements of the body that in ordinary healthy breathing there is very little expansion of the chest-walls; the function of respiration being carried on chiefly by the action of the diaphragm, even this apparatus working easily and descending only half an inch; while the thoracic movements are not more than one-twentieth of an inch; sometimes in the robust, according to Sibson, only one-thirtieth of an inch.

How is it then that the air is changed in the upper lobe with so little expansion of its case? The explanation appears to me as follows; and although it is advanced simply as a theory, I must say that I know not otherwise how to account for some of the phenomena that occur in auscultation—as, for example, why the respiratory murmur should be heard so loudly in some cases of cavity when we know by inspection that there is no expansion of the corresponding chest-wall.

A vacuum would be produced by the action of the diaphragm but that the lung expands at its lower border in correspondence with its action. This expansion can only act directly on the descending bronchial tubes, but air is drawn by this means along the main bronchus. It will rise and fill the upper lobe if any air has passed out of it. This withdrawal of air is, I believe, effected by



means of the primary current which is forced out of the lower part of the lung by the action of the diaphragm ; which current by its passage outwards in some measure exhausts the air of the upper lobe, taking a portion with it, just as happens in the spray-producing instruments by the action of the hand-ball.

The activity of the upper lobe is certainly less than that of the lower lobe, and it is less altered in dimensions by compression ; hence it follows that its function is not so actively performed, the ventilation of this portion is not so complete, the bronchial tubes are more open, and the circulation is not so active.

In forced breathing the thorax is more active, while the action of the diaphragm is also increased. The movement begins with the diaphragm, and the chest gradually expands from below upwards. Ransome made experiments by fixing tapes of paper on the chest, and he found that the order in which the tapes were torn was from below upwards.

The lower rib is the first to rise and the last to descend. Under these conditions the thoracic and abdominal walls advance from one to two inches, and the girth of chest and belly is increased two, three, or even four inches, the diaphragm descending one or two inches.

The diaphragm by its action lengthens the lungs, which are thus lowered about two inches. It lowers and lengthens the heart about an inch and a half, and it depresses the liver from two to three inches. During deep inspiration the lower margin of the lung descends (to quote Sibson) in front from the sixth to the seventh cartilage, in the axilla from the seventh, eighth, and ninth ribs to the eighth, ninth, and tenth ; behind, from the tenth to the twelfth.

During the descent of the heart the lungs expand and cover a great portion before exposed, the apex is lost and the inspiration is felt at the xiphoid cartilage, and the vessels descend with the heart just as the bronchi descend with the trachea and the larynx in company



with the lungs. The stomach, spleen, and kidneys are in like manner depressed, the pylorus being thrown down two inches, and the kidney one inch.

In extreme inspiration the spine is fixed and the action of all the upper muscles of the trunk is called into play, and the clavicles and the shoulders are raised. The sternum moves forward and upward, the lower portion having the more motion.

The greatest movement of the ribs takes place in a line half-way between the sternum and the chest, the fifth rib having generally the greatest movement.

Respiration is the same in children of both sexes up to fourteen years of age, but after this the respiration in women becomes less diaphragmatic and more thoracic, and in adult females the thoracic movement is greater than in men. This sexual difference of respiration has a very marked effect in disease, and attention will be drawn to some distinctions between the two sexes in auscultation, and also in pathology. This difference is also important in the examination of the patient; and whereas it is necessary to examine the whole chest in a man, it is sufficient in early cases of disease to examine only the upper part of a woman's chest, front and back, and convenience is really in accord with the requirements of the case.

The influence of respiration in disease has been greatly overlooked, and its action as a morbid agent is only recognised in the inhalation of irritative particles—smoke carbon, fluff of wool, stone, clay, and iron; but its activity in planting morbid secretions and blood has escaped recognition. In a treatise on "Pulmonary Hæmorrhage" I have endeavoured to show how blood becomes planted in the lungs by the inspiratory force, and a full description will be found in another page; but a considerable pathological experience convinces me that this is also true of morbid secretions which are affected not only by the force of inspiration, but equally with all other matter by the force of gravity.

The result of this experience is to show that fluids



slowly secreted in the lungs have a tendency to assume a position in accordance with the laws of gravity, and seek the most dependent parts, especially the back; and matter, fluid or solid, which comes under the influence of inspiration is planted in those regions in which the action of inspiration is not only *first* felt, but also *chiefly* felt, owing, in the latter case, to expansion of the lung.



## CHAPTER IV.

## ON THE MODE OF CONDUCTING AN EXAMINATION.

WHEN the student has thoroughly mastered the topography of the chest and its movements in respiration, he will be in a position to make a physical examination of the chest, and a few remarks may be offered on the proper method of conducting such an examination.

For the examination of men the body should be stripped to the waist, the patient standing upright in front of the examiner with his arms hanging down by the side. The position which is most convenient will depend upon the relative height of the examined and the examiner. If the patient is tall it is difficult to examine the supra-scapular region, and for this purpose a sitting posture should be assumed, the patient reclining back when the anterior surface of the chest is examined, and sitting forward, with back slightly arched and arms hanging down between the knees, for the examination of the posterior surface.

If the upright position be assumed for the examination of the posterior surface, which is generally by far the most convenient posture, the patient should stand at ease with the hands clasped, the arms hanging down in front, the back slightly arched, the shoulders dropped, and the head inclined downwards.

The object of this position is to insure the forward movement of the shoulder-blades and a relaxed condition of the shoulder muscles, which would, if contracted, interpose a quantity of non-sonorous tissue between the lungs of the patient and the ear of the examiner.

In the examination of women, exposure should be as far



as possible avoided. From the peculiarities of respiration, to which I have already alluded, and from the conformation of the chest in women, the upper part of the chest is the most important region for examination ; and sufficient evidence is generally to be obtained as regards the anterior surface of the chest by opening the dress in front without any undue exposure. When the cardiac region has to be examined the stays must be removed, and a thin undergarment of linen only should cover the chest. For the examination of the back, which should never be omitted, the dress should be loosened and thrown upwards and backwards from off the shoulders ; in this way there is no difficulty in making a full examination of the back down to the waist without offence to the most fastidious.

All flannel and thick tissues should be removed, as they interfere and mask by their own rubbing and creaking the proper sounds of the chest.

If the patient be examined in bed, a pillow should be so arranged behind, close to the lower part of the trunk, as to avoid any unnecessary exposure. The examiner needs to observe the greatest delicacy in his treatment of women ; let him be as tender as possible in the use of percussion, let him avoid all roughness and harshness of manner, making no more examination than is absolutely necessary, exploring the chest rapidly but quietly, and he will have little difficulty in securing the goodwill and confidence of his patient, while he obtains all the evidence which is requisite for the complete diagnosis of the case under consideration.



## CHAPTER V.

## INSPECTION.

THE ocular examination of the chest is termed inspection, observation being made by the eye on the form, size, condition, and movement of the chest.

A great deal of knowledge may be obtained by a glance, if only the eye be properly trained, and it is a very good exercise for the student to endeavour to find out simply by inspection how close a diagnosis he can make on a case of pulmonary disease.

The size and form of the chest should be first noted; the condition of the surface, the skin and the muscles, the distance between the ribs, the symmetry of parts, and the relative movements of respiration on both sides and in various parts. The nicest appreciation of movements is often obtained by holding the head to one side, and below the shoulder of the patient, and glancing upwards so as to obtain a view of the surface outline of the chest against the light from the nipple below to the opposite shoulder above. In this way, and especially by testing both sides, a very accurate idea of the relative expansion of different regions can be obtained more readily and satisfactorily than can be done by the instruments of precision invented for this purpose.

As an example of the advantages of inspection over measurement, the following case may be given :—

A patient is suffering from an empyema, or collection of pus in the left side. The right lung being extremely expanded to make up for the pressure on the left, measurement shows that the size of the right side is seventeen



inches and five-eighths, that of the left side being seventeen inches and three-eighths.

In this case measurement alone is neither a proof that disease is present, inasmuch as the right side is usually larger than the left, nor does it indicate on which side the disease is present.

The eye at once on inspection detected a localised bulging in the left infra-scapular region, and a view of the front part of the chest showed that the right side was rounded, while the left side was flattened—the expansion of the right lung being due to a compensation for the empyema.

Measurement by instruments of all kinds is useful for registering signs for future reference; but for the purposes of diagnosis the trained eye is a more delicate and useful instrument, and of the greatest precision.

The following points should engage the attention of the examiner:—

#### **The Condition of the Patient.**

This may be soon gauged by practice, and an experienced eye can form an opinion not only as to the state of health of the patient, but in great measure as to the general mode of life.

For robust health the chest should be full, and its lines rounded: the bones should be well covered, no deep cavities below or above the clavicles, the skin soft and clear and moist, the muscles plump and rounded.

**Colour** must be also observed as well as form. The general hue of health is a rosy ruddy colour, which is lost in disease. In anæmia the predominant colour is an ivory-white; the lips are pale. In chlorosis a slight green tint is sometimes perceptible, and a deathly hue of white is indicative of anæmia perniciosa.

Diseases of the heart give indications of their presence; aortic regurgitation being shown by a peculiar straw colour; mitral regurgitation by purple lips and cheeks. Liver disease is shown by a tint of jaundice; while amyloid disease is often indicated by a peculiar patchy, pink and yellow, appearance of the superficial capillaries of the skin.



Fatty degeneration is suggested by the arcus senilis of the pupil, and yellow patches on the conjunctiva often indicate renal disease.

Syphilitic cachexia is marked generally by a peculiar copper hue.

Malignant disease is also recognised by its dark tint and peculiar aspect.

The mode of life may in some measure be ascertained. If the skin is muddy and unhealthy, and the cheeks be patchy, the circulation not being vigorous, the surmise may be hazarded that little exercise is taken, that the patient keeps indoors, or is confined for long hours at work.

Beer-drinking is detected by the condition of the veins, which are full and distended, especially those of the extremities; by the condition of the breath, which has a peculiar sweet smell.

Spirit-drinking is shown by the harsh, dry condition of the surface, and by the smell of alcohol in the breath.

The nose, it should be mentioned, can detect many diseases—for example, diabetes, ozæna, uterine disease, and even syphilis.

**Form of Chest.**—The typical form of the healthy well-developed chest of an adult man needs little description. It should be symmetrical, well covered, the muscles full and round, the clavicles horizontal, the shoulders level, and the bones but little visible.

For the full-developed form of chest, systematised exercise of all the thoracic muscles is requisite, and in practice, certain trades and occupations are found to develop certain muscles on one side more than the other. As a rule, the right side is larger than the left, unless the individual is left-handed.

But peculiar forms of chest are compatible with health, though not with strength. Rapid growth during youth often results in malformations of the upper part of the trunk. A lad at school may grow at the rate of six or eight inches in the year. This growth, if left to itself



without care and attention, monopolises the resources, and the chest consequently expands very little—nay, may actually diminish in girth, narrowed by the elongation of the rest of the body; or from want of due nutrition the body may be stunted. From some peculiar circumstance or occupation it may grow to one side, or the shoulder may droop, or the head may stoop. All these conditions do not indicate disease, but simply imperfections of the body, which demand attention.

Besides these imperfect conditions are others which are due to disease—of which there are five notable varieties. Of these, two are due to conditions of disease occurring during early life, when the bones are soft and yielding.

**Rickets**, a disease of childhood, results in the production of a pear-shaped chest with lateral incurvation, from the inability of the softened and yielding ribs to resist the pressure of the atmosphere when the diaphragm acts in inspiration. This is called the rickety chest.

**Pigeon breast** is the result of cough, catarrh, whooping-cough and enlarged tonsils in childhood. The breast-bone is unduly prominent, the ribs of the upper anterior part of the chest are flattened, and the lower ribs are driven inwards.

An illustration of this form of chest is given in the accompanying woodcut; and the transverse form of the chest showing its triangular shape on page 39.

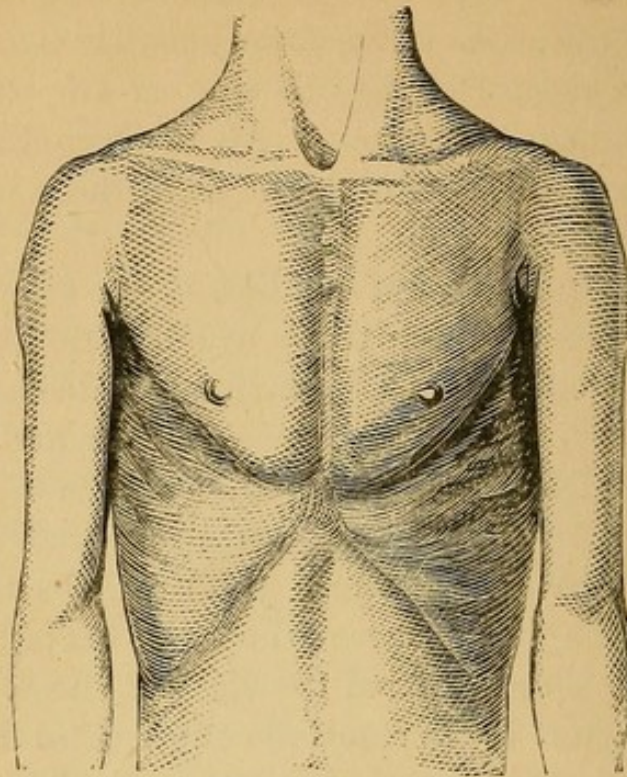
Deformities of the chest are also caused by spinal weakness and curvature, the contour of the chest corresponding to the curve of the spine. The shapes of the lungs within the chest are modified accordingly, and very often subject to emphysematous dilatations, from the excessive pressure to which portions are subjected in order to fill up the cavity of the thorax.

A transverse furrow which passes downwards from the end of the sternum across the anterior surface of the chest is due to imperfect expansion of the lungs during early life. It is sometimes called Harrison's sulcus, inasmuch as he proposed to make use of it with reference to the



position of the liver ; but, as Gee remarks, it only indicates what was the margin of the liver.

FIG. 5.



Pigeon Breast.

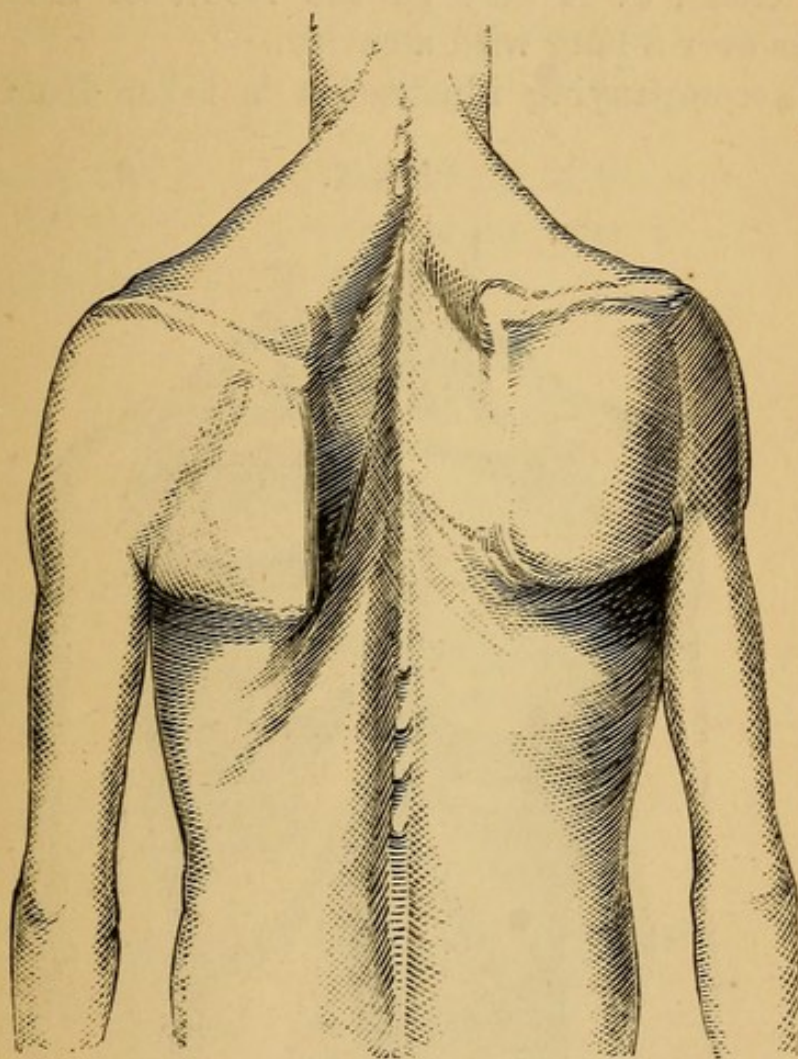
Besides these alterations of the chest, which are due to childhood's diseases and arrested or too rapid growth, there are certain forms which have been long recognised as connected with phthisis. They have hence been called phthinoid chests ; but it would be a grave error to suppose that they are structural malformations which necessarily precede or predispose to phthisis. There are certain forms of narrow and long chests which represent hereditary weakness ; but deformities of the chest are the result of the action of time—frequently the proof of previous disease ; and the alar and flat chests, which have been supposed to indicate a tendency to phthisis, are really caused by it, and only by a very chronic form of this disease. They may result from other conditions unconnected with phthisis, the alar chest being produced by atrophy of the muscles supporting the scapula and the shoulder-joint ; flat chests may arise from the weakness



or disease of early life. There is no form of chest which may not be attacked by phthisis, and the indication of a predisposition to phthisis is shown rather by the evidence of structural delicacy than by malformations; and frequently the subject of phthisis presents all the signs of symmetry and even beauty of figure.

The **Alar Chest** receives its name from the wing-like prominence of the shoulder-blades as seen from the back.

FIG. 6.



Alar Chest.

It is due to dropping of the shoulders, the glenoid cavity not being held in its proper position by the supporting muscles, the trapezius and pectoral: the spine of the scapula has an oblique direction downwards, the inferior angle being tilted upwards and outwards.



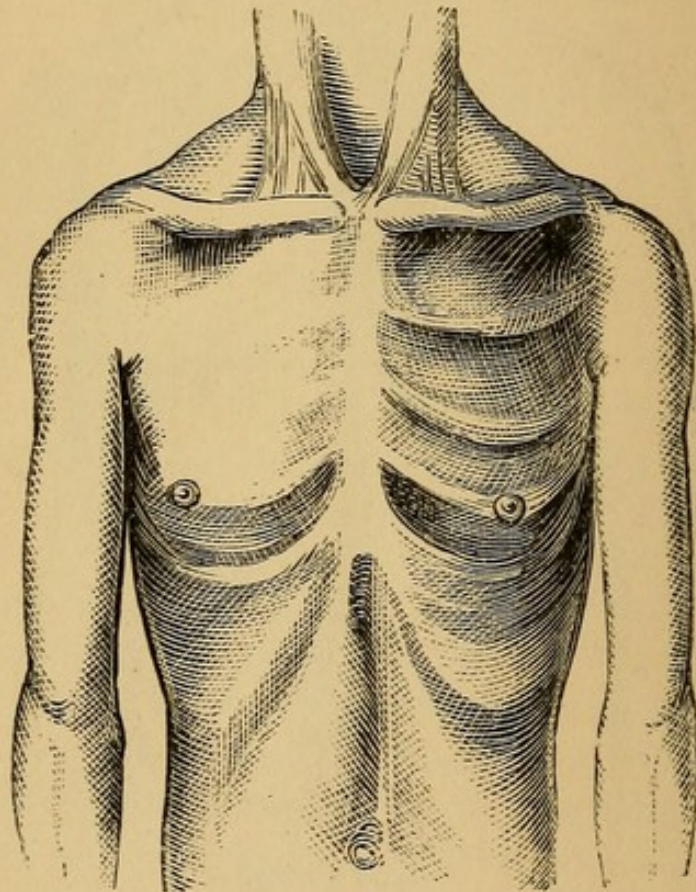
An illustration of this form of chest is given ; it is taken from a chronic case of phthisis of three years' duration, with adhesion of the pleuræ and a fibroid condition chiefly of the left side.

This **deformity** may be simply due to feebleness of muscle and wasting, or it may result from adhesive pleurisy with contraction of the lung, the result of chronic phthisis.

The **flat chest** is either the result of previous alteration of the chest, or it may be the result of atmospheric pressure over a lung with a cavity.

The accompanying illustration is taken from a very

FIG. 7.



Flat Chest.

marked case of flattening, chiefly of the left side, both sides being diseased. This was a case of arrested phthisis of two and half years' duration and the heart was very much pulled up towards the left shoulder.

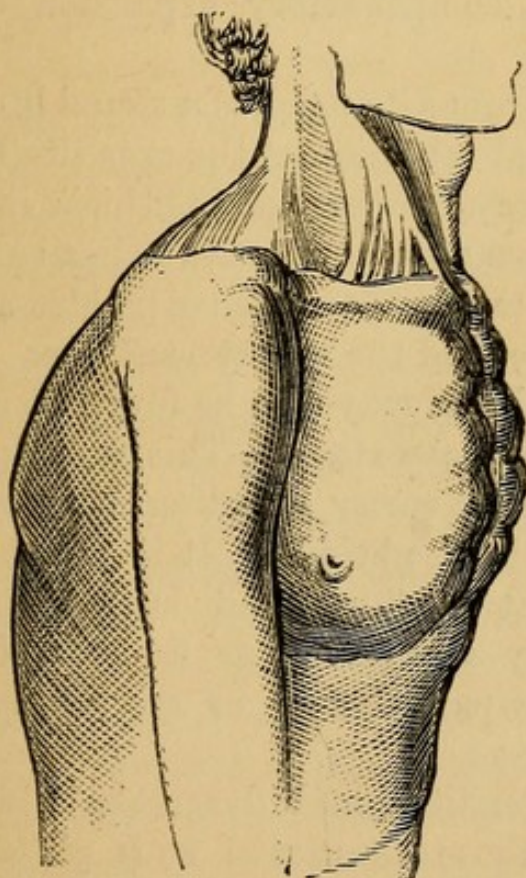


I have given on another page the transverse outline of this chest. (See page 39.)

Another form of chest of a very marked character is the circular, **barrel-shaped chest of Emphysema**, of which an illustration is given. The transverse form of the chest approaches very closely to a circle, the ribs being in a position of extreme distension; the manubrium sterni is thrown far forward, and the supra-sternal fossa, or the fonticulus gutturis, as it used to be called, is much hollowed; all the neck muscles are put on the stretch and the sternum is curved, the xiphoid cartilage being pulled in by the continued drag of the diaphragm and the pressure of the atmosphere.

The spine and ribs corresponding to the lower part of the thorax behind are often much curved. This is the bulging chest of emphysema.

FIG. 8.



Emphysematous Chest.

It may be remarked that whereas the chest in emphy-



sema assumes a form of extreme expiration, in chronic phthisis with pleuritic adhesions the chest takes the form due to extreme inspiration.

The following brief summary of the changes which take place in the form of the chest from disease may here be given:—

They are either due to **bulging** or **shrinking**; either of these may be general or local, bilateral or unilateral.

**Enlargement of both sides** is a common result of emphysema: it occurs occasionally with pneumothorax, with or without emphysema, the sound lung expanding to perform the function of the opposite one, crippled by disease, and in double pleurisy with effusion.

**Enlargement of one side** is usually the result of pleuritic effusion, from the presence of serous fluid, the result of acute or chronic pleurisy; of pus, or the presence of blood in the pleura, a very rare event; and of pneumothorax, without compensatory expansion of the sound lung.

It also arises from expansion of a sound lung consequent upon the crippled condition of the opposite lung. This is a very common result in arrested phthisis, in cirrhosis and fibroid contraction of the lung with adhesive pleurisy. The sound lung is sometimes so much expanded as to encroach several inches beyond the mid-sternal line.

Unilateral bulging may also be found in cancer.

**Contraction of both sides.**—This condition occurs from general adhesive pleurisy affecting both sides more or less, with or without phthisis. It is rare to find double pleurisy without phthisis, but such cases do occur, according to my experience, as a result of drunkard's dyscrasiæ, accompanied with or caused by cirrhosis or granular kidneys.

The three constitutional dyscrasiæ which encourage pleurisies are those associated with phthisis, drink, or syphilis.

**Contraction of one side.**—This is the ordinary result of all forms of pleurisy, with or without previous effusion.



It is the frequent concomitant of phthisis in all its forms. When the adhesions are firmly attached to the diaphragm the shoulder is pulled downwards, and the ribs slope obliquely towards each other.

This condition also occurs as the consequence of pulmonary collapse, especially in children.

**LOCAL ALTERATIONS.**—These alterations take the form of **bulgings** caused by pressure from within outwards, or depressions due to pressure from without inwards, from the force of the atmosphere.

**Local bulging** occurs from collections of fluid—serous, purulent, or bloody—from solid tumours, arising from scrofula, aneurism, lymphadenoma, cancer, syphilis; and from hypertrophy of the heart and the presence of fluid, serous or purulent, in the pericardium. Enlargements of the liver sometimes throw the lower ribs of the right side forward.

Local swellings may be caused by collections of fluid in cysts or abscesses in connection with some part of the chest-wall; sometimes, although rarely, in connection with fistular sinuses through the thoracic pleura. They may be also due to fatty cysts and to external cancer.

**Local depression** of the chest-wall results generally from the absorption of fluid after an ordinary pleuritic effusion, or as a result of the evacuation of pus from an empyema, either naturally through the lung and bronchial tube, an occasional termination, or artificially by tapping.

It is also a frequent result of a phthisical cavity: the more chronic the progress and the greater the amount of recovery the deeper will be the depression—proportionately to the size of the cavity. Local retraction takes place in a marked degree after profuse hæmoptysis. This is due to the contraction of the fibrin of blood which is inhaled by the gasps of the patient into certain parts of the lung. Local hollowing of the lower part of the sternum is artificially produced by the pressure exercised in various trades—as, for example, by shoemakers.

**Measurement of the Chest.**—For all practical pur-



poses a double tape measures the chest and the amount of expansion on each side accurately enough. The two tapes should be strongly connected behind, and the marking should be on the upper border of one and the lower border of the other. The junction of the tapes should be applied to the spine, and the tapes brought round in front to the mid-sternal line. It is convenient to mark this with ink or aniline pencil.

The stethometers which have been invented by the legion, are vain for practical use, although some of them have been found of great use for physiological experiments. Callipers are convenient for measuring cross distances.

The girth of the chest in the robust adult man varies, according to height and form, from thirty-three inches to forty-one inches.

Maclaren, in his work on "Physical Education," gives a table of averages for 100 boys of each age from ten to eighteen. At ten years the average girth of chest was found to be  $25\frac{1}{4}$  inches, with an additional inch for each year afterwards up to fifteen, when the chest grows larger; at

16	being	$32\frac{1}{2}$	inches,
17	„	$34\frac{1}{4}$	„
18	„	$35\frac{1}{4}$	„

He found the average of the first 100 names on the book of the Oxford Gymnasium, all at or under nineteen years of age, to be thirty-three inches.

**Graphic representation** of the chest is obtained by a very simple instrument, **the cyrtometer**, of which the cheapest form is that suggested by Dr. Gee—two pieces of composition gas-pipe drawn out to the diameter of the eighth of an inch, and united by india-rubber tubing.

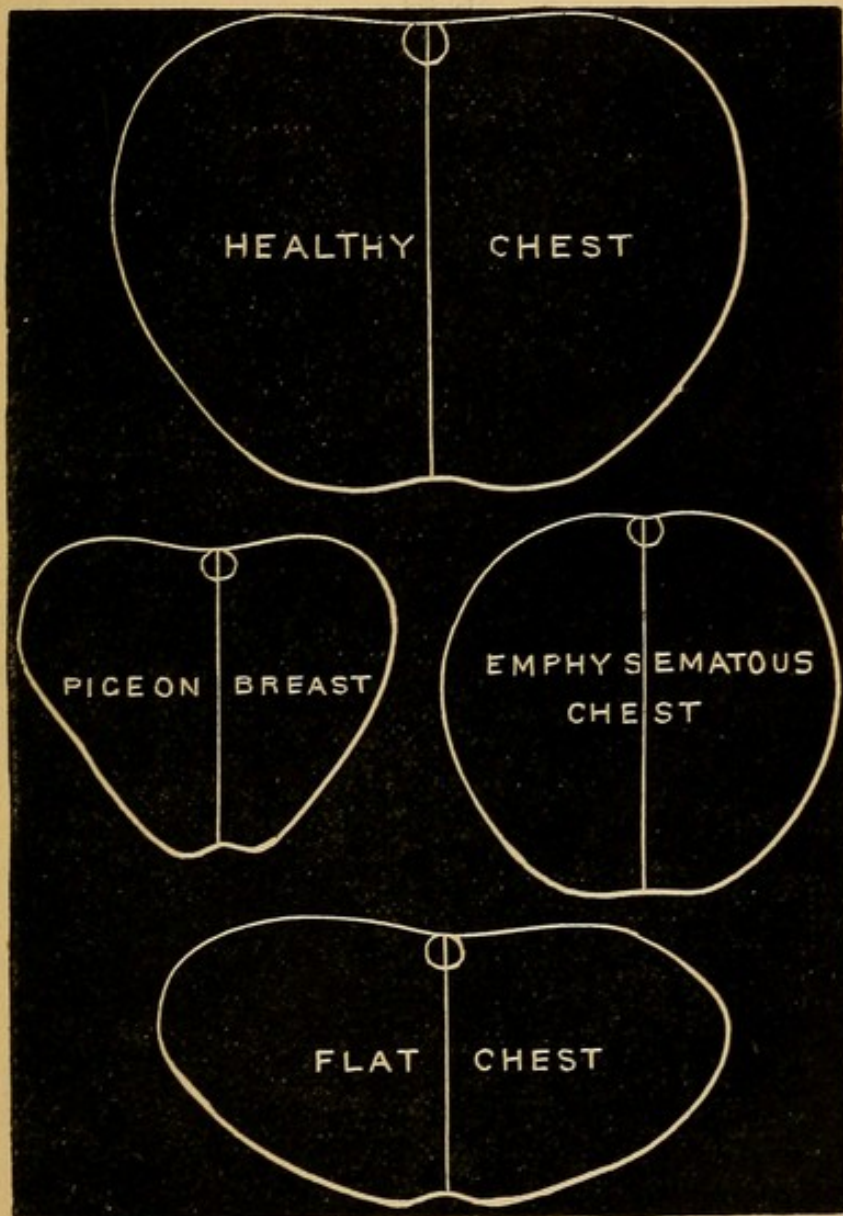
The joint is applied to the spine, and the pipes are accurately fitted round the chest. They are then removed and laid on paper, when an exact tracing may be made, giving the outline of a transverse section of the chest. The chests of phthisical patients are sometimes very tender, and the cyrtometer, when used for them, should be pliant.



The transverse outline of a well-formed healthy adult chest should be regular and symmetrical in form, the right side of the chest usually a little larger (a quarter to one inch) than the left side. In left-handed persons the left is the larger side. In the regular male type of chest the distance between the sternum and spine (**antero-posterior diameter**) should fall short of the diameter between the right and left sides (transverse diameter) by one-fourth.

The proportion of the antero-posterior diameter will be

FIG. 9.



Transverse outlines of different forms of Chest.

three; that of the transverse diameter will be four. In



the regular female type the proportion is as four to five. Much attention has been paid to the use of this instrument by Woillez in France, and by Dr. Gee in this country; and reference may be made to the works of these authors, and also to a thesis by Dr. Norman Moore on its use in detecting hypertrophy of the heart, for a full description with figures.

The chief practical advantage of the cyrtometer is obtained with reference to the diminution or increase of pleuritic effusion, and in such cases its application is of the greatest service in estimating the progress of the case.

All deviations from regularity of type should be carefully considered and accounted for, but as a rule it must be stated that the cyrtometer indicates changes that require time, and detects past conditions more readily than recent alterations; and I have been unable to convince myself that it surpasses in precision and nicety a well-trained eye, although it is very useful for the purposes of registering present states for future reference. Illustrations are given of the chief typical forms of transverse sections of the chest in conditions of disease taken from those chests of which drawings have already been given.



## CHAPTER VI.

## PALPATION.

EXAMINATION by touch is termed **Palpation**, and by it certain phenomena are detected which appeal to the sense of feeling but are hidden from the eye, and not revealed always to the ear.

The sense of touch is valuable for the purposes of diagnosis, and in the following ways:—By the tactile perception of—

1. The quality of matter.
2. Vibrations of sound through various media.
3. Vibrations of friction.
4. Fluctuation of fluid.
5. Impulse and thrills of the heart and vessels.

1. The quality of matter, as regards its hardness, softness, and resistance, has attributes which may be readily learnt from touch.

The healthy resilience of the lungs, especially as gauged in the intercostal spaces by the tips of the fingers, must be learnt by practice.

Any deviations from the normal condition as regards smoothness of surface and solidity of matter are soon detected.

Glandular enlargements, common in scrofulous patients, especially children, and in lymphoma, are discovered by pressing the thumbs or the ends of the fingers deep into the supra-clavicular spaces: hardness and irregular nodular swellings may be thus discovered, which give at once a clue to the case, although their nature may be open to doubt.

Resistance is also felt by the finger when it is percussed by the other hand, and this is a great advantage to be derived from mediate percussion.



2. Vibrations of sounds caused by the voice of the individual examined are transmitted to the palm of the hand placed on the surface of the chest, and the intensity of these vibrations is modified by circumstances.

This vibration, or **vocal fremitus**, does not correspond with vocal resonance. The perception of tactile sensibility not being so fine as that of hearing, voice sound may be loud, but its fremitus imperceptible. In great measure the fremitus is proportionate to the slowness of vibrations, and the lower the note of the voice the greater the fremitus. This is illustrated by a very simple experiment. If the hand be placed upon the flat sounding-board of a pianoforte, and the keys be struck in succession from the bass to the treble, it will be found that, although the intensity of the sound may be the same, the fremitus is diminished on going up the scale, and is entirely lost with the treble notes.

Fremitus is more manifest over the right lung than the left, chiefly in infra-clavicular, infra-scapular, and inter-scapular regions; very intense over the trachea; more marked when the patient is lying down, and in thin persons.

It is much diminished in the region of the liver and spleen; absent in the superficial cardiac region, where the heart is uncovered, and often absent in women, children, and fat people. The voice of a bass or contralto is more palpable than that of a tenor or soprano.

Vibrations may not be sufficiently rapid to produce a musical sound, and fremitus may be felt when the sound is inaudible. The vibrations of a string over a sounding-box may be felt long after the sound from the string has ceased to be audible, and the vibration of a big bell is very perceptible after its sound has ceased.

The passage of vibrations from one medium to another is attended with loss, the vibrations being reflected, and thus the passage of fremitus from lung tissue through a layer of fluid is much diminished. But this is by no means universally the case.

A large collection of fluid may be present in the pleural



cavity, and the fremitus may be intense. In proof of this I would cite a case which has long been imprinted on my memory. The physician, under whom the patient was, considered the case from these physical signs to be one of pneumonic consolidation. The patient died, and his right side was found crammed with serous fluid.

The phenomena of fremitus in the presence of fluid have received much attention, but the conditions which modify vibrations are so complex that the point is not yet decided; and I would specially direct the attention of all observers to the careful investigation of the subject.

I have not been able to substantiate the correctness of the notion which supposes fremitus to depend upon the character of the fluid, diminished fremitus being attributed to pus.

The pressure of the lung, consolidated or compressed, against the walls of the chest, conducts the vibrations of the voice to the thorax, and fremitus is not often completely annihilated, except in women or children.

Solidification of the lung intensifies vibration, but this condition could not be established by this sign alone.

Aneurisms and tumours that come to the surface of the chest annul fremitus, but it is increased if the lung be compressed between the chest-wall and the tumour.

3. Vibrations due to friction may be felt in pleurisy, pericarditis, peritonitis, and in loud rattling rhonchi. These latter are very perceptible in children suffering from fluid in the tubes, the result of catarrh or teething, and in the moribund, from feeble power of expectoration; and it is very necessary to bear in mind that as the infant and child cannot expectorate, any secretion into the lungs is apt to remain there for some time.

The vibrations from splash in pneumothorax may also be felt, and in one remarkable case I remember to have felt the splash more distinctly than it was indicated to the ear by tinkle.

4. Fluctuations of fluid may be felt when such a collection is tapped with one finger and the waves of impulse are transmitted to the fingers of the other hand



placed at some little distance off in an intercostal space.

Pulsations of vessels or of cardiac impulse are transmitted through fluid, sometimes simulating aneurism. This may occur in effusions into the pleura and pericardium (serous or purulent).

5. Impulses and thrills of the heart and vessels are very important signs in the detection of diseases of these organs.

In all cases of thoracic disease, one of the first points to engage the attention (after a full inspection) is the position and character of the impulse of the heart. In weak hearts this is sometimes felt and not seen. In nervous excitement the impulse is rapid and sharp; in diseases of the heart, with hypertrophy, the impulse is prolonged and heaving; in dilatation, its area is generally increased.

The impulse of the heart varies slightly with the position assumed; in the supine it is diminished, by decubitus on the right and left sides it inclines over to the same side. Alteration of the position of the impulse, due to dislocation of the heart, results from the presence of fluid in the pleura, in the pericardium, from empyema, aneurism, and mediastinal tumour, and very commonly from adhesive pleurisy, with phthisis or fibroid pulmonary disease. Local impulses not connected with the heart itself may be due to aneurisms, or collections of fluid and tumours near a pulsating vessel.

**Thrills**, or vibrations resembling those produced by the purring of a cat, and hence called "*fremissement cataire*," are caused by the rippling current of blood passing from a narrowed orifice into a dilated cavity; and hence these vibrations are felt in roughened valves and dilated ventricular and auricular cavities, and in cases of aneurism.

In the consideration of the indications afforded by palpation as regards impulses and thrills, note should be taken of the position, the extent of the impulse, its regularity and occurrence with reference to the action of the heart, and the presence or absence of thrill.



## CHAPTER VII.

## THE CLINICAL ACOUSTICS OF PERCUSSION.

HITHERTO the methods of examination under consideration have been concerned only with the eye and the touch, needing no scientific knowledge to understand the laws to which the observed phenomena are subject. But in examination by the ear some knowledge of acoustics is necessary before we can understand what sounds mean and why they are altered. I have no intention of entering upon an abstruse discussion of the laws of sound, or I should probably find myself landed in some of those difficulties and dilemmas which those who have gone before me have not altogether succeeded in escaping; and I shall only attempt a brief account of the characteristics of sound and its modifications, with a few practical illustrations.

Sounds of all kinds are produced by a succession of shocks received on the membrane of the ear, and translated by the auditory nerves to the brain. Noises are due to the jarring of such shocks hitting the tympanum in irregular succession, a disagreeable and sometimes painful impression being thereby produced; just as the irregular flickering of a dying lamp produces a disagreeable impression on the sight. When the shocks are regular and sufficiently rapid in vibration a more pleasing effect results, and this regularity constitutes the condition necessary for musical sounds.

For present purposes, the verbal description of sound, it is necessary to remember that sounds differ to individual ears as colours do to individual eyes; and, as many persons are colour-blind, so many are deaf to musical



sounds; and there is always great difficulty in conveying a notion of a particular sound by words. In giving a description of a particular colour we have to make a comparison with some well-known standard colour to which similarity may be shown by carefully chosen epithets; and in a similar manner sounds must be described by similitude with some well-known standard sounds. The comparison must be made with regard to certain attributes or characters of sounds, which may be here limited to three:—

1. **Intensity.**      2. **Pitch.**      3. **Quality.**

1. The **intensity** of a sound is readily appreciated and indicated by epithets representing its degree of loudness or softness; a sound may be of great, moderate, or feeble intensity, according as it is loud, moderately loud, or soft.

2. **Pitch** requires more delicacy of appreciation, and in some measure the refinement of a musical ear. Pitch is proportionate to the number of vibrations by which a sound is produced; the greater the number of vibrations in a certain time the higher the pitch, the fewer the vibrations the lower the pitch.

Attention should be particularly directed to this attribute of sound, as it is of the greatest value in diagnosis.

3. **Quality** indicates peculiar characteristics of sound apart from pitch and intensity. The sounds of a violin and a horn have different and peculiar qualities of such a kind as to enable an experienced ear to recognise at once from which instrument such a sound has proceeded. The description of quality is one of the greatest difficulty, and we have to resort to well-known sounds of ordinary life for comparisons, making use of such epithets as cooing, bubbling, whistling, snoring, and the like.

Other characteristics of sound—namely, duration and continuity, or interruption—indicate peculiarities in the production of those sounds, to which future reference will be made.



The use of percussion in eliciting knowledge of the solidity or hollowness of substances is known in ordinary life from the custom of the carpenter when he wishes to learn the position of a solid joint which will take a nail ; he taps the wall with his hammer, and finds out by the sound what he wants.

The resonance derived by percussion from matter is dependent upon molecular structure, elasticity, density, and the presence and contiguity of air ; the immediate percussion of solid substances results in eliciting sounds more or less resonant, according to their vibrating properties.

Lax tissue without air—as, for example, the liver, the heart, the kidneys, and spleen—yield to percussion a non-resonant sound which is termed “dull.” Dulness of percussion means absence of resonance. It is sometimes called flatness.

Resonance, on the other hand, is elicited from vibrating walls enclosing more or less air. This is the condition which obtains in the chest, the walls of the chest which encircle the air-containing organs (the lungs) vibrating under percussion, and yielding a comparatively resonant sound.

The sound elicited by percussion from the chest-walls, with healthy lung beneath them, being the standard sound of health, all other sounds, whether resonant or non-resonant, are compared with this **normal resonance** ; sounds that have more resonance are called hyper-resonant ; those that have less, sub-resonant.

But even with a healthy state of lung, the sound elicited by percussion varies under certain conditions of the lung, and some explanation is demanded upon this point.

The pulmonary resonance is derived from a number of little sacs containing air, and the exact sound elicited from them depends upon the tension of these sacs. A drum, which is an air-containing instrument, with a top and bottom formed of membrane, can have its note altered



at pleasure by tightening or loosening the membrane—the tighter the parchment the higher the pitch. The same result would follow putting the drum before the fire and expanding the air. In the same manner, the air-sacs of the lung, the more they are expanded the higher the pitch of their note of percussion, the shorter their vibrations, until all pitch is lost, and the note of percussion becomes hard, wooden, and closely approximating to dull.

A simple illustration of these points may be obtained by filliping the cheek with the finger. With the mouth open and the muscle relaxed, a dull flapping sound of non-resonance is heard on percussion, to which, however, a certain pitch may be given by stretching the muscle by opening the jaws widely. We have here an instance of muscle when contracted giving a higher pitched sound than when relaxed. The same result is obtained by percussing the thigh when relaxed and when firmly flexed.

If the mouth be closed and the cheeks inflated, a distinct tone is produced as soon as a certain tension of the muscle is thus obtained. This tone, which is termed **tympanitic**, from its resemblance to the note of a drum, can be altered in pitch by increasing the tension of inflation. When the cheeks are puffed out very tensely indeed the note gives way to a dull, hard, wooden or muffled sound, which closely resembles the non-resonant note of liver or muscle. From these simple experiments it is evident that, with elastic membranes (in order to obtain a tone) a definite tension must first be obtained, the *lax* membrane giving no distinct note. When this point is arrived at, further tension raises the pitch of the note until the vibrations fail to be appreciated by the ear, and a muffled or non-resonant sound is then produced.

It is easy to show that this is true also of the percussion note of the lungs. At the beginning of inspiration percussion elicits a comparatively low-pitched sound. The pitch of this is notably raised at the end of inspiration,



and still more so after a forced inspiration. The student should assure himself of the truth of these observations by percussing the healthy chest in different degrees of breathing.

The same rule holds good with regard to large quantities of air beneath the ribs, as in pneumothorax. The ribs may be regarded as so many vibrating rods enclosing air. If free vibration be allowed—that is, if the pressure of the air be not excessive—a marked resonance of percussion is obtained, which, if it attain to the dignity of tone, will be called tympanitic. If the tension of the air be very great (and this occurs in cases where the opening from the lung into the pleura is small, indirect, and easily closed on expiration), then the vibrations of the ribs may be so interfered with that the tympanitic sound, which rises in pitch as the air-pressure increases, becomes harder and more wooden, and yields at length a non-resonant sound. I have already observed that the ribs are to be considered as the vibrating walls of an air-containing cavity. Now the larger the space which is occupied by air within the thorax the more nearly the chest approaches to the condition of a barrel or wine-jar, and in such a condition percussion gives rise to a reverberating or echoing sound resembling that obtained from striking an empty cask—hence called amphoric.

The **amphoric sound** is imitated by pursing the lips and filliping the cheek, when a noise resembling fluid pouring out of a wine-bottle is produced. The amphoric sound is due to reverberation in a large hollow body with a narrowed opening. If the sides of the resounding-box are very rigid, if there be fluid present at the base of the box, or if the cavity be struck by ringing metal—as, for example, with a pliable metal hammer—a metallic clank is superadded, which appears to be due to the addition of a high-pitched overtone occurring at a very short interval of time afterwards.

I have thus very briefly indicated some modifications



in the sounds of percussion which are technically called—

Resonant.	Sub-resonant.
Hyper-resonant.	Muffled.
Tympanitic.	Dull.
Amphoric.	
Metallic.	

The first column indicates the terms applied to percussion sounds in order of resonance; the second, in order of dulness.

It remains to mention one sound of dubious import—the cracked-pot sound, or *bruit de pot félé*. It is produced by a sharp and hard percussion, by which air is driven out through a small opening, forming a little explosion, and it will be more fully explained in another place, where its application to the detection of disease will be discussed.



## CHAPTER VIII.

## PERCUSSION.

PERCUSSION is the art of eliciting sound by striking either with the fingers or with instruments especially adapted for the purpose, and enables the examiner to estimate certain qualities, having special reference to resonance or non-resonance, of the part struck.

Percussion may be **immediate** when the striking fingers or hammer are applied directly to the part examined, or it may be **mediate** when something is interposed to receive the force of the blow—the fingers of the other hand or a small piece of wood or ivory.

No other instruments are required for the purpose by the ordinary observer but the fingers, and it is only for the purposes of class demonstration that a pleximeter should be used; the less show there is on the part of the examiner, especially in the exhibition of instruments, the more likely the patient is to undergo with equanimity what is to most a sufficiently trying ordeal. Moreover, the use of instruments prevents that appreciation of resistance which is an important item of the knowledge obtained from manual percussion.

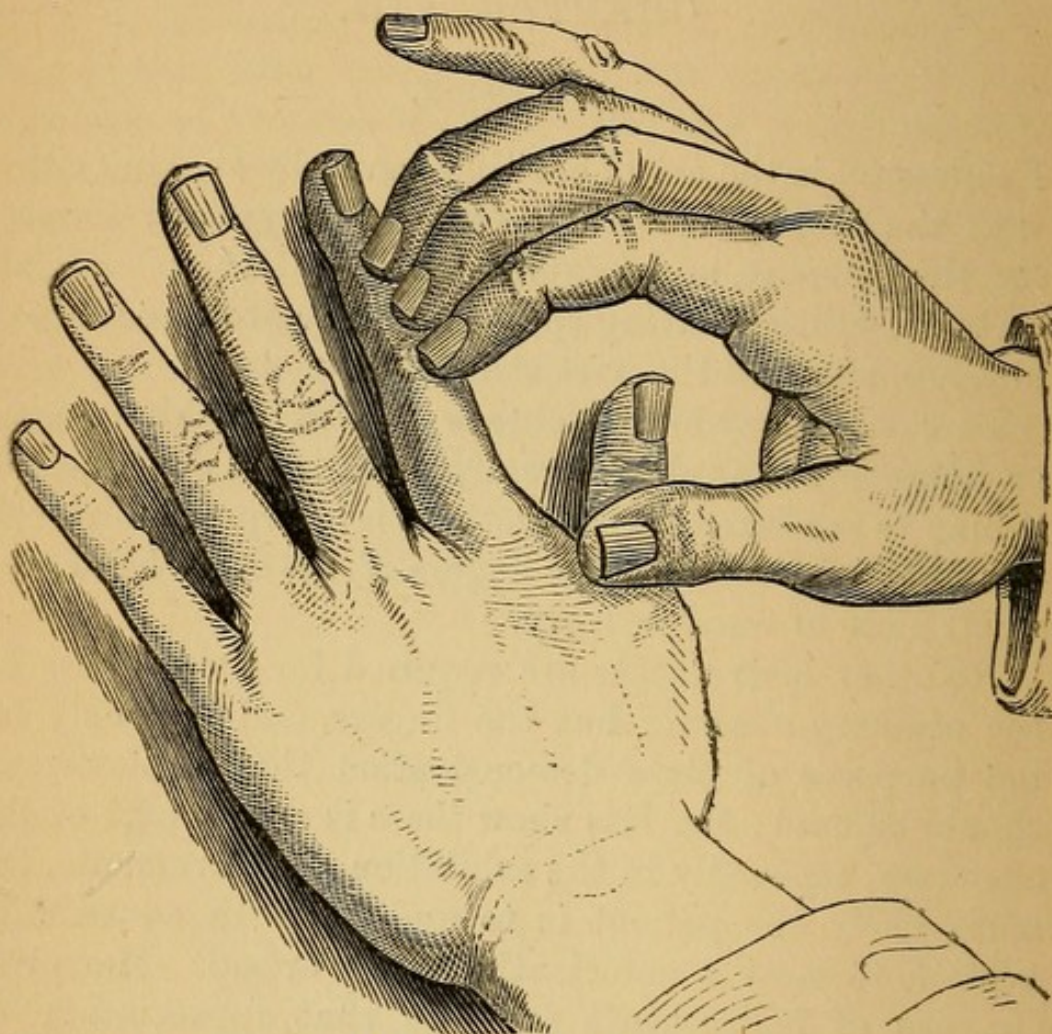
**Mode of Percussing.**—The art of percussing properly is not easily attained, and demands considerable practice before the skilled knack is acquired. It in some measure resembles the art of pianoforte playing, and there are few performers who can be said to have a really good touch.

For the best manner of mediate percussion, one finger, the forefinger of the left hand, should be placed with the palmar surface on the chest at the part to be percussed,



the other fingers being elevated clear of the chest surface (as in the accompanying illustration), and the blow should be struck by the first three fingers of the right hand closely held together without being touched by the thumb

FIG. 10.



Hands in Percussion.

and little finger. The fingers should be so bent (all the phalangeal joints sharing in the bend) that the last joint may strike vertically upon the finger of the left hand. The blow must be given with the ends and not the pulps of the fingers, and solely from the wrist, the forearm not coming into play. The blow ought to be smart and short, the percussing fingers being instantly removed; like the action of the pianist when playing an octave passage staccato, or of a pianoforte hammer on its wire.

The proper art of percussion can only be acquired by



long practice, and the muscles of the wrist require to be specially trained and strengthened for the purpose.

It is sometimes convenient in percussing a small spot to use only the second or the third finger for delivering the blow, and some percussers never use the first finger at all.

Percussion may be made in two ways: with a slight blow, thus eliciting vibration from the immediately subjacent parts—this is termed **superficial percussion**; or with a strong heavy blow, eliciting sounds connected with deeper-seated regions—this is called **deep percussion**.

The first study which should engage the attention of the student is the standard sound of percussion, which indicates a healthy and natural condition of the chest and its organs; and to this sound, upon which the practitioner is so often called to express an opinion, much time may be devoted with abundant profit.

For this purpose a healthy man should be selected, of adult age, with not much muscular development—the thinner the chest the better, as long as the man is in a condition of health—and the percussion note of his chest examined.

Percussion of a healthy chest yields a slightly different note in different regions of the chest and in different individuals, according to the external conditions of the chest and some other circumstances; and for the determination of a condition of health the best result is obtained from comparing the sounds obtained in different regions of the chest; but it is of paramount importance that some idea of the approximate note of health, and the range of sound which is consonant with a condition of health, should be retained in the memory.

Fortunately for the physical examiner, the concert pitch of the lungs is seldom everywhere obliterated, and there is generally some region where the true pulmonary resonance can be obtained.

Those parts of the chest will of course yield the greatest resonance in which there is the least quantity of material



interposed between the fingers of the percussor and the lung.

The anterior part of the chest yields a more resonant sound than the posterior, partly because the muscles and walls are not so thick, and partly because the front portion of the lungs is the most expansile.

The standard pulmonary resonance may be obtained by percussing the infra-clavicular region, between the clavicle and the third rib, and the upper axillary regions.

There are two advantages incidentally belonging to the first region as the source of the typical pulmonary sound, to which all other percussion sounds are to be referred; one is, that this region is easily examined by simply loosing the upper part of the patient's dress; and the other is, that this region is first affected in many forms of pulmonary disease.

Besides the note elicited from percussing over pulmonary tissue there is another normal or healthy resonance, of a peculiar hollow hyper-resonant quality, obtained by percussing over the trachea and large tubes, hence the name **tracheal** is applied to it. The regions which yield this sound to percussion are given in the next chapter.



## CHAPTER IX.

## PERCUSSION IN HEALTH.

THE percussion note of the healthy lung must be considered with regard to those three qualities to which attention has already been directed—viz.:

1. **Intensity.**            2. **Pitch.**            3. **Quality.**

1. Its **intensity** will depend in some measure upon the blow of percussion, and upon the thickness of the tissues which overlie the ribs and intervene between the lungs and the percussing fingers.

It is diminished by thick layers of muscle, of fat, and by rigidity of the costal cartilages.

It is increased by expansion of the lung, by elasticity of the cartilages, and absence of fat and muscle.

The average intensity must be described as moderately loud.

2. **Pitch.**—This must be considered as low in note, inasmuch as almost all others are higher.

3. **Quality.**—This can only be learnt by practice. It is very peculiar and characteristic of elastic or spongy tissue: hence it has received the epithet **vesicular**.

In describing healthy normal resonance, we may say that the intensity of the sound varies according to individual conditions, that the pitch is low and its quality is vesicular. The exact appreciation of what is meant by these words can only be obtained by clinical experience.

The student should devote some time to the percussion of healthy chests, in order to acquaint himself with the peculiar characters of the pulmonary resonance.

After this has been learnt, he may study the variations



which are to be found in the different regions of the thorax, consonant with a condition of health.

### REGIONS OF THE ANTERIOR SURFACE OF CHEST.

**Supra-clavicular.**—Vesicular quality, marked above the clavicles; it becomes tracheal in quality near the sternum, the result of percussing over the tube.

The height of the apex is an important object for percussion, which in a condition of health extends a distance of three finger-breadths above the clavicle; the right being a little higher than the left, and in females higher than in males.

**Clavicular.**—When percussion is made upon the bone the note is altered, and becomes short and ringing. Near the sternum it becomes tympanitic in resonance, because of the proximity of the trachea; near the humerus the resonance is lost.

**Infra-clavicular.**—Vesicular resonance well marked, particularly on percussing in the intercostal spaces. Near the sternum the trachea imparts its peculiar resonance; this is also evident between the second and third ribs in the second intercostal space, under which space the large bronchi lie, the left being a little lower than the right.

**Mammary.**—Resonance not so vesicular, on account of the intervention of muscle.

There is a difference on the right and left sides.

The position of the liver and heart must be studied, inasmuch as these organs modify the percussion note.

**Right side.**—From the fourth rib to the sixth the vesicular resonance is gradually diminished, becoming more dull until at the sixth rib the dull note due to the liver is obtained.

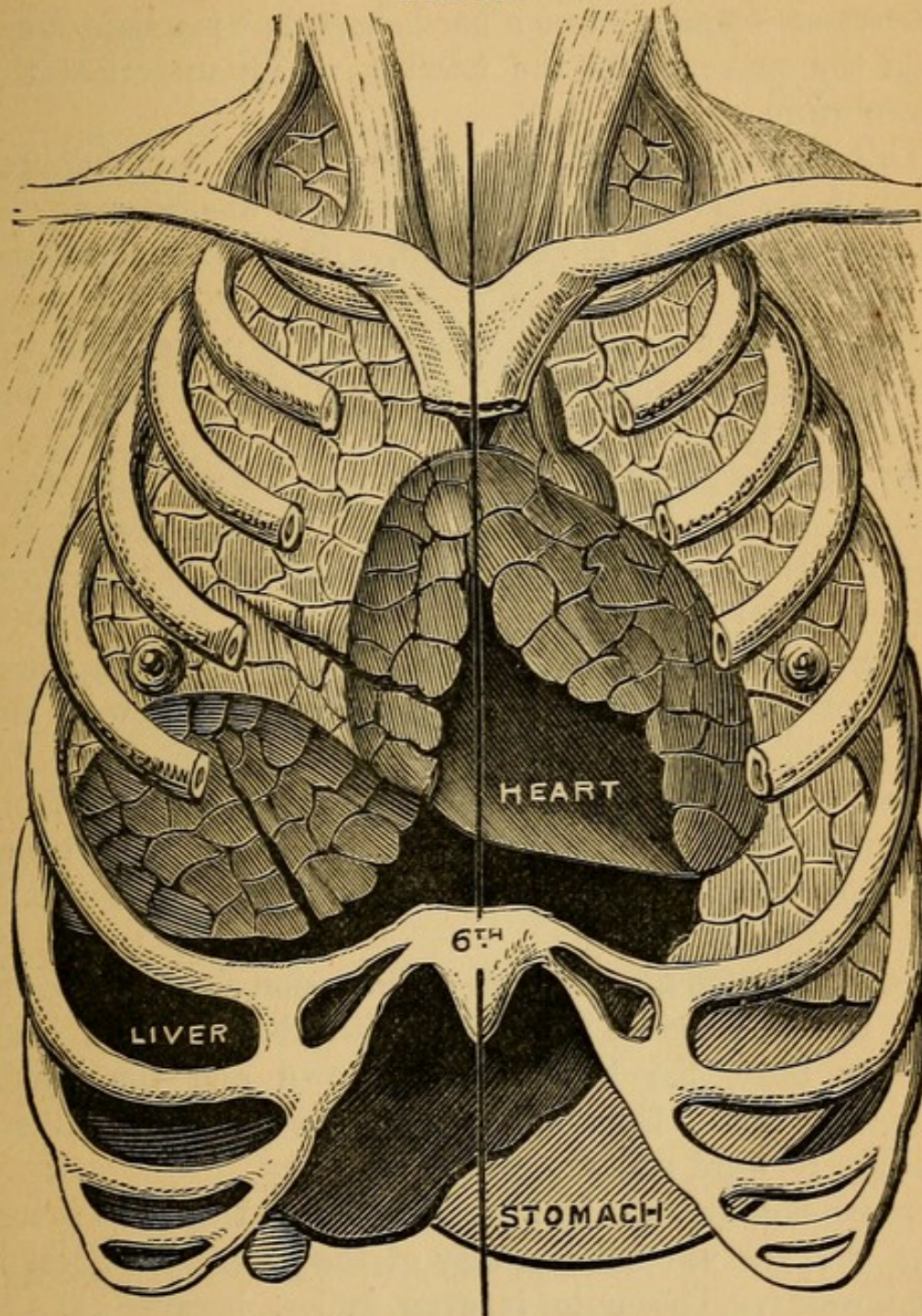
Between the third and fifth ribs, close to the sternum, the position of the right auricle and ventricle of the heart give a dull note.

**Left side.**—Resonance diminished and dulled, from the



presence of the right and left ventricles of the heart, from the third rib to the fifth intercostal space. On the outer portion of this region the stomach increases the resonance of percussion, and a tympanitic note is elicited.

FIG. 11.



Front Thorax.

**Infra-mammary.**—In this region there is again a difference on the two sides, owing to the positions of organs.



**Right side.**—Dull percussion, due to the subjacent liver.

**Left side.**—Inner half dull, owing to the presence of the liver; outer half tympanitic, from the presence of the stomach.

**Sternal.**—**Upper** gives a hard short, but resonant note, from the presence of the trachea, which underlies the manubrium.

This is an important region for percussion in enlargement of the glands, and the presence of aneurism of the transverse portion of the arch, and some mediastinal tumours.

**Lower** has a dull note, owing to the presence of the heart. This extends from below the second rib to the xiphoid cartilage.

**AXILLARY.**—**Upper** of both sides very resonant, with a good vesicular quality; the lung lying close beneath the ribs and being very expansile here. This is an important region for percussion.

**Lower.**—**Right side** yields a dull note from the presence of the liver, which is marked on a level with the eighth rib.

**Left side.**—Anterior portion generally tympanitic from the presence of the stomach, the posterior portion being dull from the position of the spleen between the ninth and eleventh ribs.

**POSTERIOR SURFACE.**—Percussion should be made over this surface of the chest with the patient's back slightly bent, the head looking down and the hands clasped in front: the muscles should be lax, the shoulders brought forward and dropped.

**Supra-scapular.**—The presence of the trapezius interferes with the vesicular quality of the percussion note.

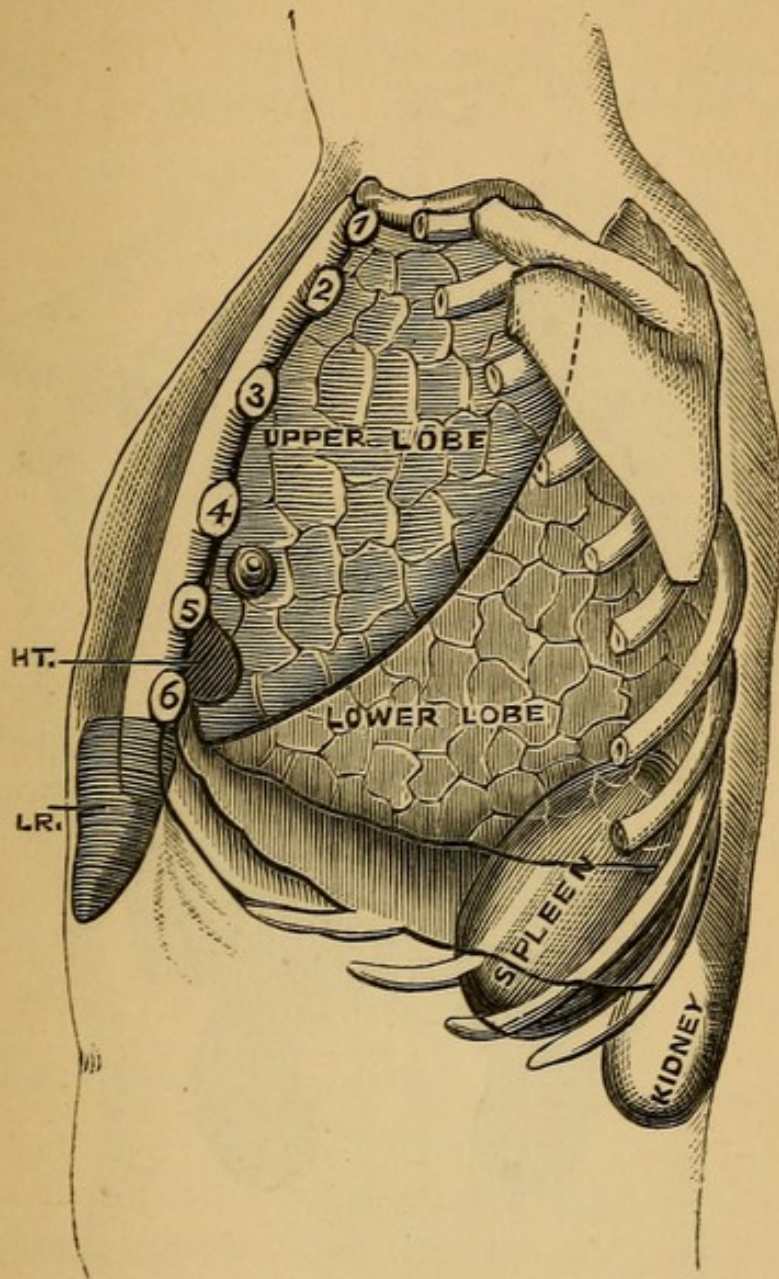
**Scapular.**—The note is even duller, because of the interposition of the shoulder-blade and its muscles.

**Inter-scapular.**—The note is more resonant, and becomes tracheal in quality half-way down between the base of the scapular spine and the inferior angle, on



account of the position of the large bronchi which bifurcate to the lungs (between the fourth and fifth dorsal vertebræ).

FIG. 12.



Side View of Thorax (Left).

**Right.**—Tracheal resonance marked.

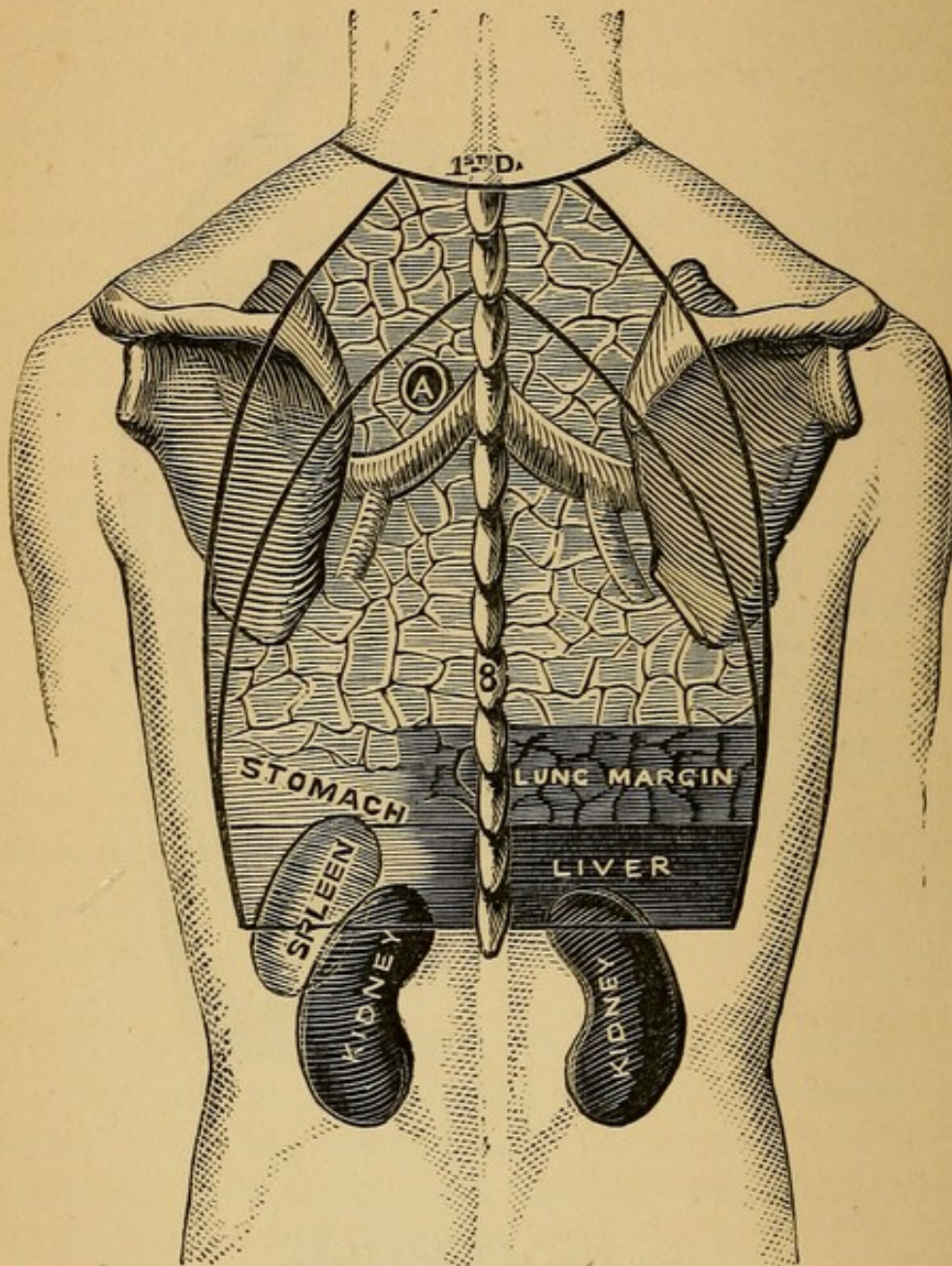
**Left.**—The note is duller on account of the aorta, which arches over the bronchus.

**Infra-scapular—Right side.**—Resonance intense and vesicular as far as the eighth rib, where the liver dullness is evident, becoming very marked at the tenth rib.



**Left side.**—The dulness due to the liver is only evident near the spinal column on this side, and soon gives way to tympanitic resonance due to the stomach. Near the

FIG. 13.



Back View of Thorax.

axilla the presence of the spleen is indicated by dulness of percussion between the ninth and eleventh ribs.



*Synopsis of Percussion Sounds in Health.*

Vesicular Sound .....	Resonant .....	Infra-clavicular .....	Right and Left.
		Clavicular .....	” ”
		Supra-clavicular .....	” ”
Sub-resonant.....	Diminished .....	Upper Axillary.....	” ”
		Infra-scapular .....	to eighth Rib.
		Mammary .....	Right.
Tracheal Resonance .....	Tympanitic Resonance .....	Supra-scapular.....	Right and Left.
		Scapular.....	” ”
		Inter-scapular .....	Left.
Dull.....	Dull.....	Mammary .....	Left.
		Lower Axillary.....	Right.
		Infra-scapular .....	Right and Left.
Dull.....	Dull.....	Infra-mammary.....	Right.
		Upper Sternal.	
		Infra-clavicular.....	near Sternum.
Dull.....	Dull.....	Inter-scapular .....	Right.
		Lower Axillary.....	Left.
		Infra-mammary.....	Left.
Dull.....	Dull.....	Infra-mammary ....	} Right, over Liver.
		Lower Axillary.....	
		Infra-scapular .....	{ Right and part of Left, over Spleen.

**Thymus Gland.**—It must be pointed out that in childhood the thymus overlies the lungs in front, under the upper part of the sternum, disappearing at puberty. It reaches its maximum size at the end of the second year, and measures two inches in length, and one and a half inches in width. It extends a little above the sternal notch, and descends as low as the fourth cartilage.

**Alterations in the Percussion Note from Respiratory Movements.**

The amount of air taken into the lungs varies according to the force of inspiration. In ordinary quiet respiration, when the chief if not the only movement is effected by the diaphragm, the amount of air is comparatively small, but with a full inspiration, in which action the muscles of the thorax participate with the diaphragm in expanding the chest-wall, the quantity of air is much increased, and the air-sacs are fully dilated; the cavity of the chest is also increased in size. Here there are two conditions which alter the sound of the percussion note: the increased



capacity of the chest gives to deep percussion a fuller, more intense note; but a less strong percussion will elicit, especially at the end of inspiration and at the beginning of expiration, a higher pitched and a more muffled note. The muffling of the note is due to the tension of the walls of the air-sacs, and the same result may be obtained by inflating a lung out of the body—a sheep's lung may be readily obtained for the purpose—and inserting the nozzle of a bellows into the bronchus: the more the lung is inflated the more muffled the note of percussion becomes, and the higher the pitch.

The difference between the percussion notes in ordinary and forced respiration is more marked on the right side than on the left.

It is important that the student should make himself acquainted with this difference, which involves a little attention and some delicacy of ear: the peculiar muffled note which occurs on percussing a distended lung is of great importance in diagnosis, as will be shown in another page.

Besides the variations in sound just mentioned are others due to the alteration of place which the organs below the diaphragm assume under the movements of the diaphragm.

The liver moves down during inspiration, and returns again to its first position during expiration. Under a forced inspiration there is a movement downwards of an inch and a half.

Forced expiration gives a rise of two and a half inches to five and a half inches, so that between the furthest limits is an interval of from **four to seven** inches.

Behind, the line of dulness is lowered from **one to two** inches by deep inspiration.



## CHAPTER X.

## PERCUSSION IN DISEASE.

THE normal sounds of a healthy lung having been studied, the student may turn his attention to the investigation of other sounds derived from percussion in disease. The variations that occur in vesicular resonance, putting aside for the present those in which dulness of any degree participates, are chiefly noteworthy for alterations in pitch, the pitch being always raised. Attention has already been directed to the difference in pitch between the pulmonary resonance from a lung before inspiration and after deep forced inspiration, the pitch being invariably higher. From what has been said in the chapter on the Acoustics of Percussion it is evident that by subsequent degrees of distension the pitch becomes higher and higher, until the vibrations are not appreciated by the ear, and a tense muffled sound is elicited.

A high-pitched percussion note is obtained not only in an artificial expansion, as in forced inspiration, but it is also to be found in that condition in which one lung has to do the work of the other which is crippled by disease, the sound lung taking up what is termed **compensatory breathing**.

For this purpose, not only are the air-sacs fully expanded, giving rise to a high-pitched percussion note, but the lung itself expands in volume to meet the call which is made upon it.

It is necessary to percuss, therefore, across the mid-sternal line, as one lung will sometimes encroach very far—as much as four inches—across the line, and invade the territory of its fellow lung.



This usually happens in greatest extent when the left lung is diseased with phthisis or fibrosis, the heart being drawn out of place and dislocated.

In such a case, although the pitch of the percussion note is raised, the vesicular resonance is marked and its intensity increased, on account of the increased size in the resounding-box of the chest, to which some additional effect is probably given by the expanded condition of the bronchial tubes.

High-pitched resonance shows that there is a considerable strain on the portion of lung percussed—not a good sign, inasmuch as it indicates an extent of disease elsewhere.

But the distension of the lung may proceed further, as in the case of complete or partial loss of elasticity, leading to excessive dilatation of the air-sacs. This is **emphysema**, and the true note of this emphysematous distension when excessive is not the hyper-resonant note, which is given in all books, but a decided muffled, non-elastic, or hard sound, which I consider to be peculiarly characteristic. This note can be obtained by superficial percussion only, and is generally overlooked because the examiner prefers to elicit the noisy resonance which an emphysematous chest delivers when it is percussed with a full blow.

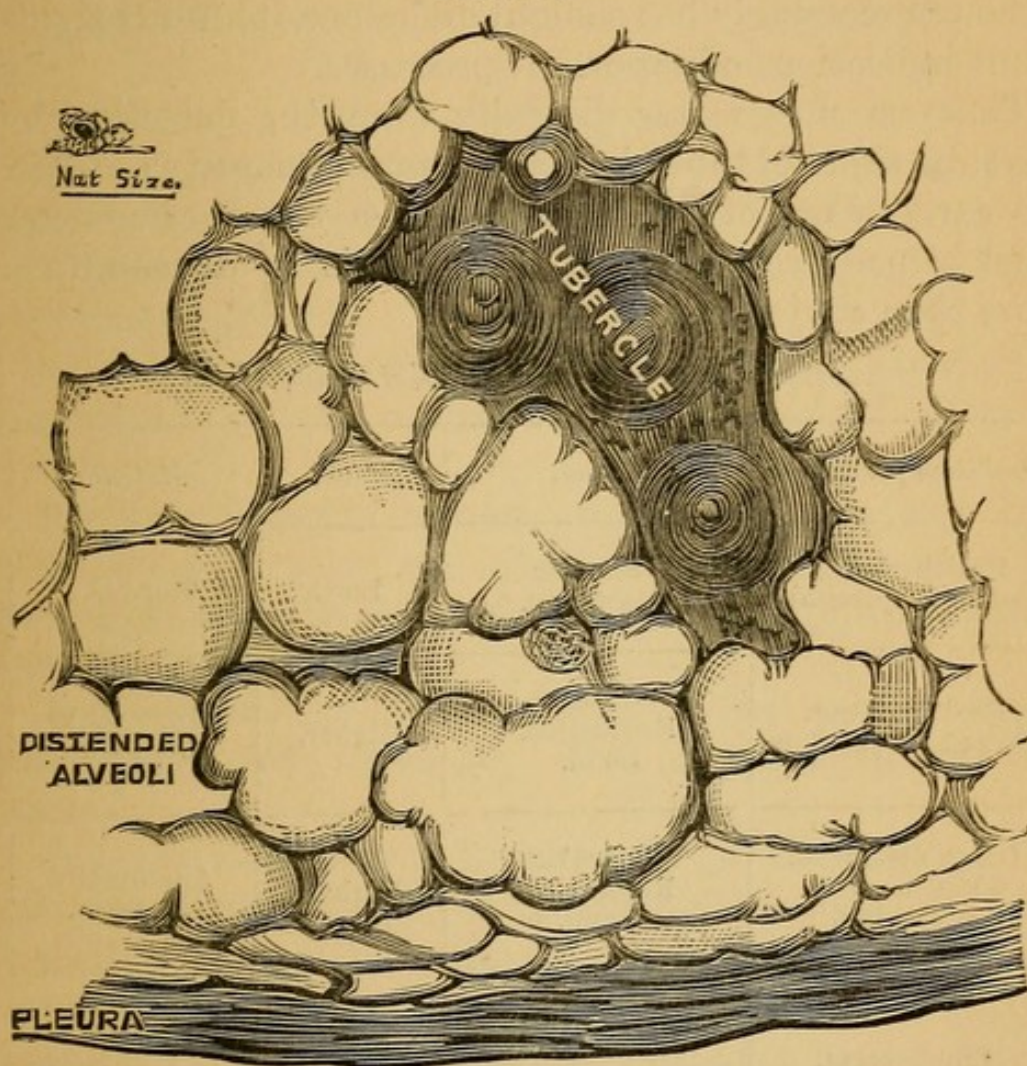
The student is very apt to think that no one can mistake a case of emphysema, and instances of the disease occur which can readily be detected at a glance; but after much experience of the errors of others, from my position as pathologist, I must state positively that there is no condition for which other diseases are more frequently mistaken than emphysema; and when students report a fatal case as one of emphysema, I look to find acute tuberculosis, a condition which resembles emphysema very closely.

This brings me to the consideration of the percussion note in **tuberculosis**. The invasion of a lung by an out-crop of miliary tubercle necessitates extreme distension



of the collateral tissue, and hence the external surface of a tuberculous lung removed from the body has a similar appearance to an emphysematous one, being pale, grey, and much distended. Even when it is cut into the distension of the air-sacs is very apparent, and the microscope shows distinctly how much this condition prevails. In acute

FIG. 14.



#### Miliary Tubercle.

tuberculosis, therefore, percussion elicits a short high-pitched muffled sound from the tense lung; this is the characteristic note of acute tuberculosis, especially in the front of the chest. I shall have to refer to this point again in discussing the physical signs of tubercle.

The amount of distension and the amount of tubercle are not always proportionate, inasmuch as the presence of previous disease may render a small amount of tubercle



fatal; hence the note when tympanitic is sometimes resonant, sometimes muffled.

The percussion note at the posterior surface of the chest is often dull, the expansion of the lung being to the front, and the tubercle especially conglomerated towards the back, and the lung here condensed.

Variety in pitch requires great discrimination, and although seemingly but a slight difference, it affords significant indications of important processes.

There is no greater difficulty than the detection by physical signs of tubercle, even in large quantity; and in the early stage of incipient infection the thermometer must be used constantly if diagnosis is to depend upon more than conjecture.

*Synopsis of the Signs of Distended Lung.*

<i>Condition percussed.</i>	<i>Sound.</i>	<i>Pitch.</i>	<i>Extent.</i>
Healthy Lung. Before Inspiration.	Vesicular Resonance.	Low.	Normal.
Healthy Lung. Forced Inspiration.	Vesicular Resonance. Loud.	Higher.	Increased normal.
Lung distended. Compensatory Breathing.	Vesicular Resonance. Loud.	Higher.	Abnormally increased.
Emphysema.	Tympanitic Resonance on deep percussion. Muffled on superficial percussion.	High.	Increased.
Acute Tuberculosis.	Tympanitic Resonance (in small quantities), Muffled (in great). Dull percussion behind.	High.	Not increased (generally).

The next modification of sound to be described is that in which the vesicular resonance is diminished in various



degrees, or completely annulled; this diminution or absence of resonance constitutes **dulness** of percussion. It may be slight, marked or absolute, the degree being proportionate to the absence of air.

In dull percussion, resonance of the vesicular character is more or less abolished and the pitch is raised.

Dulness is caused by various products of disease.

By fluids	}	In the pleural sac—subpleural.
or		In the lung—intra-pulmonary.
solids		Outside the lung—extra-pulmonary.

**Subpleural Fluids.**—Accumulations of serum, pus, and blood, as in hydrothorax, empyema, hæmothorax.

**Subpleural Solids.**—Blood coagulum, thickened pleura, and foreign growths.

**Intra-pulmonary Fluids.**—Serum, pus, and blood with inflammatory fluid, exudates of all kinds, and hydatid cysts.

**Intra-pulmonary Solids.**—Solid exudates into the air-sacs—*e.g.*, pneumonic, thickening of the lung tissue, foreign growths invading the lung, blood fibrin inhaled into the lung.

**Extra-pulmonary.**—Aneurisms of the aorta, malignant and other growths, and enlargement of the glands.

Dulness is also caused by **absence of air** from the air-sacs, as in collapse and compression of the lung.

Collections of fluid in the pleural sac, not confined and bound down to the chest-wall, are diagnosed by alterations in the situation of the percussion-dulness by alterations in the position of the patient. This is a most important distinction which should never be neglected in percussing regions which give a dull sound. Some change, however, takes place in the position of a lung simply from gravity.

Ordinary pleuritic effusions are especially to be detected in this manner, the lung, if not adherent to the side, floating to the top of the fluid, and the fluid always tending to gravitate towards the most dependent part.



The presence of fluid within the lungs gives a dull sound on percussion only when the air is greatly displaced; examples occurring in marked pulmonary congestion and in pulmonary apoplexy.

Pulmonary cavities full of fluid, abscesses, and hydatid sacs give dull notes.

Tubercle seldom gives dulness of percussion in front, unless the lung be also affected with much catarrhal pneumonia, or unless it be in very large masses, as occurs occasionally in old chronic cases in advanced years, or with a fibroid condition of lung; then it is formed in large broad bands, and yields a dull note; dulness, however, may be detected behind.

Thickened pleura seldom gives any marked dulness of percussion, but I have seen cases in which the pleura has been so thick as to give a percussion note of a very dull character.

The third class of sounds elicited by percussion are those which are remarkable for absence of vesicular murmur, absence of dulness, and the presence of a peculiar resonance. These are called, according to their peculiar qualities—

1. **Tubular.** 2. **Tympanitic.** 3. **Amphoric.**

1. **The tubular (tracheal) sound** is elicited on percussion over the trachea in the upper sternal, or near the sternum in the infra-clavicular region. It has a hollow resounding quality, with ample vibrations, unlike the short vibration that characterises vesicular resonance.

It is heard in percussing regions under which are solids or fluids in close contiguity with a bronchus or the trachea, and as a rule is to be found in infra-clavicular, upper sternal, or infra-mammary regions in front, and the inter-scapular behind.

It occurs in cases of consolidation, of collections of pus overlying a bronchus, enlarged glands pressing upon the trachea, and occasionally at the base of the lung when dilatation of a bronchus is present, which is associated sometimes with compression of the lung. It has also



been noticed in deep-seated cavities containing air with solid tissue intervening between it and the thoracic wall.

Simple dilatations of the tubes will give the same note.

2. **Tympanitic resonance** derives its name from the drum-like quality of its sound, which is generally clear, prolonged, and of varying pitch, according to the tension of the elastic walls which surround the cavity yielding this sound.

In its typical form it is obtained by percussing a distended stomach, the note of which may be low if the tension be not great; or it may be high, and the vibrations proportionately short, if the tension be very great.

It is found under the following conditions:—

1. In pneumothorax, from the collection of air in the pleural cavity.
2. In cavities, the result of phthisis, situated immediately beneath the chest-walls with little tissue intervening.
3. The tympanitic resonance of the stomach or colon may be transmitted generally over the left side. I have known this mistaken for pneumothorax; the stomach being very much elevated by contraction of the left lung.

The sound elicited by percussing deeply over an emphysematous chest may be called tympanitic, and it partakes of this quality from the increased size of the thorax, from the increased quantity of air, and from dilatation of the bronchi.

Superficial percussion, as I have already remarked, elicits what is really the tense tympanitic sound, muffled and quasi-dull in quality. The same hardness of sound is characteristic of the highest note of the piano, which has none of the resonant quality of the bass strings.

Tympanitic resonance is not uncommon in the upper anterior region of the chest when the lower portion is compressed by fluid, and it is sometimes present in pneumonia of the upper lobe during the stage of hepatisation.



Upon the causation of this there has been much discussion.

Skoda, who was the first to draw attention to it, taught that it was explained by the fact that the lungs always yield a tympanitic sound when they are partially deprived of air, appealing for support of this opinion to experiments performed on the dead body; such experiments must, however, be always regarded suspectedly, inasmuch as they cannot be assimilated to the manifold conditions of life.

Williams believed that the resonance had a tracheal quality, and attributed it to condensation of the lung over the large tubes, the sound being caused by their hyper-resonant properties. This theory, which was also held by Hudson, has been in great measure adopted by Walshe; and as long as the resonance is confined to the bronchial regions—that is, close to the sternum in the second interspaces—this interpretation holds good.

The two conditions are not exactly, as the lawyers would say, on all-fours. When the sound is noticed in apex pneumonia, it is generally, *meo judicio*, towards the end of the disease, when the patient and the lung are recovering. Now, in pneumonia the lung is much enlarged and swollen by the fibrinous exudate into the alveoli, and not only is the lung much denser, but it is also of greater bulk than natural, as shown by the indentations of the ribs upon the surface.

If it happens that the upper lobe in its recovery, as the exudation is absorbed, becomes expansile to air—the chest-wall, however, being still thrown forward by the bulky swelling of the non-absorbed portions of the affected lung—the expanding tissue will have to meet the surface of the chest from the pressure of the atmospheric force; the air-sacs will thus be over-distended and give a percussion note of distension. This is the quality of note which, to my ear, is sometimes elicited under such circumstances.

The condition which also obtains in pleuritic effusion, when the lower part of the lung is compressed by the







emptying itself into a bronchus, the pus being ejected by the mouth and air taking its place.

It may also be heard in cases of pleuro-pneumonia, especially when the walls of the chest are very elastic.

**Cracked-pot sound** (Laennec's *bruit de pot fêlé*) is very similar to the chinking produced by striking the hands (placed together loosely, palm to palm) upon the knee. The sound is due to the sudden expulsion of air.

It is found in air-containing cavities with a free communication with a bronchus, and when the mouth is open, or when the chest is very yielding over a solid lung.

It has more interest as a sound than importance as a sign. It should be remembered that it can only be produced generally once or twice at a time, and requires an interval of rest before it can be reproduced.

**The Regional Distribution of Sounds.**—However practised an auscultator may be, he knows that, in addition to his knowledge of sounds, he must be furnished with the knowledge of the localities which certain forms of disease principally affect, in order to give a probable interpretation to sounds which may receive more than one explanation.

The regional distribution of sounds should especially engage the attention of the student, the significance of which will be best appreciated by a close attention to pathology. Diseases, like plants, have special habitats, and the physician looks to find certain diseases planted in certain districts.

There are some broad distinctions which may first be insisted upon—

1. Diseases arising from stasis of the bloodvessels, congestions of all forms have a tendency to gravitate, and appear in the most dependent parts.
2. Pleuritic effusions are situated near the diaphragm, in the lower half of the chest.
3. Diseases arising from irritation of the air-passages affect the apex and the expansile parts of the lung. This is also true with regard to the inhalation of matter, whatever that matter be.



**Vesicular resonance**, detected passing across the mid-sternal line from one side, and belonging to the lung of that side, indicates a crippled condition of the opposite lung.

Resonance found in exaggerated degree in the axillary regions and lower portions, with indications of phthisis elsewhere, would raise a suspicion of tuberculosis—if associated with aggravation of symptoms.

**Dull percussion**, in its various degrees affecting the **lower posterior borders** of the lungs (especially the right lung), suggests congestions, croupous pneumonia, or pleuritic effusions.

When situated beneath and **close to the lower angle of the scapula** (especially on the right side), percussion dulness indicates pulmonary apoplexy.

**Over the upper lobe**, in moderate degree, it suggests catarrhal congestion, forms of phthisis, and hæmorrhagic inhalation; in an absolute degree, the various pneumonias (croupous, catarrhal, scrofulous), and fibrosis of the lung.

**Over the middle portion** of both lungs dulness indicates brown induration, dependent upon mitral regurgitation.

Empyemata are generally localised in the lower axillary region of either side.

The dulness of enlarged glands and of malignant growths is to be looked for in the upper sternal region, or in the inter-scapular region close to the spine.

Dulness from aneurism is found in the upper sternal region, often to the right side, sometimes to the left; also in the left inter-scapular and infra-scapular regions.

Tympanitic resonance, when found in the upper portion of the chest, is due to cavity; when found in the lower part, it is due to air in the pleural sac.

These suggestions may be put into the following schema, but it must be understood that exceptions occur to the rules here laid down, and several diseases are omitted, as being infrequent, and detected better by other signs.



*Regional Significance of Dull Percussion.*

		<i>Right Side.</i>	<i>Sternum.</i>	<i>Left Side.</i>
<i>Anterior Region.</i>	Supra-clavicular.	Phthisis, advanced Fibroid thickening. Apex pneumonia. Large glands. Aneurism of Innominate Artery.	Scrofulous glands. Malignant tumours. Aneurism of Arch of Aorta.	Phthisis, advanced Fibroid thickening. Apex pneumonia. Large glands. Aneurism of Left vessels.
	Infra-clavicular.	Phthisis (all stages). Fibroid thickening. Congestion. Pneumonia { Croupous. { Catarrhal. { Scrofulous. Brown Induration. Aneurism of Ascending Aorta.	Aneurism of Arch. Malignant tumours.	Phthisis (all stages). Fibroid thickening. Congestion. Pneumonia { Croupous. { Catarrhal. { Scrofulous. Brown Induration. Aneurism of Descending part of Arch.
	Mammary.	Pneumonia. Pericardial effusion. Empyema. Hydrothorax.	Pericardial effusion.	Pneumonia. Pericardial effusion. Empyema. Hydrothorax.

<i>Posterior Region.</i>	Supra-scapular.	<i>Phthisis</i> , second stage particularly. Pneumonia, Congestion.	Phthisis, second stage particularly. Pneumonia (not common). Congestion.
	Scapular.	Phthisis (all stages). Fibroid disease. Pneumonia.	Phthisis (all stages). Fibroid disease. Pneumonia.
	Inter-scapular.	Phthisis. Tumours pressing on Bronchus. Aneurisms. Large glands.	Phthisis. Aneurism of Descending Aorta.
	Infra-scapular.	Pneumonia, croupous. Pulmonary apoplexy. Congestions. Compressed lung. Pleural effusions.	Pneumonia. Congestions. Compressed lung. Aneurism of Descending Aorta. Pleural effusions.
<i>Axillary.</i>	Upper.	Phthisis. Fibroid Disease. Inhaled blood.	Phthisis. Fibroid disease. Inhaled blood.
	Lower.	Empyema. Pleural effusions.	Empyema. Pleural effusions.



## CHAPTER XI.

## AUSCULTATION, AND THE STETHOSCOPE.

**Auscultation** is the term used to indicate the examination of the chest by the sense of hearing, the ear being applied to the surface to listen for the sounds caused by respiration, the action of the heart and the voice under conditions of health and disease.

Auscultation may either be **immediate**, the ear being applied directly to the surface of the body; or it is **mediate**, some substance or instrument capable of transmitting sound being interposed between the ear of the observer and the chest of the individual examined.

The most convenient form of instrument is the single stethoscope—a cylinder of pine-wood expanded at one end—to be placed on the chest of the patient, and fitted above with a large circular flat expansion, the ear-piece applied to the ear of the observer.

An illustration of a shape which is recommended for ordinary use is given, with the size of the mouth- and ear-piece. The whole length of the instrument should be seven inches, the diameter of the cylinder five-eighths of an inch; the ear-piece being about three inches in diameter, and the mouth one and one-eighth of an inch.

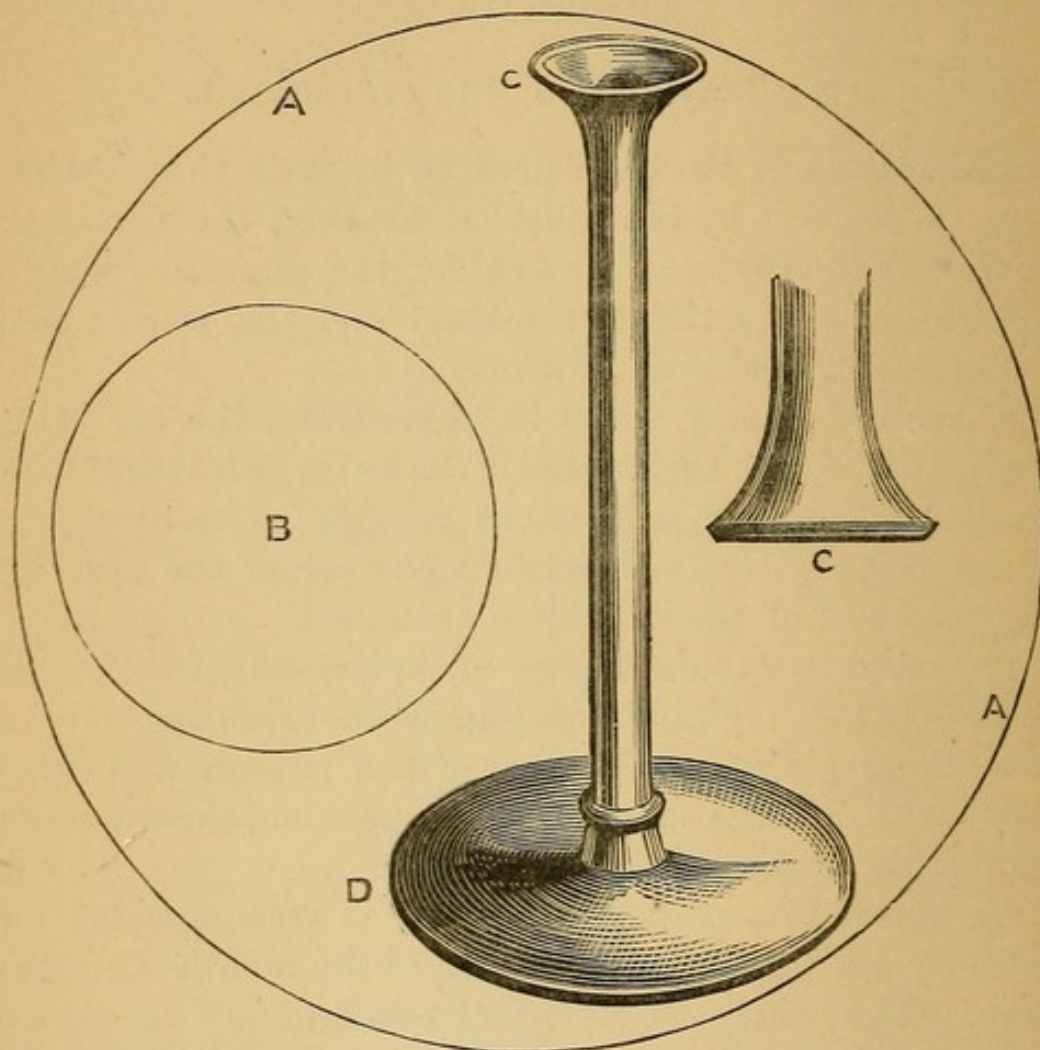
The ear-piece should be slightly hollow, and adapted to the ear. If carefully made with good pine, the stethoscope can bear a good deal of use without breaking. If a tougher instrument is required, it may be made of American birch.

The metal stethoscopes are extremely objectionable, and should not be used, inasmuch as they impart a metallic sound of their own to all vibrations transmitted through them.



The double stethoscope, of which an illustration is given, is of great value, increasing the sounds heard over the chest, and preventing any extraneous noises from reaching the ears during auscultation. It also has the advantage

FIG. 15.



Stethoscope.—A, size of ear-piece D. B, size of mouth C.

of using both ears equally, and the patient can be examined by it with greater ease.

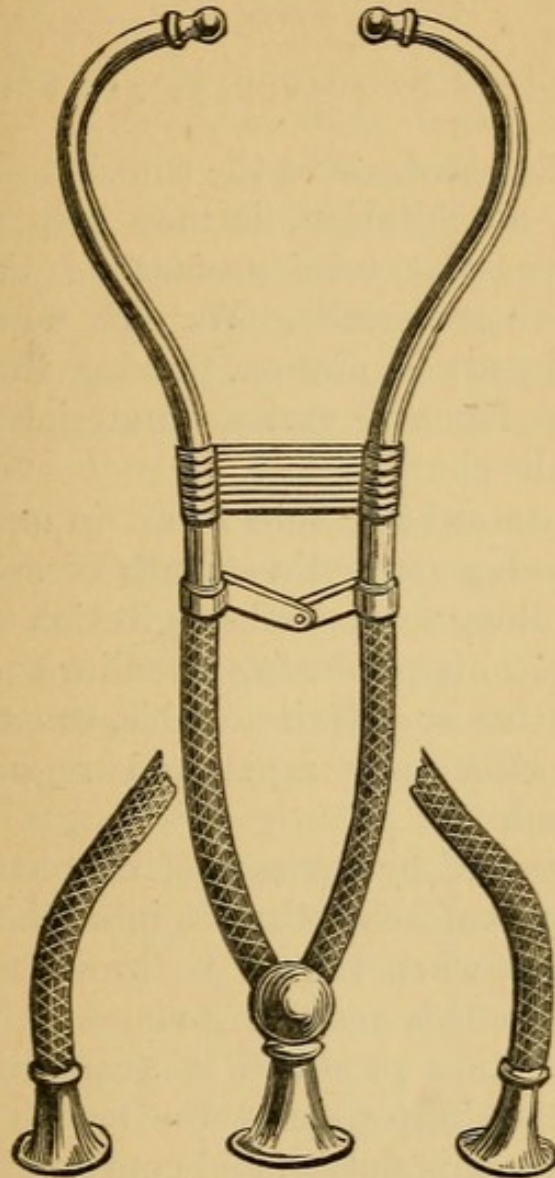
This stethoscope can be furnished with one end or two. With two mouth-pieces it can be used for comparison of sounds in different regions, which is often advantageous in determining the exact localities of cardiac sounds.

The first mediate instrument used by Laennec on the occasion of the discovery of mediate auscultation (the



embryo, in fact, of the stethoscope) was a quire of paper tightly rolled up into cylindrical form: the present shape of the stethoscope resulted from modifications suggested by Piorry upon the form of instrument devised by

FIG. 16.



Double Stethoscope.

Laennec. When the instrument is being used it must be held lightly like a pen, placed quite flat upon the surface, and closely applied with gentle pressure to the part examined.



## CHAPTER XII.

## THE ACOUSTICS OF PULMONARY AUSCULTATION.

BEFORE proceeding to describe the sounds to be heard and listened for in auscultation, it may help to clear the ground if I give some brief account of the influences which modify these sounds. We are now considering sounds caused by air in motion, passing through tubes, and transmitted through various materials to the ear placed outside the chest.

If the air contained in a tube be set in motion—as, for example, by blowing across the mouth of such a tube, or if it be driven along a tube—the agitation gives rise to a series of vibrations producing peculiar sounds; sometimes only a flutter or puff is audible, sometimes (if the vibrations are sufficiently rapid and regular) musical sounds are heard.

Sounds are caused by currents of air passing through tubes, the air column being thrown into vibration. This especially happens when the air is thrown against edges and broken up; in this manner giving rise to fluttering noises similar to that produced in an organ-pipe by air driven against the sharp edge at the mouth of the pipe. The pipe takes up this flutter, and converts it by its own resonance into a musical note. In the production of voice sounds, the air is thrown against the vocal chords, and into the resonant cavity of the mouth. In the production of pulmonary sounds, air is drawn through the glottis into the tubes, a puff or blowing sound being thus produced.

Sound is very much diminished in passing from one kind of substance to another—it is, in fact, diffracted in the



same manner as light, and it is broken up when passed from the vocal chords through healthy lung to the surface of the chest. If the stethoscope be placed in the trachea the voice sounds will be heard clear and loud; but let it be placed in the axillary region, and an indistinct buzzing, from confused vibrations, can only be heard—articulate sounds being lost.

This may happen in some measure from the closed condition of portions of the lung, which is not fully and rigidly expanded in health; but whatever be the true interpretation, the fact is indubitable.

In conditions of disease, however, the voice sounds and the blowing tubular sounds formed in the trachea are transmitted with intensity; and it appears that intensity is retained by passage along rigid tubes, as well as by conduction through solid matter.

The difference in the intensity of a sound transmitted through india-rubber piping and a rigid cylinder is very great. Biot found that he could hold conversations through an iron pipe 3000 feet in length. This explanation appears to me the best adapted to meet the phenomena of transmitted voice sounds, which will receive further consideration when Bronchophony is discussed.

Experiments made on lungs removed from the body, or on the dead body, even with the organs *in situ*, are to my mind of no value; and some very valid objections against conclusions derived from such experiments have been concisely urged by Walshe.

Some experiments recorded in the *Revue Mensuelle* for 1877, by MM. Bondet and Chauveau, are worthy of consideration in illustration of this subject.

The experiments were made on a mare affected with inflammation of the left lung, which was consolidated. Auscultation of this lung detected the presence of a double blowing sound over the upper half; and over the trachea a double tubular sound, having the same character as those heard over the lung, but of much greater intensity. An incision was made in the trachea, but a little blood



dribbled into the tubes from the wound, giving rise to coarse bubbling rattles, and an interval of time was allowed to elapse until the sounds had returned to their previous condition. When the wound was kept open inspiration was no longer blowing, while expiration was still but only slightly blowing. On one or two occasions the sounds heard with such intensity over the consolidated lung were temporarily completely suspended and annulled, evidently by the presence of a plug of mucus in the tubes, as they were restored after the animal had coughed the plug away.

On the healthy side the vesicular murmur was normal and distinct.

An india-rubber tube, with reed attached, was then inserted into the trachea, and air was forced through the reed. The noise was most distinctly heard, and it seemed to be produced close to the ear listening over the consolidated portion of lung. The healthy tissue, on the contrary, failed utterly to conduct the sound.

It is quite evident from these experiments that sounds produced in the trachea or larynx are not transmitted along tubes surrounded with healthy lung tissue, but are passed without loss along tubes surrounded by solid matter. But it is also evident that the blowing sound in the trachea was heard, although in diminished intensity, over the solid lung; and, moreover, that this was caused partly by the passage of air through the glottis, inasmuch as it was lost when the air passed through the wound in the trachea.

The abolition of the sounds from the presence of a plug in the tubes appears to prove that the sound is transmitted mainly by the air in the tubes.

These experiments are of a simple character, but they throw light on many obscure points which deserve full consideration.

A great deal of discussion has been raised with regard to the cause of the vesicular pulmonary murmur, which I shall not reproduce here, inasmuch as I believe the con-



clusions of Barth and Roger (which are supported by their experiments) and those of Walshe are correct—namely, that the sound is complex, that the true vesicular sound is found in the air-sacs, but that some portion of the pulmonary sound is transmitted from the bronchial tubes. The conditions which appear to affect the transmission of sounds from the trachea are very complicated; the condition of the tubes, the tension of the air-sacs, the close approximation or adhesion of the surface of the lung to the chest, and the presence of foreign matter in the place of air, all have to be considered in this complex problem. One lesson at least we may learn from all these difficulties—that the interpretation of the significance of any sound for the purpose of diagnosing the condition of the thoracic organs can only be made after careful deliberation with respect to all the signs and symptoms which may result from a full examination of the patient, and reason must decide upon the varied phenomena which are presented, not only to the ear but to all the other senses.

Tubal sounds are modified by the presence of fluids which give rise to bubbling sounds of more or less fluidity or viscosity, according to the nature of the secretion. Often vibrations are thus set up in the tubes which produce sounds differing in pitch, according to the size of the tubes in which they originate; the large tubes sounding deep snoring notes, the small tubes high-pitched, whistling, or wheezing notes.

Sound is very much broken up and diminished by having to pass from one medium to another; a fact which is illustrated by a simple experiment. Put a solution of a carbonate (potash or soda) into a large glass, and ring the glass by tapping it: the sound is free and clear. But add a little acid to the watery solution, bubbles of carbonic acid are given off, and if the glass be touched now it will be found that the sound has gone, only coming back when the effervescence ceases.

So also sound in passing from lung to fluid is much



broken up, but it would be a mistake to suppose that because little sound can be heard through pleuritic effusions that fluid therefore does not conduct sound, the fact being that the velocity of sound in water is more than four times its velocity in air. The sounds of respiration are not only diminished by diffraction in passing through another medium, but also, it must be remembered, that the pressure of the fluid prevents the expansion of the lung. The heart's sounds, it must be mentioned, are heard in such cases.

The amphoric and metallic sounds that are heard in some cases of disease are additional sounds (overtones) added to the primary sound. If a jar or bottle be blown into, a distinct additional sound is heard, as well as a low note, which may be called the fundamental note; with the metallic sound the overtone is of a remarkably ringing high-pitched character, and well imitated by dropping a pin into a glass.



## CHAPTER XIII.

## AUSCULTATION OF THE HEALTHY LUNGS.

THE person under examination should assume the position which has already been recommended as the best suited for percussion of the chest; the erect posture is the easiest, but if it be more convenient for the patient to be seated, the examiner should kneel on one knee by the side, in order that his head may not be much bent. Dress should be removed, so as to have at least the upper part of the chest uncovered; with men it is much better to have the shirt taken off. Before entering upon a description of the sounds of respiration, I must first warn the student of certain fallacies to be avoided in the shape of—

**Extraneous Sounds.**—Flannel, rough linen or cotton, chest-protectors, and all manner of rustling stuffs should be put out of the way, or crepitant and friction sounds, entirely obscuring diagnosis, will assuredly be heard on listening through the stethoscope. The hands should be kept from the chest, as their rubbing becomes very audible. The creaking made by stays renders it advisable that these articles of dress should be taken off.

**Sounds of Hair.**—In some persons the presence of much hair about the chest is a serious obstacle to free auscultation, and a rough skin, as, for example, ichthyosis, and the like.

**Muscular Murmur.**—This sound is heard during the contraction of muscle; more superficial than the pulmonary sound, low in pitch, and of a continuous rumbling quality; it occurs where the muscles are thickest under the humeral end of the clavicle, and above and below the spine of the scapula.



It is easy to solve any difficulty with regard to this sound, by making the patient hold his breath and contract the muscle under the stethoscope; the muscular murmur will then continue, the pulmonary sounds being silent.

Professor Haughton has some interesting remarks on this sound. Dr. Wollaston, who called attention to it in 1809, likened it to the rumbling of distant wheels.

Haughton found that it corresponded to DDD slightly flattened, or two octaves below the ordinary bass D, corresponding to thirty-six vibrations in the second.

From a calculation made with regard to the vibrations of wheels, their rumble corresponded to thirty-five vibrations per second; a calculation which curiously supports Wollaston's description.

Remarkable instances sometimes occur and the following case is worthy of note:—It was observed in a man who was largely employed in delivering circulars in London, and from his insufficient food suffered much from muscular exhaustion and over-walking. The muscles were peculiarly irritable, and subject to fibrillations and twitchings. The pulmonary sound was almost obscured by the noise of the muscular murmur.

### **THE RESPIRATORY SOUNDS OF HEALTH.**

The air is conducted from the mouth through the glottis and trachea into the large bronchi. These tubes subdivide rapidly and terminate in minute bronchioles, which conduct the air to the spongy elastic air-sacs situated round the thoracic walls.

The sounds which are heard in various regions during the act of respiration are modified according to the parts over which auscultation is directed; and it is necessary to distinguish three kinds—

1. **Tracheal.**
2. **Bronchial.**
3. **Vesicular.**

1. **Tracheal.**—This sound is heard by placing the stethoscope over the trachea or larynx.

The quality of sound is that of a harsh rushing cur-



rent to and fro (up and down) through a tube; it can be imitated by pursing the lips together and drawing air in and expelling it rather sharply.

This quality is termed tubular, and this sound gives the characteristic indication of what is meant by this term.

The pitch is high, and the intensity varies according to the act of respiration, being loud in deep breathing. There are differences with regard to the inspiratory and expiratory sounds, which demand attention.

**Inspiratory.**—**Quality**, tubular; **pitch**, high; **intensity**, variable; **duration**, shorter than the inspiratory act.

**Expiratory.**—**Quality**, the same; **pitch**, higher; **intensity**, greater; **duration**, often longer than the inspiratory.

There is a short interval which separates these sounds.

2. **Bronchial.**—This sound is a modified form of the tracheal, than which it is less intense, not so whiffing in quality, nor so hollow; it may be imitated by not closing the lips so tightly as for the tracheal, the opening being larger, and the sound resembling that of “who.” As compared with the pulmonary sound it is harsher, higher pitched, and tubular.

This sound is heard on listening over the second interspace close to the sternum, in front on either side, and behind, in the middle of the inter-scapular regions of either side between the fourth and fifth dorsal vertebræ.

3. **Vesicular Murmur.**—This term, as being that used by Laennec, I have kept to indicate the sound of respiration. In its typical form it is best heard in the upper axillary region, and also in the infra-clavicular, which is usually the region appealed to for the sound of healthy breathing. This murmur is peculiar to a condition of healthy elasticity of the air-sacs: the sound is never simulated by disease, and affords conclusive proof of a healthy condition of the part from which the sound issues. It may



be studied with advantage in children, owing to its greater intensity in early life. Its intensity varies with individuals; it is loud in childhood, feeble in old age, and louder in women than in men.

**Quality.**—To this the term vesicular may be applied, the character of the sound being soft and rustling. Laennec compared it to the sound made by a bellows without a valve; a better comparison is the soft murmur of a summer breeze through the leaves.

The difference between the sounds of inspiration and expiration is very important, and must be carefully studied and learnt.

**Inspiratory sound** begins with a soft distant whisper, increasing with inspiration, and becoming rustling in quality at the end of the act. It is heard from beginning to end of the inspiratory act.

**Pitch**, low but higher than that of expiration.

**Expiratory sound** begins with the gentle rustle and passes into a low-pitched breath or puff, which is neither vesicular nor bronchial in quality. The sound goes away from the ear, and is much shorter than that of inspiration.

**Pitch**, lower than that of inspiration. **Intensity**, less.

*Synopsis of Respiratory Sounds.*

<i>Characters.</i>	<i>Inspiratory.</i>	<i>Expiratory.</i>
Quality.	A soft breath, ending in a rustle.	A rustle, ending in a soft puff.
Pitch.	Higher.	Lower.
Intensity.	Greater.	Less.
Duration.	3.	1.

The following are the regional peculiarities in the respiratory sounds:—

**Tracheal**, heard at the upper sternum.



**Bronchial**, in front at the second intercostal space, right and left, at the back between the fourth and eighth dorsal vertebræ, more marked on the right side.

**Vesicular**, best heard in the upper axillary, infra-clavicular, infra-scapular, and mammary regions.

There are some differences in the respiratory murmur of the two sides.

<i>Sound.</i>	<i>Right side.</i>	<i>Left side.</i>
Inspiratory.	Less intense. Less vesicular. Higher in pitch.	More intense. More vesicular. Lower in pitch.
Expiratory.	Longer. Higher in pitch.	Shorter. Lower in pitch.

**Vocal Resonance.**—When the stethoscope is placed above the sternal notch upon the trachea, and the person examined speaks, the voice is transmitted with great and even painful intensity, the sounds being concentrated into the ear, the words being imperfectly articulated. At the upper sternal region this resonance is diminished, and it becomes weaker as the stethoscope is carried down the bronchi. Over these parts the articulation of words becomes very imperfect, the sound being diffused and no longer appears to be concentrated directly into the ear, but to come from a distance. This constitutes **Normal Bronchophony**.

When much pulmonary tissue intervenes, and the stethoscope is placed over regions where the vesicular murmur is best heard, the articulation of words is obliterated, an indistinct buzzing solely is heard, the voice sound scarcely reaching the ear at all. Voice resonance varies in individuals, and it is increased or diminished by the following conditions:—



<b>Increase.</b>		<b>Diminution.</b>	
Loud	} voice.	Feeble	} voice.
Low-pitched		High-pitched	
In men,		In women.	
In old people,		In children.	
In front,		Behind.	
Right side,		Left side.	
Clavicular	} regions.	Cardiac	} regions.
Inter-scapular		Hepatic	

**Whisper.**—Articulated speech formed in the mouth by the air of expiration (or inspiration) without the intervention of voice sounds produced by the vocal chords, constitutes whispering.

Voice sounds can be formed by singing with the open mouth without articulation.

Articulated sounds can be produced with laryngeal or true voice sounds, by ordinary speech, or by vocalising words (in songs), or

Articulated sounds can be produced without laryngeal sounds, as by whispering.

The chief advantage of the whisper appears to lie in the total absence of vocal fremitus, which interferes to a great extent with the appreciation of transmitted sounds; moreover, sometimes the voice is transmitted with such vibration and force as to be extremely painful to a delicate ear. This sound must be conducted by the air in the tubes; from the trachea the whisper is conducted to the ear as a high-pitched blowing sound, the words being transmitted but not distinctly, while from the pulmonary tissue it is conducted as a feeble, low-pitched blowing sound (often it is inaudible), louder generally on the right than the left, and of lower pitch on the right side.

**Fremitus**, the palpable vibrations of sound, has already been described in the chapter on Palpation.

**Cough.**—In health the act of coughing is not accompanied by a shock of succussion, but gives rise to a hollow sound, heard with the expiration over the trachea, and to



a short, dull, diffused sound heard over the pulmonary tissue.

By the act of coughing air is forcibly expelled from the lungs so as to induce free inspiration and expansion of the air-sacs with the next breath, and by removing plugs of mucus which obstruct the bronchial tubes; or by driving air into fluids, which otherwise remain quiescent and silent, it may be made subservient to the purposes of diagnosis.



## CHAPTER XIV.

## AUSCULTATION IN DISEASE OF THE LUNG.

**Morbid sounds**, caused by various conditions of disease of the lungs, may be classified under three headings:—

1. **Variations of vesicular sounds,**
2. **Alterations of respiratory sounds,**
3. **Adventitious, or new sounds,**

1. Under the first head may be described the phenomena of those conditions which induce variations (including total suppression) of the vesicular sounds, as regarding

- a. The intensity of the vesicular murmur.
- b. The delivery or rhythm of respiration.

The intensity of the vesicular murmur may be—

**Increased, Diminished, Suppressed.**

**Increased Vesicular Murmur** (Exaggerated).—

This is due to functional activity of one lung or a portion of lung from morbid diminution of function consequent upon disease in another part.

It is sometimes termed

Puerile	}	Breathing.
Supplementary		
Compensatory		

It foreshadows the substantial presence of disease in some other region, and is more intense in proportion to the vicinity of the disease. Its extent must be carefully examined and tracked (if necessary) across the mid-sternal line; if the opposite lung be useless from phthisis, fibrosis, or perforation, the sound lung may increase to a very large extent.

**Diminished Vesicular Murmur.**—This may occur



simply from functional weakness, the result of constitutional debility or of some enfeebling disease; it may be due to disease extraneous to the lungs, as in paralysis, peritonitis, ascites, and abdominal tumours of all kinds: to organic disease involving the respiratory apparatus, as in obstructive disease of the larynx and bronchi, and diseases of the lungs and pleura.

It may be persistent, as in prolonged and continued organic diseases; or it may be intermittent, as in pleurodynia, asthma, and temporary obstructions of the air-passages.

The most common persistent diminution results from **pleural adhesion.**

It must also be remembered that the chief part of the inspiratory act being due to the contraction of the diaphragm, anything which interferes with its full action will diminish the vesicular murmur of inspiration in the upper lobe.

**Suppressed Vesicular Murmur.**—This is complete absence of sound—silence during respiration. It may arise from complete obstruction of the air-passage, the effusion of fluids into the pleural sac, or the presence of air there.

It is also due to chronic conditions of fibrosis with complete adhesion of the pleura. These silent conditions must not be passed over as unimportant: the absence of sound is much more difficult to diagnose than its presence.

**Alterations in Rhythm.**—The actual duration or the relative proportion of inspiration and expiration may be altered, and give rise to corresponding alterations of sound.

Inspiration may be morbidly shortened, or it may be deferred.

**Shortened inspiration** may be due to hampered expansion of the lung, as from adhesion of the pleura, or from incipient and painful pleurisy.

If the air-sacs are occupied, as, for example, in early



stages of consolidation, the inspiratory act may continue after the sound has ceased.

**Deferred inspiration** is an important sign in emphysema. The respiratory act begins and continues before the air-sacs respond to the expanding air. It also occurs in distension of the lung from any cause, whether it be compensatory or the result of tubercle.

Expiration may be morbidly shortened or prolonged.

**Shortened expiration** occurs as a result of distended lung: the complete closure of the air-sacs is prevented in consequence of the extra atmospheric pressure which strains the particular regions distended. The dissemination of hard tubercle in certain air-sacs compels the extra distension of others in the vicinity.

**Prolonged expiration** may be due to diminished elasticity of the lung, and is a frequent sign of emphysema. To such an extent may this be carried as to reverse the ordinary proportions of inspiration and expiration.

Prolonged expiration has been by many considered as evidence of phthisis and an indication of an early deposit of tubercle, but as long as the pitch of the sound is normal in character, no importance can be attached to this sign. It must be remembered that the inspiratory sound may be considerably prolonged, on the right side particularly, and yet be quite consonant with a healthy state of lung. As this is very generally believed to be an important sign, I shall quote the opinions of Walshe and Flint, with whose conclusions I perfectly agree.

Walshe says\* on this sign: "The signification of the expiratory sound was in some degree mistaken by its re-discoverer, Jackson; he exaggerated its specific importance as a diagnostic sign of tubercle, and in this he has been very generally followed. Many persons forget that what may appear in a given individual as compared with another, prolonged expiration, is really in him a natural state."

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\* Diseases of the Lungs and Heart, p. 100.



Flint says :\* “ A prolonged expiration at the summit of the chest on the right side is sometimes incorrectly considered to be evidence of phthisis. It is to be recollected, in the first place, that prolongation of this sound with a normal pitch and quality is never evidence of solidification of lung, either from phthisis or any other disease ; and, in the second place, even if the pitch be high and the quality tubular, that it is not to be regarded as abnormal, provided the inspiratory sound is unchanged and other signs of disease are not present.”

Vesicular resonance alone, which is simply prolonged, must not be accepted as evidence of disease.

**Suppressed expiration** is only heard under one condition of disease—an old contracting cavity. I have now watched many cases, especially in young persons, in whose lungs a cavity has formed and healed, and the only sign revealed to the ear was a complete suppression of the expiratory sound. In a number of cases this has been observed on the left side ; in some cases the cavity has been so small and the improvement so great that little retraction was perceptible, but in other cases flattening has taken place, together with some dislocation of the heart upwards.

The explanation of this depends upon the contraction of the tube communicating with the cavity, which will admit air in free expansion, but soon shuts so as to prevent at least any sound of expiration. The sign is by no means infrequent (if the number of healed cavities be taken into account), and is of great diagnostic value.

The inspiratory sound is usually in these cases high-pitched but still vesicular, in consequence of the expansion of lung tissue in the vicinity of the cavity.

The evidence in proof of this statement is derived from observations extending over some months, and even years, during which time the cavity has been detected from its origin to its contraction. In other cases the presence of

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\* On Auscultation, p. 110.



the cavity has been substantiated by other physical signs.

Respiration may be **hesitating** in its delivery; there may be some interruption to free breathing, instead of a continuous delivery. This constitutes stammering, or wavy respiration. Now, as long as this interruptedness is not regular and uniform it is generally due to nervous respiration, and is to be found in hysterical and hypochondriacal individuals, especially at a first examination. If this be the cause, it passes off when the patient has become more tranquil.

If respiration be short and jerky the interruption may be caused by pain, and this condition is found in pleurodynia and early pleurisy.

Marked **jerking** occurs when partial or long adhesions have taken place, the continuous expansion of the lung being thus prevented; this may be found in progressing adhesion of the pleura in the middle region, between complete adhesion and incipient pleurisy. Under these conditions, however, it coexists with other signs.

**Cogged respiration** is a sign of more importance, and I believe is found in the early stages of cancer when obstruction of the bronchial tubes is produced by this cause. In such a state of disease the cogging is very slow, with long but regular intervals between; it is most marked during expiration.

It is also met with, as Walshe observes, in connection with tubercle, and I have found this more especially at the back of the lungs. This kind of cogging is very fine, the intervals being small and frequent, and is probably caused by agglutination of the tubes, in which a thick glutinous exudation is often found from tubercle. It is sometimes heard at the apices, but I have more often met with it at the bases of the lungs.

2. The **second class** of sounds comprises those modifications in which the vesicular murmur is altered in quality and pitch.

One of these is a dry papery quality of the vesicular



murmur; an early indication, in my opinion, of syphilitic pulmonary disease. Many instances of this have come under my observation, and it is a noteworthy sign of the pulmonary disease which is found coexistent solely with syphilitic cachexia; I conclude therefore that it is specific. In more advanced stages it is accompanied with considerable harshness of sound, which is sharper and more blowing in character.

This latter sound also belongs to peribronchial thickening of all kinds when it is tubular in character, and to thickened states of the pulmonary tissue when it is high-pitched.

The last quality of sound is especially heard in cases of profuse hæmorrhage from the lungs, and I believe it to be due to the inspiration (insufflation) of blood into the lungs, the blood corpuscles actually passing into the alveolar tissue and stiffening it.

3. **Adventitious sounds** are those which have no similarity to healthy sounds, and indicate in all instances disease of some kind.

Although it is not desirable to refine overmuch in the verbal description of sounds, it is necessary to separate into distinct classes sounds which are capable generally of some peculiar interpretation; but the student must distinctly understand that the object in view is a different one from that of insinuating that a particular process has throughout its progress one special sound pathognomonic of that process solely. The varieties of sound are as different as the shades of colour; and, although broad distinctions of a general kind may be put on paper, the difficulties of defining a sound in practice and of forming a prognosis from sounds are almost overwhelming; and the marvel is, not that errors should be committed, but that a just estimate of the process and progress of a disease should so often and with so much accuracy be made.

The classification which I shall adopt as best suited for practical use is the following:—It will be found convenient to consider morbid sounds as arising from—



- a. **Diseases of the alveoli.**
- b. **Diseases of the bronchial tubes.**
- c. **Destructive diseases of the lung.**
- d. **Diseases of the pleura.**

Some preliminary remarks are necessary as to the terms used, as it has frequently happened in works on this subject that one sound is expressed in different terms, and consequently a good deal of confusion has arisen. The terms used here shall be as far as possible those that were introduced by Laennec, but they cannot always be used in exactly the same manner and with the same limitation; besides which it is necessary to use additional terms of English origin, sanctioned by the authority of English writers.

The terms used by Laennec were the following :—

1. The *râle crepitant humide*, or *crepitation*.
2. The *râle muqueux*, or *gargouillement*.
3. The *râle sonore sec*, or *ronflement*.
4. The *râle sibilant sec*, or *sifflement*.
5. The *râle crepitant sec à grosses bulles*, or *craquement*.

The terms in general use in England have been the word *râle* adopted from the French, signifying rattle; *crepitation*, signifying a crackle; and *rhonchus*, adopted from the Greek, signifying snoring. Unfortunately confusion has arisen from the use of the last term *rhonchus* as a substitute for *râle*, and the word has been used by Williams and Walshe in this sense. The objection to this lies in the fact that simple bubbling has no snoring quality about it, and I have used the term *rhonchus* solely to indicate the snoring sound heard in diseased conditions of the large bronchi.

The terms used here will be the following :—

- Crepitation**, a fine dry crackle.
- Rale**, the rattle of bubbles bursting.
- Rhonchus**, a snoring sound.
- Sibilus**, a whistle of varied pitch.
- Gurgle**, a hollow gurgling rattle.



Other words and adjectives will be used to indicate the special quality of sounds in proportion to their approximation to any of the above terms, and it will be my endeavour to keep the divisions distinct and simple, rather than by over-refining to multiply differences and increase confusion.

**Râle** was the term applied by Laennec to the sound caused by the passage of air through liquids, the typical râle being the loud rattle of the dying; the air passing through the secretions collected in the tubes and the bases of the lungs owing to the strength of the patient being no longer effective to expel them.

Adventitious vesicular sounds may be divided into the following classes; and they will be named mainly with reference to their degree of moisture, although their respective significance is very different:—

- |                             |                              |
|-----------------------------|------------------------------|
| 1. <b>Fine crepitation.</b> | 3. <b>Subcrepitant râle.</b> |
| 2. <b>Crepitant râle.</b>   | 4. <b>Bubbling râle.</b>     |

1. **Fine crepitation** indicates the crackle of a dry but sticky surface, imitated very exactly by rubbing a lock of hair close to the ear between the finger and thumb. Laennec compared it to the crackling of salt in the fire. This sign is the most distinct and best defined of any throughout the range of auscultation.

It may be described as composed of a multitude of minute dry crackles, following closely and regularly one upon the other during the act of inspiration solely. The idea of bubbling is not present to the mind when this sound is heard, and indeed it is often difficult (except by the to-and-fro nature of its production) to distinguish between the fine crepitation of pneumonia and the fine friction of the pleurisy which so often accompanies the former disease. Flint has a very graphic description with reference to this sound of pneumonia, which is worth quoting. He remarks of it: "A distinctive feature is the abrupt development of the crepitant râle; a shower of crackles, as it were, springs up at the end of a forced inspiration." If I may venture on a simile, I should say that it reminds me of the explosion



of a maroon rocket bursting suddenly into a shower of crackling and brilliant stars.

The similarity of the sound produced by rubbing hair or squeezing a piece of spongy india-rubber suggests that it is caused by the successive unfolding of surfaces agglutinated together, and this theory accounts for the limitation of the sound to inspiration.

This sign is characteristic of pneumonia of the croupous form, but not of that form only, as it is sometimes heard in catarrhal pneumonia, a disease which has a different termination, duration, and prognosis.

I have watched cases of catarrhal pneumonia beginning with this sign and rapidly going on to consolidation, simulating very closely the croupous form of pneumonia in physical signs, the aspect and subsequent condition of the patient sufficiently indicating the nature of the disease, which has been afterwards determined by actual inspection in the dead-house.

The fine crepitation of croupous pneumonia is of short duration, and is accompanied by symptoms indicating great disturbance. Catarrhal pneumonia is more insidious and prolonged; other forms of pneumonia and congestion sometimes simulate this sound very closely, but it will be sufficient to mention the fact.

**Regional significance.**—Croupous pneumonia attacks lobes; catarrhal pneumonia attacks lobules. In the first case, large tracts of the base or the apex are involved; in the latter, the spots over which the sounds may be heard are more limited, and not generally occupying the borders. They are more central; moreover, isolated patches sometimes occur with intervening lobules unscathed.

**2. Crepitant rale.**—This term is used as intermediate between fine crepitation, which is of a dry, sticky character, and the true rale, which has a bubbling quality. It is synonymous with the moist crackling rhonchus of Walshe, or the humid crepitation which is of classic use in the Brompton Hospital. It will be seen that I have adopted partially a term of Laennec's, but it is to be hoped that



no confusion will be thus created, as the term has a distinct significance of its own, and intended to apply solely to the peculiar class of signs which are heard in initial stages of phthisis.

The crepitant râle is a crackle of larger size and more viscid character than fine crepitation. It consists of a series of few short, high-pitched, irregular crackles heard during inspiration, and the interpretation which may be put upon it is this : the unfolding of air-sacs full of viscid secretion or exudation of a catarrhal type. It may be found in the vicinity of nodular tubercle, but it is not due to this form of tubercle simply, although it may be due to that impairment of the alveolar wall which constitutes a main feature of the tubercular process.

A full and excellent description of the nature of this process in its various stages will be found in Dr. Green's work on Pathology.

The condition of the alveolar tissue is estimated by the high pitch of the sound induced by the amount of thickening present, and by the blowing quality of the expiratory sound. When these two signs are present the limitation of the sound to inspiration is not necessary for determining the character of the process ; and it may with some confidence be asserted that a serious lesion of the pulmonary tissue is taking place. The chance of recovery will depend upon the constitutional power of repair which belongs to the condition of the patient ; but, as far as my judgment goes, it should be considered as a structural ulceration, and not as the result of a general infection. If the recuperative power is at a minimum, the destruction will go on rapidly, and may lead to infection ; either general, resulting in acute general tuberculosis ; or local, resulting in lobular tubercle.

**Regional significance.**—The regions which this sound generally frequents are the infra-clavicular and the supra-scapular ; and the latter region should always be examined, inasmuch as the sound may be heard there when it is inaudible in front.



To the same category must also be referred two other sounds which have a similar significance, and are imperfect utterances of the moist crepitant râle.

These are the **viscid crackle** and the **occasional click**.

The **viscid crackle** appears to belong to a more chronic condition of exudation than the moist crepitant râle, and it is heard when caseation is present, or when a chronic fibroid condition of the lung tissue is softening. It is usually a sign of some chronicity.

The **occasional click**, if solitary, is suspicious of lurking mischief, or of recovery from previous disease; but if this sound be scattered and heard in various parts of the chest, it is of very serious import.

This is almost the only sign which may be heard when the chest is completely invaded by scrofulous pneumonia; and it affords a good instance of the silent advance of a very pernicious disease.

When scattered clicks are heard over the chest, it means that the lobules are choked with exudate—either the result of an extensive scrofulous pneumonia, or it may be a rapid deposit of lobular tubercle in the midst of a caseating catarrhal pneumonia.

All these signs—the **crepitant râle**, the **viscid crackle**, and the **occasional click**—belong to similar processes, and all come under the category of phthisis. Sometimes, in whatever form they are subsequently revealed, they remain silent until started by a cough-breath; and it is therefore very necessary to test the condition of the lungs by a cough followed up by deep inspiration, or those ominous heralds of future events may escape notice.

**Exceptions to the Rule.**—Viscid crackles and clicks do occur as a result of chronic bronchitis; but if innocent in nature, they are only to be heard in one or two places, over large tubes, or accompanied with tubal sounds, which additional signs sufficiently guarantee, in most instances, their inactive character. At the same time, for accurate diagnosis, the whole aspect of the case must be carefully



considered, especially whether the patient be losing flesh or not.

These sounds also follow the clotting of blood in the alveoli. In profuse hæmorrhage blood passes into the lungs (chiefly by insufflation) and gradually clots. This is detected by the bubbling râles which follow a profuse hæmorrhage, and give way gradually to a viscid crackle; and finally an occasional click, as the blood coagulates and hardens.

These sounds may be interpreted and estimated at their proper significance, in accordance with the history of the case, as regards the previous occurrence of hæmoptysis; besides this, the peculiar localities in which blood is landed by the inspiratory force help the diagnosis.

These localities are the upper lobe of the lung above the root (generally beneath the first intercostal space, close to the sternum), in the axillary region, in the fourth or fifth intercostal space, and at the base of the lung in the lower axillary region, or in the infra-mammary region.

3. The **subcrepitant rale** is a term used generally to signify a fine bubbling sound heard with inspiration, and frequently with expiration also; the typical quality of which is represented during the stage of resolution in pneumonia.

The distinction here intended between this sound and the bubbling rale is chiefly viscosity, a quality of great importance, as indicating the probable character of the exudation fluid.

As with the crepitant rale, modifications due to imperfect and incomplete formation of the sound are heard, the full sound being sometimes only brought out by the **cough-breath**.

The subcrepitant rale is heard in congestion, local pneumonia, brown induration, pulmonary apoplexy, in purulent bronchitis, and from the presence of pus or blood in the bronchioles and air-vesicles.

It is extremely difficult to decide upon the various high-toned sounds which are comprehended under this



term, and there are no defined limits between one class of sounds and another. Distinctions of diagnosis will depend upon the history of the case and the regional habitat of the sound.

4. The **bubbling râle** resembles the sound produced in blowing soap-bubbles. These râles may be coarse or fine, dependent upon the size of the bronchial tubes in which the bubbles are formed. They accompany both inspiration and expiration; in its typical form they are heard in œdema of the lung, in congestion of the bronchioles, and in capillary bronchitis.

These larger and moister sounds may also be heard under conditions resembling those which I have described as giving rise to the subcrepitant râle—namely, the presence of blood in the tubes and the liquid resulting from the resolution of pneumonia. But the distinctive character of this sound is typically heard in œdema arising from dropsy of the lungs.

In the mechanism of all these sounds the fine bronchioles probably participate; but inasmuch as the sounds have a vesicular and not a tubal quality, I have considered that the subject would be simplified by the present arrangement.

In almost all cases the significance of the râle may be tested by the examination (microscopically or macroscopically) of the matter expectorated.

#### **SOUNDS DUE TO DISEASES OF THE BRONCHIAL TUBES.**

**Tubal sounds** are those which are found in tubes, and are distinct from the previous class already described, inasmuch as they possess no vesicular quality, are continued during the act of inspiration and expiration—a simple prolonged sound in contradistinction to the repetition of bubbling sounds. They may be subdivided into two classes:—

1. The wheezing vibratile sounds of various pitch, due to vibrations within resonating tubes.



2. The so-called tubular or whiffing sounds transmitted through rigid tubes.

1. The first set of sounds have received various names, according to their quality and pitch; it will be convenient to term them—

**Rhonchi**, and it may be remarked that the pitch of a note, the result of snoring, varies from a high squeak to a deep bass; and these rhonchi may be called **sibilant** or **sonorous**, according as they are high or low pitched; and from the pitch a diagnosis may be made of the size of the calibre of the tubes affected. These sounds depend generally upon vibrating pellets of mucus within the tubes, and, unless very abundant, they can be silenced by cough.

2. **Tubular sounds** have a distinct significance of their own; both the sounds of respiration are of a blowing character, similar to the normal bronchial sound of health, and it has the quality which belongs to currents of air passing through a tube. It is sometimes called blowing respiration, or breathing. The characteristics of this sound are the following:—

In pitch the inspiratory sound is high, and the expiratory sound is still higher; and whereas the inspiratory sound is a little shorter, the expiratory sound is considerably prolonged.

The attention of the student must be particularly directed to the high pitch of the expiratory sound, and to the fact that it is much higher than the inspiratory sound. Upon this point, which is due to Flint of New York, rests the distinction between tubular and cavernous sounds.

Tubular or bronchial respiration is solidification of the lung, caused either by exudation of a firm fibrinous material into the air-cells, or by compression of air out of the air-cells by an accumulation of fluid, or by the presence of a tumour.

The usual explanation given of the sounds here described is conduction, but there are many difficulties in the way of accepting this theory; and it appears to me that



a simple and probable explanation may be given by the transmission of sound through rigid tubes.

**Regional significance.**—Tubular breathing of large extent, heard in the infra-clavicular and infra-scapular regions, may be due to pneumonic consolidation, and this is more probable if the axillary region also yield these sounds; but compression of the lung is generally met with, at the base from fluid, and in the upper portion from aneurism and tumour; so that region is not a satisfactory guide to diagnosis. If the sound be heard in the inter-scapular region on either side, the pressure of an aneurismal tumour should be suspected.

#### **SOUNDS OF DESTRUCTION.**

These sounds have peculiar characters and significance, and they ought to have their proper place assigned to them, neither in the vesicles nor in the tubes, but in those portions of lung tissue which have become altered and excavated by disease. The sounds are neither vesicular nor tubular in quality; they are of two kinds, dependent upon the presence of air only, or of air and fluid in the excavated part.

The fluid sound is a *râle*, and may be called the **gurgling rale**; the general quality is a sharp repeated crackling gurgle, with inspiration or expiration alone, or with both together, the fluid being of a viscid character; it may be heard at any time during the progress of inspiration, and is often started by the impulse of coughing. It is heard only when hollow pouchings or sacs have been formed (however small or large) during the process of excavation.

If the tissue surrounding and forming the pouch be hard and dense, and the cavity be small, the gurgle becomes a sharp rattle, resembling the rattling of peas in a bag; if, on the contrary, the cavity is large, not smaller (I think from my observations) than a thumb-nail or nut, then a distinct cavernous or hollow character modifies the sound, and this modification forms the second kind of sound resulting from destruction of the lung.



**Cavernous respiration** is a hollow deep-toned blowing sound produced in a cavity not smaller than a nut. The pitch of the inspiratory sound varies with the size of the cavity and the condition of the surrounding tissue, being raised if the communicating bronchial tube is rigid from thickened or consolidated tissue in its vicinity. The inspiratory pitch is, however, lower than that of the bronchial inspiratory sound.

The expiratory sound is lowered in pitch, and the distinction between bronchial and cavernous breathing is dependent upon this difference of pitch in the expiratory sound.

In bronchial or tubular breathing the pitch is high.

In cavernous breathing the pitch is low.

This sound is produced by the cavities of phthisis, and it is seldom to be heard under other conditions. An ampullatous or dilated bronchial tube, especially when surrounded by thick tissue from compression, for example, gives rise to a sound of this character, but the dilatation must be very large to depress the pitch of the expiratory sound. Without denying the possibility of this, I must say that I have not substantiated it yet as causing the true cavernous sound.

**Regional Significance.**—Solitary basic cavities are extremely rare, and when this sound is heard over the base of the lungs, or their lower part, it may be due to gangrenous excavation, to abscess, or to the clearance of an old pulmonary apoplectic patch. It must also be mentioned that cavities may form from the clearance of hæmorrhagic nodules, and their sites are usually the upper lobe and the axillary regions of the lung.

**Amphoric breathing** is simply a modified condition of the cavernous sound; it resembles the sound caused by blowing across the mouth of a bottle or flask; the special quality is due to the additional overtones produced by the reverberation or echo of the original sound. The conditions necessary for its production are—a large hollow formed by more or less rigid walls, with a comparatively



small opening leading to the bronchus; it is characteristic of a large body of air which is not removed entirely by the act of expiration. (Flint.)

It is heard in large excavations with thin walls and adhesions to the thorax, and is not uncommon in the elastic chests of the young.

**Regional Significance.**—When heard over the upper part of the chest it indicates a large cavity, when heard over the lower half it indicates perforation of the lung, and communication with a large body of the air within the pleural cavity, which constitutes pneumothorax.

**Metallic sounds** (metallic tinkle) have a clear high-toned metallic (silvery) quality, and resemble those produced on dropping a pin into a glass, or by pouring a few drops of water from a height into a glass bottle containing a small quantity of water. It is due to the vibration of a surface of water producing sounds in a resonating cavity; it is to be heard in some cases distinctly following the primary sound, and results from secondary vibrations. It may be produced by any agitation and movement when the conditions of the chest are suitable for its production—by breathing, voice, cough, or change of position; it may be heard in various regions, but its usual situation is half-way up the chest, on a level with the nipple.

As a rule, it must be considered as diagnostic of perforation of the lung, with air and fluid in the cavity of the pleura, either from a cavity having burst through the pleura, or from an empyema having burrowed its way through the lung.

It is sometimes, though rarely, heard in pulmonary cavity, and occasionally is simulated by the metallic tinkle of a distended stomach.

**Splashing sound** from **succussion**, or the jogging of a patient with air and fluid in his chest, is one of the oldest physical signs, and was known to Hippocrates. It resembles the sound obtained from shaking a decanter half full of water, or from rolling a cask nearly empty. It can be heard sometimes at a considerable distance from the patient, and by the patient himself. The best method



of eliciting it is to shake the patient with the arm of the observer round the body of the patient, the ear being applied to the affected side behind. It is only heard in pneumo-hydrothorax.

### **PLEURAL SOUNDS.**

The three sounds just described are, strictly speaking, pleural sounds, inasmuch as they are formed in the pleural sac; but as it was convenient not to break the thread of continuity when the sounds due to destruction of the lung were being described, they have been kept under that class.

The sounds caused by a diseased condition of the pleura alone are few and simple, and may be all classed under one head, that of—

**Pleural Friction.**—They consist of a series of superficial, to-and-fro rubs, which may be imitated by putting the stethoscope on the palm of the hand and rubbing the back of that hand with the finger.

Adjectives may be added to express the exact quality of the rub; it may be simply a graze or creaking, or grating in character.

When fully developed, the sound accompanies both acts of the respiration: when imperfect, it may be only evident occasionally, and with inspiration solely. Sometimes it is so fine as to be easily mistaken for pneumonia, and even when it is coarser it is often extremely difficult to determine whether it is within or without the lung.

It indicates roughening of the pleural surfaces which in health move freely without the slightest noise, and it results from the fibrin of pleurisy (recent or old), from cancer, and from tubercle of the pleura.

In empyema a more viscid rub, of a dull, crackling quality may be heard, formed by the cohesion of pus to the surfaces of the pleura. When heard, it is a very characteristic sign.

### **VOCAL RESONANCE.**

Certain modifications of the voice are effected by the presence of disease, and it is necessary to make the patient use his voice sometimes in various ways. Human beings can speak (articulate), whisper (words without voice), sing



and vocalise (words with the singing voice), and all these modes of using the organs concerned in the voice have to be brought into play. The normal sound of the voice through the pulmonary tissue has been described, and the modifications it may undergo are the following :—

The intensity may be increased or diminished.

**Increased vocal resonance** is due to diminution of air in the lungs, either from compression of the air-sacs or thickening of the alveolar tissue, or the presence of solid matter in the lumina of the air-sacs. But it also occurs in some cases of emphysema and of pneumothorax.

It is heard in a variety of diseases, and indicates a greater density of tissue than is natural, or a ready communication between the air and the chest-wall.

**Diminished vocal resonance**, or suppression of voice, occurs when fluid or air intervenes between the lung surface and the chest-wall, when a collection of fluid (as in abscess) is present within the lungs. In emphysema this also occurs, but it should be noted that exactly the reverse condition (as stated above) sometimes obtains, and emphysema and pneumothorax may increase the intensity of the voice.

**Bronchophony** is the intense transmission of voice sounds which occurs in solidification or compression of the lung. It is not that articulate speech is transmitted in perfection to the ear, but the voice sounds are concentrated and high-pitched, accompanied often with a metallic clang of high-toned character. Articulation may be only heard as an indistinct buzz, and the voice may be much intensified; and the definition of bronchophony must be the intense transmission of the laryngeal voice.

It is heard in the consolidation stage of pneumonia, in the compression of lung by fluid or by tumour; and its cause is probably due to moderate or incomplete rigidity of the tubes.

**Regional Significance.**—In the upper lobe it may be due to pneumonic consolidation, or to the presence of tumour, malignant or aneurismal.



In the base it may be caused by the pressure of fluid (pus, serum, or blood) upon the lower part of the lung; or, if on the left side, from aneurism.

In the inter-scapular regions, if it be heard only close to the spine, it is probably caused by enlarged glands or aneurism.

The important part of bronchophony is not the non-articulate nature of the sound transmitted, but the pitch and intensity; the pitch being high and the tone metallic.

**Pectoriloquy** must be defined as the transmission of articulate sounds, with or without the laryngeal voice, in all their distinctness to the ear; a sensation as if the uttered speech of the patient passed immediately into the ear of the examiner.

In order to assure oneself of this, it is necessary to close the ear from all external sounds in order that the speech of the patient may not be conveyed from outside; and if the single stethoscope is used, the free ear must be stopped.

The difficulty, however, of stopping all outside sounds makes the use of the whisper more valuable, and, moreover, fremitus does not occur as with the full voice.

Pectoriloquy may result from the presence of a moderate-sized cavity with smooth walls, with a direct and somewhat large communication with a bronchus, or from a solidified lung; the mere transmission of articulate speech is no arbiter between these two conditions, and the only qualities that can be trusted is the pitch of the voice.

If it be high-pitched, of a clanging character, with a metallic silvery quality (like the tone of a silver cornopean), it is due to solidity; if, on the contrary, it be low-pitched, hollow, and echoing in quality, it is cavernous.

In this differentiation of pitch the whisper aids considerably, in cavities being especially hollow in character.

Pectoriloquy is probably the result of transmission of sound through large air-containing channels with rigid walls.



**Ægophony** is the addition of a sound of tremulous or bleating character to bronchophony. It receives its name from its resemblance to the bleat of a goat, and can be imitated by playing in schoolboy fashion on a comb covered with a piece of thin moistened paper.

It may be elicited by vocalisation (articulate words with the singing voice), and appears to depend upon the free transmission of vibrating sound along a rigid tube, additional vibration being caused by moistened membrane.

It used to be considered pathognomonic of fluid in the pleura; but it may be heard when the surfaces are only slightly moistened, and in the lower part of a large cavity kept moist with secretion. It certainly is diminished in some cases by alteration in the position of the patient, and this probably results from the separation of the lung from the chest-wall by gravity. I observed this in a case of empyema in the left inter-scapular region; the pus had been evacuated through the lung, and no fluid was obtained on introducing a fine trochar; the agophony, which was marked over the left bronchus in the upright position, disappeared when the patient was prone on his belly. It is heard above the level of fluid when the surface of the lung is only separated by a slight interval from the chest-wall, and its usual habitat is in the inter-scapular region of either side. It may, however, be heard elsewhere, in the axillary region. It is a doubtful sign, and indicates density of lung with some vibrating moistened surface.

**Cough sounds**, sometimes called tussive signs, result from making the patient cough. In every examination this is a very necessary proceeding—necessary for the purpose of dislodging any temporary obstruction from the tubes, or of forcibly inflating the air-vesicles, and thus separating the agglutinated surfaces or clearing the lumina choked with exudation.

For this the patient should be instructed to cough, and draw a deep breath immediately afterwards, and to this



I have given the term **Cough-breath**, which sufficiently indicates its meaning.

By this method of examination we often succeed in detecting a crepitant râle of important significance, which otherwise would remain obstinately silent.

In cases of large cavity the fluid often accumulates, especially during the night, so as to block up the entrance of the bronchus which supplies the air, and sometimes to such an extent as completely to fill the cavity. Under such conditions the only sign that may be at first present is absence of murmur (silence) and dulness of percussion; but as soon as the cough forcibly aërates the fluid, a crackle—sometimes an explosion of painful intensity—is heard with great distinctness.

Trabeculæ in large cavities often vibrate in a remarkable manner with the impulse of the cough, and sometimes a distinct twang is thus produced. This sign is of value, inasmuch as strings traversing a cavity cut up the cavernous sound, and suppress it.

This twanging cough is also heard when the whole lung is excavated and traversed by long trabeculæ.

A brassy vibrating cough is heard in chronic inflammatory conditions of the mucous membrane, with a relaxed condition of throat and vocal chords; and in less intensity, accompanied with a ringing high-pitched metallic clang, it is heard in irritable stomach with gastric cough.

The sound of cough is sometimes, although not always, transmitted through solids; it is generally suppressed by fluids.

There still remains one other sound to be mentioned—the **bell sound**—which is elicited over a large air-containing cavity by striking together two coins placed upon one side of the cavity while the ear is applied to the opposite side; this is sometimes of a very sonorous character.



## CHAPTER XV.

## PHYSICAL SIGNS OF DISEASES OF THE RESPIRATORY ORGANS.

HAVING now discussed the various signs which the physical examination of the chest discovers in health and disease, we may group them together in order to show how special pathological conditions may be detected by their means.

The student is urged to omit no opportunity of making himself fully acquainted with the macroscopical appearances of pathological conditions; and I would entreat him to remember that he may never have such opportunities of observation afforded to him after his student days are over, when his time is taken up with daily visits to the sick, and he has little leisure and no facilities for making observations on the dead. Hand in hand, then, with clinical study let pathology be conducted; and, after some knowledge of the elements of auscultation has been acquired, it is always advisable to keep in the mind's eye an imaginary picture of what would be found if the actual condition of the parts examined was exposed to view.

All observations made at the bedside should be entered in a book, and it is a very good plan to write out the whole account in full at the end of the day. This is a method well calculated to insure accuracy of description, and to strengthen the memory of facts.

*Forsitan hæc olim meminisse juvabit.*

In the following description of diseases, with their appropriate signs, symptoms, and pathology, I shall in certain measure adopt the alphabetical order, which favours a simple arrangement:—

**Asthma—Symptoms.**—Spasmodic attacks of dyspnoea of an aggravated form, occurring usually after midnight,



by which the patient is compelled to resort to means for regaining breath—sitting up, throwing open the windows, forced expansion of the chest. The chest in spite of efforts is stubbornly fixed, and noisy stridulous breathing is to be heard some distance from the patient.

The disease is especially hereditary; besides which form is that produced by irritating vegetable matter—hay asthma. It also occurs from heart disease.

**Pathology.**—Inasmuch as asthma only ends fatally when complicated with other pulmonary structural changes which destroy the muscular contractility and induce changes in the tubes, no description can be given of the appearances due to asthma alone.

**Signs.**—**Inspection** during the attack shows very laboured movement of the thorax; the sternum thrown forward, the clavicles raised, and all the muscles on the stretch—the exertions of the individual being directed to the forcible expansion of the thorax; intercostal spaces depressed from imperfect expansion of the lungs; supra-clavicular fossæ much deepened; the epigastrium sinking in.

**Percussion** note is not as clear as in health, and generally the resonance is diminished.

**Auscultation.**—Absence of respiratory vesicular murmur, the air not reaching the lobules; harsh hissing sound (due to dryness and contraction of the tubes) of peculiar character heard all over both sides of the chest. Asthma is detected between the attacks by this dry hissing sound—the dry sibilant rhonchus, with imperfect vesicular murmur. The character of the sound is pathognomonic of the disease.

It should be remembered that undeveloped forms of asthma are innumerable. Spasmodic and night coughs, night dyspnoea, are usually traceable to hereditary asthma.

**ATELECTASIS**, or imperfect expansion of the lung, is the condition present during foetal life, and after birth expansion may be incomplete, and the atelectasis con-



genital, or it may result from some pulmonary disease, causing obstruction of the air-passages. This was pointed out first by Sir James (then Dr.) Alderson.

**Symptoms** are those due to impeded respiration, dyspnoea, and lividity of surface; the child refusing food, peaking and pining, finally dying from apnoea or convulsions. It is a sequela of acute bronchitis and of measles, both lungs sometimes being affected at their bases.

**Pathology.**—Dark purple patches of a wedge shape are observed on the surface of the lung, collapsed and depressed below the rest. They sink in water, having no air, the vesicles being glued together, and not with the granular surface of pneumonia, for which condition this used to be mistaken. A thick viscid plug may be generally detected in the supplying bronchus.

**Signs** are those due to condensed tissue.

**Percussion** over the part affected, dull.

**Vocal resonance** increased and bronchophonic.

**Auscultation** shows absence of the vesicular murmur and tubular breathing.

In the neighbourhood the lung is distended, and gives rise to signs of emphysema.

**ACUTE BRONCHITIS—Symptoms.**—Tightness of the chest, a sense of constriction, as if a band was tied round it, pain and soreness behind the sternum, oppression and weight in breathing, dyspnoea, cough, expectoration of clear frothy mucus, occasionally streaked with blood if there be much vascular congestion.

**Pathology.**—The initial stage is shown by congestion of the vessels distributed beneath the basement membrane of the bronchial tubes. This is followed by œdema and infiltration of serous fluid into the basement membrane, which becomes thickened and swollen. Finally, desquamation of the columnar epithelium takes place, followed by secretion of catarrhal mucus from the surface of the tubes and the mucous glands.

**Physical Signs.**—Inspection only conjectures the



disease from the amount of cyanosis, or blueness of the skin. The thoracic veins are sometimes enlarged if the amount of disease be great. If severe, the thoracic movements are fuller and more frequent.

Percussion gives no decided indication.

**Auscultation** elicits rhonchi, dry or moist, according to the stage of the disease and the amount and quality of the secretion. They are sonorous, low-pitched, snoring, or cooing sounds, if the large tubes only are involved; but sibilant, whistling, high-pitched, if the small tubes are concerned.

The points to which attention should be directed are—the size of the tubes and the extent of lung involved; the viscosity or fluidity of the secretion, and its amount.

**Capillary bronchitis** is of more serious import than inflammation of the large tubes. It occurs often in childhood, and is detected by the bubbling râles, generally at the posterior bases.

**CHRONIC BRONCHITIS.**—**Symptoms** are less urgent and severe than those of the acute disease; principally cough, dyspnœa, tightness of chest, and more or less viscid, sometimes purulent, sputa.

**Pathology.**—Long-continued and repeated attacks of inflammation lead to peribronchial thickening, irregular or ampullatous dilatation; there is then considerable hypertrophy of the muscular coats of the tubes, the longitudinal fibres being especially well-marked; emphysema is sometimes present.

**Physical Signs.**—Inspection gives no evidence unless the dyspnœa is marked. Percussion may give a muffled sound if there be any distension from emphysema, and this will be detected by percussing lightly; if the tubes be much dilated, a sound of greater intensity, almost amphoric in quality, may be obtained.

**Auscultation** elicits harsh bronchial blowing sounds, with dry or moist sonorous rhonchi, which become tubular if there be dilatation.

The voice sound varies very much, deficient and buzzing



in some cases, bronchophonic and pectoriloquous in others.

In these cases the heart must be carefully examined for signs of pressure on the right side, pulsation in the lower sternal region, the apex beat being obscured.

**BRONCHIECTASIS**, or dilatation of the bronchi, occurs as a result of chronic bronchitis and asthma, where the tubal structure has suffered from atrophy of the mucous coat and thickening of the fibrous layers. It is produced by inhalation of irritating particles of dust, fluff, wool, iron, clay, and stone; and it gives rise to forms of disease named according to the special trade by which it is induced—as knife-grinder's rot, filer's phthisis, &c.

**Symptoms.**—Much dyspnoea and expectoration, the sputa being plentiful, opaque and purulent; cyanosis is usually present, the right ventricle of the heart being eccentrically enlarged.

**Pathology.**—The bronchial tubes are found much thickened, irregularly pouched and dilated; alveolar walls thick, white and emphysematous. Sometimes the upper lobe is in a condition of alveolar thickening, the air-sacs irregularly dilated; while in the lower parts may be found old congestive patches of pigmented thick fibroid tissue, indicative of partial lobular congestions and hæmorrhage.

Occasionally dilatation of the tubes is observed in compressed portions of the lungs, in empyema or pleuritic effusions. It happens especially in those chronic cases where the fluid has been evacuated by natural or artificial means. A compressed lung is very loth to expand, and if the external pressure be removed the tubes will yield to the internal force of the atmosphere, and it is common to find this pouching near an empyema.

**Signs.**—Inspection observes depression of the intercostal spaces with fixity, and even retraction, of the walls of the chest.

**Percussion** generally gives a dull or muffled note super-



ficially, which may be more tympanitic with a full stroke.

**Voice sounds**, sometimes buzzing, sometimes bronchophonic, even pectoriloquous. **Fremitus** increased.

**Auscultation** shows absence of true vesicular murmur, the quality of sound being bronchial or tubular. The pitch is high, in accordance with the thickness of the alveolar tissue and the tension of the air-sacs. If the dilatations be large, the tubular sound may be almost cavernous; but the pitch of the expiratory sound is not low, although it may be very nearly of the same pitch as the inspiratory sound.

A hollow blowing sound is produced in the case of dilated bronchus from compression, and although there is often great difficulty in determining this condition, the pitch of the expiratory sound has not been, according to my experience, lowered. Moreover, the position of the sound at the base of the lung, with empyema or effusion, would indicate the probable condition of the lung. If this occurs on the left side, great resonance may be superadded by the proximity of the stomach, which is sometimes considerably dislocated upwards in such a case, and may lead to a false diagnosis.

**PLASTIC BRONCHITIS** is a peculiar and rare condition. I have only observed it associated with scrofulous phthisis; the chief notable characteristic of the disease is the expectoration of firm cheesy matter.

**Pathology.**—The tubes are distended and irregularly pouched, chiefly near the periphery of the lung, and are firmly stuffed with a hard curdy material which is formed of cells and plastic matter, apparently derived from a peculiar condition of the bronchial membrane.

**Signs.**—None can be given of a sufficiently distinct character to determine a diagnosis, but they are such as arise from consolidation within the tubes and lobules—suppression of vesicular sounds, with dulness of percussion.

The expectoration of the casts, according to Walshe,



is preceded some hours by dyspnœa and hacking cough.

This condition must not be confounded with that in which expectoration of **fibrinous casts** of the tubes takes place subsequent to an attack of hæmoptysis. It is true that expectoration of such blood casts does not often take place, inasmuch as the blood remains behind; but it is to be found in the lungs after death, if only the observer knows what to look for and where to look for it.

The following case related by Graves in his "Clinical Medicine" (vol. ii. p. 146) is worth quoting:—"A gentleman, who had been ill for some days before, was attacked with hæmoptysis. For this he was bled, and in about three hours afterwards he became collapsed, almost asphyxiated, and struggling for life; the right side of the chest expanding and contracting energetically, the left almost fixed and motionless. Dr. Stokes, who saw him, immediately changed his position, and gave him a glass of wine, when he made one more effort, and violently expectorated a coagulum consisting of fibrin, in some parts nearly colourless, forming a complete solid mould answering to the left bronchus, and its ramifications down even to some of the minuter tubes. After this he rallied."

Flint, in his work on "Phthisis," records two cases of a similar kind (p. 105). In one of these there was extreme obstruction of the tubes, and death took place. The other occurred in the person of a medical man, who suffered from several successive attacks of hæmoptysis. On the third day after a copious attack he began to expectorate bronchial casts, composed of fibrin and shrunken blood globules. Before the expectoration the patient had experienced some dyspnœa, but this passed off, and he recovered his usual condition.

**CIRRHOSIS OF THE LUNG** was a term first given by Corrigan, in 1838, to a peculiar one-sided sclerous condition of the lung.

**Symptoms** are slow and insidious. The disease begins with pleurisy or pleuropneumonia, and is characterised



by dyspnoea, dry cough, and gradual emaciation. Occasionally attacks of bronchial catarrh are present. The history of the symptoms must be considered as yet incomplete.

**Pathology.**—The appearance of the lung is peculiar and distinct. One lung only is affected; it is much dwindled and glued to the upper part of the axillary region with firm thick adhesions.

In substance it is so hard and tough that it almost creaks on section, and in appearance it is of a mottled or streaky reddish grey. The bronchial tubes are much thickened, and may be dilated into small cavities, besides which there may be excavations.

**Signs.**—These are, in advanced conditions, unmistakable. **Inspection** shows a flattened hollow chest over the part affected; the shoulders depressed; the ribs flattened; the heart drawn up, and towards the diseased side; the opposite side expanding freely, the affected side fixed.

**Percussion** gives marked dulness over the diseased lung, and high-pitched vesicular resonance over the healthy one. I have known this extend four inches beyond the mid-sternal line.

**Palpation** indicates the dislocation of the heart upwards, and towards the nipple; the heart being uncovered and evidently pulsating over a large area. Vocal fremitus increased.

**Auscultation** gives absence of vesicular murmur; tubular breathing, with or without loud bubbling rhonchi.

The illustration of the form of alar chest was taken from a case of like kind, the disease being in the left lung. Tricuspid murmur is heard in these cases.

**CONGESTION OF THE LUNG** occurs from stasis of the pulmonary vessels, producing hyperæmia in the part affected.

**Symptoms.**—Difficulty of respiration, with more or less dry cough; or in advanced stages of the disease, sputa streaked, or even abundantly mixed, with blood.

It is of common occurrence, and arises from failure of



the heart to pump the blood through the lungs, due to feebleness from age, fatty degeneration of the heart, overgrowth of the body and arrest of cardiac development; from fevers, from cold, diseases of the heart, especially those which affect the competency of the tricuspid valve; from purpura, scurvy, and hæmophilia; from cirrhosis of the liver; from copious beer-drinking, opium poisoning, and from a general constitutional tendency to hæmorrhage; and from irritation.

**Pathology.**—Portions of the lung are seen of a dark red or purple colour, gorged with bloody fluid, the tissue sodden and swollen. The portion affected may contain no air, and then it sinks in water; or on section vast quantities of frothy, bloody serum pours from the cut surface, sometimes in surprising quantity. The ordinary habitat of this condition is the middle of the right lower lobe, close under the angle of the scapula.

**Physical Signs.**—Inspection gives no indication, except as far as the air does not enter the part affected, and that portions of the ribs remain quiet and immovable.

**Palpation** gives some increase of vocal fremitus.

**Percussion** gives more or less dulness.

**Auscultation** shows absence of respiratory murmur unless there is healthy lung tissue over the patch of congestion; much crepitation, large and rather coarse, or the signs of effusion into the air-sacs. Bronchial respiration is heard only in those cases in which the vesicular murmur and expansion of the air-sacs is obliterated by the absence of air and the presence of bloody spumous fluid in the air-sacs, and where at the same time the tubes are not filled to any extent with secretion.

This is a most important condition to diagnose, as in old people, and in constitutions debilitated by excess or broken down by previous disease, the consequences may be rapidly fatal.

**COMPRESSION OF THE LUNG** results from pressure either by fluid or solid tumour; the air being more or less expelled from the lung tissue.



**Symptoms** are those due to the condition of pressure, and will be described in their own place.

**Pathology.**—The portion of lung subjected to pressure is dense and tough; the air-sacs closely pressed together.

**Signs** are those of consolidation; in all respects simulating hepatisation. (See the remarks under the heading Bronchiectasis.)

Compression prevents the formation of tubercle in the portion of the lung affected. If the pressure be diminished, expansion is generally induced in the tubes.

**EMPHYSEMA—Symptoms.**—This disease is characterised by extreme dyspnoea of a continuous character.

The symptoms of disease arise from obstruction to the respiration, and the patient presents the appearance of one striving to increase, by all the means at disposal, the capacity of the chest. The thorax is thrown forward; the *alæ nasi* are dilated; the muscles of the neck are stretched to their utmost; the complexion is muddy and of a bluish-grey hue; the extremities are cold, the pulse feeble.

There are two kinds—the hypertrophous and the atrophous. The atrophous form of emphysema is found in the aged only, and is due to senile changes: the walls of the air-sacs are destroyed without increase in the bulk of the lungs.

The hypertrophous form is that which most commonly occurs, and is notified by a barrel-shaped bulging of the chest. The lungs are enlarged and press upon the heart, which they cover in great measure, the apex being thrown down to the right.

**Pathology.**—The emphysematous lungs are easily recognised by their whitish-grey colour and their puffed-out condition. The infundibulum is first expanded, and the air-vesicles which communicate with it dilate so that at length one large bulla or cavity is formed.

In atrophous emphysema the walls are much diminished, the whole tissue sharing in the process of senile wasting;



the air-sacs communicate and lose their divisions by forming large chambers.

Senile atrophous emphysema is not a form of disease which is often presented to observation. Hypertrophous emphysema is far more common.

**Signs.**—There is perhaps no disease of which the diagnosis is generally considered to be more easy than emphysema. Nevertheless, the opinions of authorities on the physical signs vary more than with reference to any other disease, and there are some diseases which are frequently mistaken for emphysema.

**Inspection.**—The appearance of the patient indicates persistent dyspnœa. Complexion muddy, and often blue in tint; eyes prominent, nostrils dilated; muscles of neck, especially the sterno-mastoid and the platysma myoides, on the stretch; the sternum, especially the upper part, thrown forward and very convex in outline; the clavicle thrown forward; the supra-clavicular spaces depressed and much incurved at every attempt to inspire, showing the want of elastic expansion of the lungs, the intercostal spaces depressed in inspiration, more especially the lower ones, which fall in considerably. The pulsation of the heart is seen beating below the xiphoid cartilage, which is drawn in and forms a hollow, this being due to the constant tension of the diaphragm, the whole chest being rounded and in a condition of forced inspiration.

**Palpation.**—Pressure of the fingers in the intercostal spaces gives a very inelastic sensation.

Vocal fremitus is diminished.

**Percussion.**—Deep, full percussion gives an intense resonating note, which has been called hyper-resonant invariably: this intensity of note is proportionate to the dilatation of the bronchial tubes and the expansion of the thorax. The note is slightly higher-pitched, as a rule, than the normal vesicular resonance.

Superficial percussion, especially between the ribs in the intercostal spaces, elicits a tympanitic, muffled resonance of very high pitch, which is an indication of the amount of tension in the air-sacs.



Special attention must be directed to this point, as it is generally overlooked. The upper lobe is always more affected with emphysema than the other lobes, and the pitch of the note is higher in the affected parts than those which are comparatively free from the disease; and it is only by carefully and lightly percussing over the regions of the thorax that the distinction can be made. The cardiac dulness is diminished and thrown down, because of the displacement of the heart downwards and the overlapping of the distended lung.

Although emphysema in an exaggerated form can hardly be mistaken, percussors invariably attack a chest diseased in this manner, determined to elicit a noisy note which they consider to be characteristic of the disease; and I must distinctly state that no disease is so carelessly examined and so often mistaken as emphysema.

**Vocal resonance** may be buzzing or bronchophonic, and fremitus may be diminished or increased.

**Auscultation.**—The sounds which are especially significant of emphysema are deferred inspiration and prolonged expiration: the first due to the higher expansion of the lung, the second due to its impaired elasticity. Impaired vesicular sound is also present.

The sounds are high-pitched, and the expiration is even so much prolonged as to inverse the ratio between the duration of inspiration and expiration.

There is a peculiar dry crackle heard in emphysema which has attracted much attention, especially from Laennec, who described it under the term "*râle crepitant sec à grosses bulles ou craquement.*" This sound, which has a shuffling, up-and-down character, closely approximates to the pleuritic rub, and is very perceptible to the touch. It occurs irregularly with the end of inspiration and the beginning of expiration, and to my ear is very like the sound caused by drawing a child's india-rubber balloon over the palm of the hand.

If idiopathic emphysema is present, pleurisy is not likely to be present also, inasmuch as the purely emphysematous lung appears to be incapable of pleurisy: a



peculiar characteristic of this condition (post-mortem) being the absence of adhesions. In pure emphysema then pleurisy is likely to be absent, and this sound can hardly be mistaken for anything else.

Additional signs of emphysema are the depression of the diaphragm and liver—the heart displaced downwards; the right ventricle being brought to the front, while the apex is thrown inwards, and pressure is evident on the right side, from the presence of a tricuspid regurgitating murmur.

The sounds of the heart are very much diminished by the overlapping of an emphysematous lung.

Senile atrophous emphysema is diagnosed by the weakened respiratory murmur, the impaired inspiration and expiration, and by the high-pitched note of percussion.

The student is especially cautioned against the hasty diagnosis of this disease, which results from the facility of diagnosis afforded in most cases by inspection. The diseases which are likely to be mistaken for emphysema are—

Simple dilatation of the lung from hypertrophous enlargement consequent upon extensive disease elsewhere in the lungs.

Dilatation of the lung from acute tuberculosis, a condition which is very seldom diagnosed, and which emphysema closely resembles.

Pulmonary thrombosis and embolism, leading to dyspnoea and dilatation of the lung.

Pulmonary fibrosis, with contracted lung and dilated tubes, and perhaps pneumothorax.

**FIBROSIS**, or fibroid disease, differs from cirrhosis of the lung in not being confined to one lung, and it may attack portions of the lung.

It appears to result from chronic pneumonia generally of a congestive or catarrhal type.

**Symptoms.**—The chief indications of disease are marked dyspnoea and cachexia, with occasionally abun-



dant expectoration, although that varies with the degree of bronchial catarrh present.

**Pathology.**—The lung is rendered hard and almost cartilaginous by the fibro-nucleated structure which invades the whole of the pulmonary structure. The bronchial tubes stand out conspicuously, ringed with broad white circles in the midst of iron-grey indurated lung tissue, which was compared by Addison to Aberdeen granite. The streaks of black pigment must be considered due to blood pigment, either in obstructed and obliterated vessels, or in the peribronchial and perivascular lymphatics. It is a condition which in scattered patches, especially at the base of the lung, may be attributed to old congestion or pneumonia; but in the apex form, where it occupies the upper lobe and the line of demarcation between the pigmented old tissue and the healthy, is very marked, it appears to me to be characteristic of syphilis.

An illustration, on a very small scale, is given at page 128, intended to convey some notion of the iron-grey indurated lung; it will be seen that I believe it to be syphilitic.

**Signs** are those due to induration and contraction of the part of lung affected.

**Inspection** will show flattening and fixity of the chest-wall; no expansion of that part.

**Percussion** gives a dull non-resonant note generally, but if the tubes be much dilated the resonance may be tracheal in quality, although high-pitched. I have known this condition mistaken for emphysema, a proof of resonance in the percussion note.

**Palpation.**—Vocal fremitus marked.

**Auscultation.**—Sometimes complete silence, especially if the lung be extensively adherent, and the diaphragm be drawn up. I have examined cases of this kind with the shoulder drawn very much down and complete adhesion, and absolutely no respiratory sound. If the base be free, however, the diaphragmatic action induces a current in



the upper lobe, to and fro, and then tubular respiration is heard.

Voice sounds, bronchophonic or pectoriloquous.

**Heart** very much exposed; its beat perceptible over three interspaces; sometimes a sharp systolic murmur may be heard at the base.

The opposite lung, if not affected, may show signs of distension, by a high-pitched percussion note and puerile respiration.

This condition of lung may break down, and be excavated under a condition of phthisis. When it begins to liquefy it sometimes progresses very rapidly. The sputa then assume a very characteristic green tint, owing to the black pigment with which they are tinged.

**GANGRENE OF THE LUNG** consists in the putrefactive necrosis of lung tissue, and is consequent upon pneumonia, poisoned blood, the pressure of tumours, and organic cerebral disease.

**Symptoms.**—Chiefly constitutional, indicative of great depression and prostration; much dyspnoea, the breathing being rapid and oppressed, and the expectoration of purulent sputa of gangrenous odour being pathognomonic. It is, in fact, by this symptom alone that we are sure gangrene is present. Sometimes the foetor is quite overpowering.

**Pathology.**—The disease may be general or circumscribed; it may be secondary to the pressure of an aneurism or mass of cancer; the lung encroached upon being horribly offensive, disintegrated, and shreddy; exuding on section sanious fluid, or little patches may be seen dark and soft in a similar condition. The supply of blood must be totally cut off, and this obstruction may include all the vessels supplying the part.

**Signs** afford no special indication of the disease. If it be a result of pneumonia, the sounds of crepitating and gurgling râles will be heard in the centre of the consolidated mass of lung, or if the sphacelus be connected with a bronchus there may be bubbling râles of large size



with tubular respiration. Diagnosis depends upon the odour of the breath and the sputa.

**HYDATIDS** of the lung are very rare in this country, and in the majority of cases in which hydatids have been expectorated, they have been seated in the right lobe of the liver.

In Iceland it is an endemic disease, and some patients with hydatids were brought to me when I was travelling there. The diagnosis can only be made with certainty when the cysts come to the surface, forming irregular nodular swellings in the intercostal spaces, and the presence of hydatid membrane or hooklets in the sputa is established.

**Symptoms**, similar to phthisis, and characteristic of destruction of lung tissue: pallor, emaciation, dyspnoea, cough, pain in the affected side, shoulder and arm, decubitus on the opposite side, bloody and purulent sputa followed by the expectoration of portions of hydatids or unbroken cysts, with night sweats and prostration.

**Pathology.**—Hydatids confined to the lung are rare, but less rare in the liver; in most cases they will therefore affect the right lung; the cyst being situated in the lung will press upon the external parts, the pulmonary tissue being condensed and grey.

**Signs.**—Inspection shows the side affected to be more or less fixed with unilateral bulging local, or in more places than one, and obliteration of the intercostal spaces.

Percussion note dull on the affected side, loud on the opposite side.

Auscultation gives the signs of pressure; absence of respiratory murmur, tubular breathing, and after the evacuation of the contents of the sacs, signs of cavity and gurgling.

When the hydatids pass through the lung from the liver, the general symptoms of liver disease are present, and the sputa bile-tinted; the signs then may be amphoric and metallic.

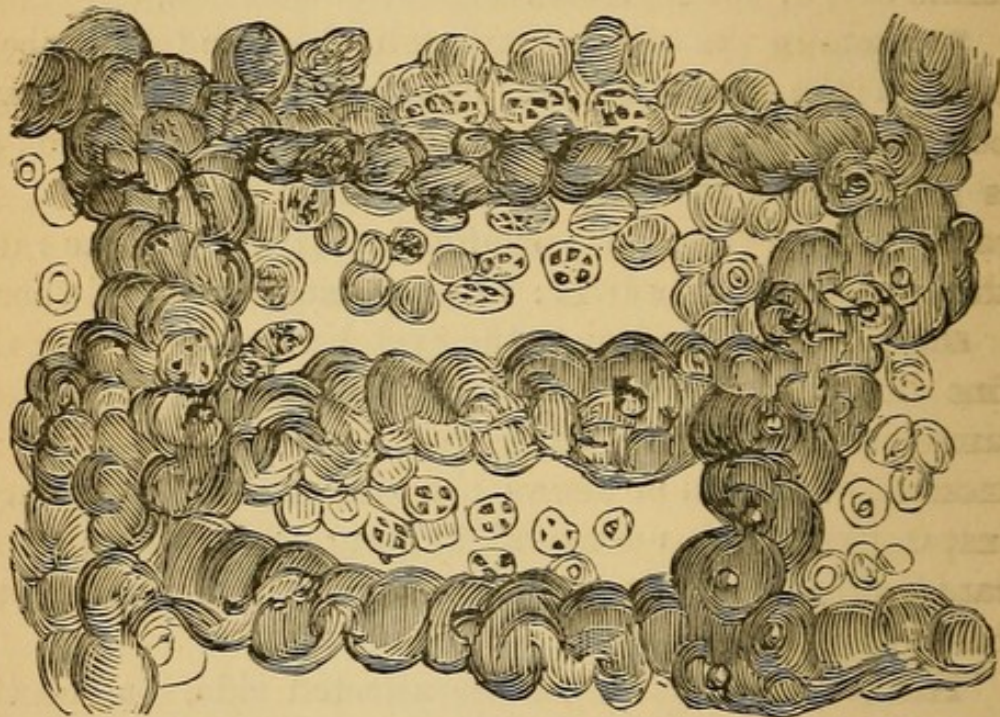


**INDURATION OF THE LUNG**, commonly called brown induration, arises from a varicose condition of the pulmonary veins, consequent upon mitral regurgitation.

**Symptoms** are chiefly due to the cardiac disease, to which are superadded dyspnœa, cough, and the expectoration of bloody sputa.

**Pathology.**—A peculiar dense tough condition of the lungs is presented to view of a dark brick-red colour: the condition is diffused over the lung, but according to my observations radiates from the root as a centre; on close inspection I have found the middle portion of the lung running across on a level with the root, chiefly thickened.

FIG. 17.



Brown Induration of the Lung.

The microscopical appearance is peculiar; the vessels are seen in a swollen and tortuous condition, the alveolar wall being also swollen; cells with blood pigment, and blood occupy the lumina. As the condition is not represented in most books, I have added here a drawing from a preparation of my own.

**Signs.**—Inspection affords none of a distinctive cha-



racter. Percussion gives marked dullness, and the region over the second interspace is that which is most dull.

Palpation, fremitus increased.

Auscultation shows density of tissue; tubular blowing respiration, absence of vesicular murmur, intense high-pitched bronchophony, and even pectoriloquy. I have known this condition mistaken for cavity.

**ŒDEMA** of the lungs takes place as the result of general dropsy, chiefly from renal disease, but it is associated also with passive congestions arising from bronchitis, cardiac disease, and adynamic conditions from fever.

**Symptoms** are by no means distinctive, and only suggest respiratory disturbance common with many other forms of pulmonary disease.

**Pathology** is very characteristic; the lung being soaked in watery fluid without any colour, and the sacs being completely infiltrated so as to be airless. The lung is generally of a light grey colour, not crepitant, and easily pitting (like an anasaruous leg) on pressure with the end of the finger; it sinks on being placed in water.

**Signs.**—The characteristic sign of this condition is the fine bubbling r le of a very liquid character, and sometimes resembling very closely the fine crepitation of pneumonia, but this bubbling r le is heard both during inspiration and expiration; it is also very regular and rapidly repeated. Its usual situation is the posterior bases of the lungs.

The palpation and percussion give variable sounds, and are not distinctive of the condition.

**PULMONARY APOPLEXY** is the term applied to extravasations of blood into the pulmonary tissue, with laceration.

It commences as a congestion, the vessel giving way from the *vis a tergo*, the intra-vascular pressure, the walls being softened by disease.

**Symptoms.**—The most important is h moptysis, which may occur in any quantity from mere streaks and specks



to very large quantities. Dyspnœa, cough, a dull, heavy pain, and discomfort on breathing, are general complaints in this form of lung disease. It used to be attributed solely to heart disease, but it may follow congestion from cold, if the vessels be overloaded; it is not uncommon with cirrhosis and in beer-drinkers, and it may result from stasis of the blood due to some derangement of the circulation in debility of the heart.

It may occur without any blood being spat out.

**Pathology.**—The appearances have been admirably described by Laennec; a patch of some size, resembling damson cheese, is seen, the lung tissue being swollen and filled with dark, black clots of blood. In old patches of some years' standing the patch becomes indurated and pigmented, the blood either clearing out and leaving a cavity, or losing its hæmatin and becoming of a firm, white fibrinous character. The position these patches affect is generally the middle of the right lower lobe, or sometimes the corresponding part of the left lung.

**Signs.**—Inspection shows imperfect expansion of the part affected.

Percussion gives decided and circumscribed patches of dulness.

Palpation perceives increased vocal fremitus, but this is a variable sign, and is dependent upon the amount of healthy tissue intervening between the patch and the chest-wall.

Auscultation shows absence of breath sounds, with occasional subcrepitant râles, increased by the cough-breath or bubbling râles if the blood be very recently effused and in some quantity.

**PULMONARY HÆMORRHAGE** occurs chiefly from the pulmonary arteries; the bronchial vessels cannot be considered as giving rise to hæmorrhage, except in congestion of the tubes and in the hæmorrhagic diathesis. To this statement there are very rare exceptions—as, for example, bronchial aneurism. Initial pulmonary hæmorrhage, according to my opinion, generally arises from structural



weakness of vessels consequent upon a certain diathesis. Initial bleeding sometimes occurs in very large quantities. It is also found in beer-drinkers, the vessels being overloaded and softened.

Secondary bleeding, after pulmonary disease has been established, may occur from congestion, ulceration of vessels, or strain upon weakened vessels.

Terminal bleeding occurring after the formation of a cavity, and often proving fatal, is generally due to the formation of a pulmonary aneurism within a cavity, or pushing through a bronchus, and to laceration of a vessel.

**Symptoms.**—The expectoration of blood. The colour of blood in hæmoptysis is generally of a bright red, and this colour is observed when small pulmonary arteries have given way; but if a large branch of the artery is ruptured, and the blood is delivered in large quantities, so as to have little chance of aëration in transit, the blood ejected is of a dark purple colour. Retained blood is always dark.

**Pathology.**—In the hæmorrhagic diathesis the lungs may be uniformly congested and in a highly-vascular condition, but in most cases the bleeding is either traced to an obtruncated vessel by the action of excavation, or to the formation of a pulmonary aneurism within a cavity or bronchus. These vary in size, from a hemp-seed to a Maltese orange.

Pulmonary apoplexy is another cause of hæmorrhage.

**Signs.**—There are no signs which give indications of approaching hæmorrhage, nor of its occurrence at the time. Cases have been reported in which pulsations and murmurs of a peculiar kind were detected, due to pulmonary aneurism, but such a case has never come under my notice, and more evidence is required on this point.

**Results of Hæmorrhage.**—These are extremely important, and have been strangely overlooked. Blood ejected in any quantity is likely during the act of passage to induce dyspnœa; the next gasp, or simply the act of breathing while the blood is flowing, lands a quantity of the blood (which is welling up in the tubes) into the



alveolar tissue. In cases of fatal bleeding patches of blood, as large as a thumb-nail, are found in the sound or opposite lung. These patches occupy certain regions under the special influence of inspiration; the upper lobe first, and in greatest quantity; then the middle of the base, or the anterior border of the lung; and, lastly, the axillary region, near the nipple.

**Symptoms.**—Dyspnoea and cough may result from this, and if the invasion of the lung be very great, emaciation follows. In many cases no result may be observed, and patients may survive the event and die of some other disease; or the nodules left from the blood may soften under septic influences, clear out and form cavities, and lead to all the symptoms and signs of ordinary phthisis.

A condition of emphysema sometimes follows, from the contraction of the nodules and compensatory expansion of the lung in the vicinity.

**Pathology.**—Hard, ivory-white fibrinous nodules are found of different sizes, from a pea to a thumb-nail, in the localities above mentioned; these soften and liquefy, ultimately leaving cavities which can be with difficulty, and only sometimes, distinguished from ordinary phthisical cavities.

**Signs.**—These are to be considered from the aspect of chronicity or the previous date of the bleeding.

If a patient applies soon after the attack, the presence of blood may be detected in the lung. This was known to Laennec, who describes the sound with his usual accuracy; but it must not be asserted that the blood started from that place—it is simply there.

Percussion and palpation give no clue to its presence in this early stage.

Auscultation shows the presence of a fine bubbling r le (or subcrepitant r le, if some little time has elapsed) during inspiration and respiration, and the situation is above the root of the lung (as Laennec knew), generally close to the sternum under the clavicle.



If the quantity of blood is very profuse, these signs may be found in the axillary region, near the nipple, or near the anterior lower border. All these regions should be invariably explored for their clues.

If some time has elapsed since the bleeding the moist sounds disappear, gradually becoming more scanty and viscid, until at length only a viscid click is heard. This is heard in very circumscribed patches, which is pathognomonic of the condition.

Percussion then begins to give evidence, and small patches of dulness can be made out.

Auscultation shows imperfect expansion of the patch, the vesicular murmur being exaggerated in the vicinity, the delivery being impeded, and jerky, free expansion being stopped by the nodule.

**Inspection** then detects a shrinking in of the soft parts and imperfect expansion of the lobe.

If the condition of the patient deteriorates, these nodules soften, giving rise to characteristic hard crackling râles, very high-pitched (like rattling peas), and eventually the signs indicate that a cavity is being formed, high-pitched gurgling râles being heard.

In this manner disease is produced which in all respects closely simulates ordinary phthisis, and in advanced conditions can with the greatest difficulty be determined clinically or pathologically.

It remains to say that tubercle may result, although the exact conditions necessary for its production are open to doubt.

In many cases of profuse initial hæmorrhage the pulmonary signs are harsh, high-pitched, imperfect vesicular murmur, which is due to the thickened condition of the alveolar tissue consequent upon the presence of blood (fibrin and blood corpuscles), which may be seen under the microscope within the walls, and by which they are stiffened and thickened.

#### **PULMONARY THROMBOSIS AND EMBOLISM.**

—For the purpose of auscultation these two conditions,



which differ mainly in their respective causation, must be considered as identical. They both produce plugging of the vessels: a very large branch of the pulmonary artery being sometimes occluded.

**Symptoms.**—More evidence is required on this subject, but the principal symptoms are severe and sudden dyspnoea; turbulent action of the heart, from the excessive sudden strain thrown upon it by the plugging of a large vessel; marked venous pulsation in the vessels of the neck, in consequence of the yielding of the tricuspid valve and the stoppage of the pulmonary current. The dyspnoea is very severe and gasping, and may recur in paroxysmal attacks, soon proving fatal.

**Pathology.**—In both cases the vessels are plugged by adherent or non-adherent clots. In embolism the plug, if it obstructs the pulmonary arteries, is transferred from the venous system generally of the lower part of the abdomen, as in phlebitis of the lower extremities.

In thrombus the plug forms from neighbouring irritation, the presence of a large cavity with the vessel exposed in close contiguity, or even the pouching of a collateral purulent bronchus.

The wedge-shaped patches are usually due to embolism of the bronchial arteries, from mitral or aortic fringing.

If the plugged artery (pulmonary) be large, the blood supply is completely cut off from the corresponding lobules, and a pale distended lung is the result; or the wedge-shaped mass and hæmorrhage result, if the vessel be not large.

**Signs** require further clinical observations. A hyper-resonant percussion note may be detected, and auscultation will indicate absence of respiratory murmur and the pressure upon the tricuspid valve; but the symptoms must be looked to as more likely to indicate the nature of the lesion than the signs. Very little attention, it must be confessed, has hitherto been devoted to this point; the condition is, however, a rare one.

**PLEURAL DISEASES.**—**Pleurisy** may be **acute** or



**chronic**, and may be a primary disease or occur as a consequence of some other disease.

**Symptoms.**—In acute idiopathic pleurisy one side suffers, and the chief characteristic symptom is the stitch in the side. This pain, which is generally described as a sharp pain as if a knife was sticking in the side, is increased by movement and respiration. It may or may not be preceded by signs, which are seldom well-marked unless pneumonia is present also.

A short, dry cough is present, with rapid breathing, which is accelerated in consequence of the restrained respiration resulting from the pain.

The pulse is frequent and hard: the urine much in quantity and deep in colour.

**Pathology.**—There are four stages of pleuritic inflammation:—

1. Hyperæmia, or the dry stage, in which the vessels are injected and the pleura becomes dry and opaque, losing its elasticity and natural transparent appearance.

2. The stage of exudation, in which lymph is thrown out on the surface of the membrane, which becomes dulled and roughened.

3. The stage of effusion, in which a quantity of serum is poured out sometimes to a very large amount.

4. The stage of absorption followed by adhesion.

**Physical Signs.**—The movements of the pleura in a condition of health are inaudible, but when the first stage of inflammation (hyperæmia) alters the smoothness of surface and causes pain during respiration, signs become manifest.

**Inspection** shows a marked diminution in the expansion and elevation of the side affected.

**Percussion** is not altered.

**Auscultation** elicits a weak superficial respiratory sound, which is diminished in intensity according to the pain of respiration, and which is jerky in rhythm. Sometimes an occasional graze is heard, which may be imitated by putting the stethoscope in the palm of the hand and rubbing the back with the end of the finger of the other



hand. Sometimes two or three grazes are heard during the respiratory act; sometimes the sound is only to be heard at the end of inspiration and the beginning of expiration: they are quite superficial sounds and close under the ear, and are sometimes increased by pressure, especially in the intercostal spaces.

**Second Stage of Exudation.**—Inspection and percussion, as in the first stage.

Palpation often detects the vibration due to the rubbing of the two altered surfaces.

Very rough sounds may now be heard with the stethoscope. Sometimes these sounds are recognised by the patient.

**Third Stage.**—Effusion of fluid into the sac.

**Inspection.**—Bulging, to more or less extent in proportion to the amount of effusion, is observed: the intercostal spaces are obliterated and are level with the ribs, and they sink in less with respiration than those on the other side.

If the amount of fluid in the pleura be large there may be displacement of the lung, the mediastina, the heart, the diaphragm, and liver.

In effusion of the right side the liver may be depressed, the right hypochondrium being prominent and the margin of the liver lower than natural. It is necessary to remember that the liver may be morbidly enlarged, a condition which will interfere with the diagnosis of effusion. If the effusion is very great the mediastina and heart may be thrown over to the left.

In effusion of the left side the apex of the heart is thrown over towards the right, and is lower than usual.

Measurement sometimes gives an increase of size for the healthy side, but inspection will show whether the bulging is local or general.

**Palpation.**—Vocal vibration is generally obstructed by fluid, while above the fluid it is perceptible.

If the effusion be very great, the perception of fluctuation may be transmitted by carefully percussing in an



intercostal space with one finger, a finger of the other hand being placed some little way off.

The cardiac impulse is sometimes transmitted.

**Percussion** gives distinct evidence from dulness proportionate in degree and extent to the quantity of fluid present.

Resonant tense percussion is found above the fluid. This is explained at page 71.

Auscultation gives negative signs: no respiratory murmur, and friction seldom is detected; it occurs sometimes above the fluid if the layer be very thin.

Often at the upper part, at the upper limits of dulness, ægophony may be heard.

If there is much compression of the lung, high-pitched bronchial respiration (the expiratory pitch being very high) is heard, and may be even heard through the layer of fluid.

It is a mistake to suppose that sounds cannot be heard through fluid: sounds are diminished by passing from one medium to another, but not necessarily obliterated.

Ægophony is usually heard close to the angle of the scapula, the lung being solidified or pressed to the back, and when there is great compression of the lung from very large effusion loud bronchial breathing may be heard all over the chest, although a mass of fluid be interposed between the lung and the ear.

Over the sound lung the respiratory sound is loud and puerile, and the sound of bronchial respiration may be transmitted at the back for a distance sometimes of five inches from the spine over the healthy lung.

**Fourth Stage.**—Absorption.

In the process of repair the fluid becomes gradually absorbed, and the parts tend to return to their original positions. The distension of the side becomes less: this may be estimated by careful measurement with the tape, or, still better, by means of the cyrtometer.

As the fluid is absorbed the respiratory murmur becomes more audible, and when the two surfaces meet



the sound of returning friction is again heard for a short time.

Even after the fluid has disappeared, marked dulness of percussion may remain for some time, with increased vocal resonance and bronchial respiration.


This is proportionate to the amount of pressure.


I must express my opinion, founded upon post-mortem observation, that compressed lung, the result of effusion, seldom returns completely to its normal condition, inasmuch as adhesion takes place before the lung has recovered.

The usual result of effusive pleurisy is adhesion, and consequently impaired, if not completely annihilated, action of the ribs to which the lung is adherent.

Absorption is noted just behind, near the spine, and in front from the infra-clavicular region; the last part to be free being the lower axillary portion.

Damoiseau has noted that the line of the fluid forms irregular curves, which become smaller and smaller as the fluid retires, assuming a parabolic form.

Occasionally a peculiar  curve is noticed which appears to be dependent upon the lower line of the lung, which is pressed upwards.

The  curve, which has received much attention, and has been well described by Garland in America, appears to be formed only when a dropsical effusion into the pleura takes place; it is rare, inasmuch as the lung must not be bound down, or the curve will be altered. The notion that the elasticity of the lung supports the column of fluid is fallacious.

**Decubitus** in pleurisy has been much insisted upon by writers as invariably on the affected side. This is very far from true, inasmuch as the side may be full of fluid, and the patient may lie on the opposite (sound) side. Decubitus appears to depend upon concomitant conditions of disease; in phthisis the patient invariably lies on the sound side, and if pleurisy supervene the rule is still observed, whatever be the amount of fluid effused. If



the amount be small and the pleurisy idiopathic, decubitus is on the affected side.

**Blood** in the pleura is of rare occurrence, except after mechanical injury, or concurrent with the presence of malignant disease in the chest. I have seen a case of simple bloody extravasation followed by coagulation and the permanent presence of blood in the pleural cavity; the man dying two years after the extravasation of blood from the results of a surgical operation for other disease.

**Pus** within the pleura, or **empyema**, results from a constitutional condition which modifies the exudation of a pleurisy. It may be set up in children, debilitated by previous disease or the subjects of the scrofulous diathesis; or it may occur from spinal abscess. In the adult it follows pleurisies, a consequence of constitutional dyscrasiæ.

**Symptoms** are those general to chronic pleurisy, and in addition to these are chills and rigors, often repeated, and pointing to the presence of pus; pulse very rapid; tongue dry and brown; hectic night-sweats, and more constitutional disturbance than in serous effusions. The thermometer must be frequently consulted in such cases.

**Pathology.**—Collections of pus within the pleura are generally found in the lower part of the chest. They have a tendency to burrow upwards in the axillary region, or behind along the spine, and may eventually bore a passage through the lung, being evacuated by the mouth, or a fistulous communication with the external walls may be formed, the abscess bursting externally.

**Signs.**—Inspection often shows considerable bulging of the affected side, with obliteration of the spaces; but this is caused by the pressure, and is not a distinctive condition of empyema alone. Sometimes œdema of the flank or surface may be observed, with an erysipelatous condition. These are certain indications of pus.

**Palpation** sometimes detects the frictional vibrations of pus in the pleura due to its up-and-down movement.



Vocal fremitus is never (according to my experience) completely absent, and sometimes quite as intense as that over the sound lung. I hesitate therefore at present to accept the doctrine of Baccelli that absence of vocal vibration and sound is distinctively pathognomonic of pus.

**Auscultation** occasionally detects, in early conditions and in cases where the lung is not closely bound down by a loculated or confined empyema, a very distinct and superficial crepitant rattle, much louder and more fluid than the simple pleuritic friction sound, but evidently superficial. I believe this sound to be due to the separation of the pleural surfaces, temporarily agglutinated by the presence of pus.

If there be much pressure and the lung be condensed the breath sounds may be heard distant, but tubular, through the fluid.

Ægophony is, I believe, rare in empyema, but I put these observations forward with diffidence, inasmuch as more evidence is required on this point.

When this condition is suggested, evidence should be obtained by the introduction of the perforated needle.

When the collection of pus has burrowed and evacuated its contents through the lung (which, according to my experience, happens in the upper axillary region, close under the axilla), the signs are those of pneumothorax, limited in extent because of the adhesions round the empyema.

There are then signs of air in the cavity, which are described fully under the heading "Pneumothorax," to which reference must be made.

The voice sounds then become pectoriloquous, articulate speech being intensely and distinctly transmitted over the cavity; the margin may be made out, not only by percussion but the buzzing bronchophony (indistinct voice and speech) which can be readily heard all round.

The lower lobe of the lung may yield respiratory



sounds, the upper being generally silent, only giving gurgles on cough.

[**Paracentesis** is performed by introducing the trocar into the chest. For this operation Laennec recommended the fifth interspace in front of the digitation of the serratus magnus; but the advantage of selecting so high a place for puncture is only due to the safety with which the abdominal organs will escape accidental wounding. For the purposes of evacuating an empyema, the lower the perforation is the better adapted it is to clear out the contents. Dr. R. D. Powell recommends the sixth or seventh space, about the mid-axillary line, for serous effusions. Bowditch operates between the ninth and tenth, sometimes the tenth and eleventh, ribs behind, in a line let fall from the lower angle of the scapula.]

**Pneumothorax**, as a condition resulting from phthisical perforation of the lung, will be described after Phthisis.

**CROUPOUS PNEUMONIA.**—This disease has been often described, and its symptoms, course, and physical signs much studied, and are well known.

**Symptoms.**—Rigors, with much heat of skin, rapidity of pulse; oftentimes delirium; sputa (scanty at first) of a peculiar character, rusty and streaked with blood; the whole disease having a course of something under three weeks, the acme of the disease being reached within eight days, but the complete recovery depending upon the amount of lung involved, the constitution of the patient, and other considerations.

**Pathology.**—The conditions which are presented to view in the post-mortem room are divided into stages.

**First Stage (Hyperæmia).**—The vessels of the lobe affected are found much enlarged and gorged with blood; the capillaries are seen to be very swollen and tortuous, encroaching to a great extent upon the alveoli.

The affected portion of the lung is red, increased in weight and density, and contains less air than it should do. A cut section yields a viscid bloody liquid.

**Physical Signs.**—First Stage.—The earliest physical



sign that can be detected in pneumonia is a harsh, high-pitched, exaggerated breath sound, not vesicular in character, because the elastic condition of the air-vesicles is lost. It is not often that one is called to cases until the stage of exudation has set in, and the crepitant râle pathognomonic of the disease is audible.

The crepitant râle has been often described, and no better imitation can be given than the sound derived from rubbing a lock of hair between the fingers.

It is a high-pitched, viscid, but not bubbling râle (Laennec's râle crepitant): it is the finest of all râles. It is formed by the forcible separation of the glutinous surfaces of the air-vesicles by the entrance of air during inspiration. Vesicular breathing is diminished; and the tissue being made denser by the tortuous and turgid capillaries, the respiratory sounds must be harsher, louder, and higher-pitched than natural.

**Inspection.**—The thorax preserves its usual condition, both as regards general form and as regards the intercostal spaces. Inasmuch as stitch from pleurisy often accompanies pneumonia, the affected side is not used.

**Palpation.**—Vocal fremitus is increased; the impulse of the heart is stronger than normal.

**Percussion.**—The sound is more or less dull.

**Second Stage (Red Hepatisation).**—This is caused by the consolidation of the pneumonic exudation: the infiltrated part of the lung being red and of liver-like consistency. There is great increase of weight and solidity, the air being completely drawn out; and a hepatised portion of the lung sinks rapidly in water. On scraping the surface of a cut section, plugs of exudation can be scraped out, leaving the alveolar walls thickened. One of the first things that strikes one on viewing a hepatised lung from the body is the great weight and size of the lung as compared with a healthy lung. It is also ribbed on the surface.

**Physical Signs.**—Inspection. Although in consequence of the expanded state of the sound side it may be



difficult to detect any difference between the diseased and sound side, in some cases the diseased side is enlarged, and in young people with elastic ribs, basic pneumonia may expand the part affected. This is also the opinion of Walshe, Ziemssen, and Grisolle.

The expansion of the chest is lessened on the affected side, increased on the sound side, and this is independent of the pleuritic stitch.

**Palpation.**—Vocal fremitus is much increased in intensity: the impulse of the heart is also frequently transmitted to touch and ear as a tremulous vibration.

**Percussion** generally yields an absolutely dull note, which may be confined to the lobar limits.

Sometimes the middle lobe is found distended and enlarged, taking the functions of the diseased lower lobe; in this case resonant percussion will mark out the position of the lobe. This distension of the lung, which gives a clear but high-pitched note, enables us to contrast in a very marked manner the note of solid lung and of healthy distended lung.

But although dulness of percussion note must be looked upon as generally characterising the stage of consolidation, it is necessary to point out that the density may be so great that the sound elicited by percussion may be drawn from the bronchial tubes, and a tubular or even an amphoric note may be heard.

This statement is corroborated by Hudson, by whom it was first observed, by Walshe, Niemeyer, and Flint.

It has been observed on the right side as well as on the left, and even the anterior base. It is possible that the sound may be due in some cases to the consonance derived from a distended stomach or colon, and this point requires further observation.

**Auscultation.**—Bronchophony and tubular breathing are the characteristic pathognomonic signs: bronchial respiration, voice sound, and bronchophonic whisper are heard in all their fulness in this stage.

The student must particularly study the pitch both of



the inspiratory and the expiratory sounds when a typical case of pneumonic consolidation is under observation.

Bronchophony and the sounds of solid material are shown by the raising of the pitch and in tubular breathing, the pitch of the expiratory sound is in a marked manner much higher than that of the inspiratory sound.

This character distinguishes the solid sound from the hollow sound: the sound of cavity being characterised by a lowering of the pitch of the expiratory murmur.

There is no greater stumbling-block in auscultation than the distinction between solid and hollow sounds, and long experience has convinced me that there is no other means for distinguishing between them than this, which we owe entirely to Austin Flint, of New York.

The **third** stage of pneumonia must be considered from two different views: either the disease progresses and the patient goes from bad to worse, the anatomical condition of the lung changing from the red hepatisation to the grey, or suppurative form; or, as more frequently happens, the patient recovers, the disease retrogrades, and the red hepatisation resolves, liquefies, and is absorbed.

**Third Stage (Progressive Disease).—Grey** (suppurative) **hepatisation.**

**Symptoms.**—These, so far as they can be relied on to indicate the unfavourable issue of the case, are very subtle: the tongue, the pulse rate, and respiration must be looked to and carefully watched. The pulse, as a rule, is very frequent and small, occasionally dicrotous; the tongue becomes dry and baked, and the respiration hurried and impeded.

**Pathology.**—The red solid look of the second stage becomes mottled and flecked with grey; eventually all the red colour disappears, and the lung becomes grey and pulpy; the alveolar contents liquefy, and the walls of the air-sacs ulcerate and break down.

**Signs.**—These are the same as those of the previous stage; and we can only conjecture the progressive condition from detecting localised patches, in which high-



pitched rattling râle or bubbling râle are evident to the ear; but throughout there may be an absence of such signs—almost absolute silence—dulness on percussion being the only sign of disease.

Third Stage.—**Resolution.**—Disease retrogressive.

**Symptoms** are those of convalescence, the phenomena of disease disappearing.

**Pathology.**—Absorption of the fibrinous exudation and recovery of the alveolar tissue.

**Signs.**—Of these the most important is the redux crepitation (*crepitationo redux*), which is moister than the fine crepitation of the initial stage, much less regular, and at the beginning of the process scanty and few in number. Like fine crepitation, they belong to inspiration; but as they become more fluid, and partake of the bubbling character, they are continued on to the expiration. The sign must be considered as, first, a crepitation due to the unfolding of agglutinated sacs; and, secondly, as a râle when the exudation liquefies and pours into the bronchioles. Hence it is really intermediate between fine crepitation and the subcrepitant râle. While this resolution is proceeding the breathing becomes less tubular, and gradually returns to its natural condition.

It is during this stage generally that Skoda's tympanitic note may be detected by percussing over the upper lobe, the lower lobe being consolidated—the explanation of which I have already given, and briefly is this:—The consolidated lung is increased in bulk very much when the upper lobe resolves and the exudation is removed; the pressure of the atmosphere must force the surface of the lung to meet the walls of the chest, which are thrown forward; hence the air-sacs are over-distended, and give a hyper-resonant note (see page 70).

**Catarrhal Pneumonia.**—It is somewhat strange that croupous pneumonia should have occupied so many volumes, and that catarrhal pneumonia, a more insidious and pernicious disease, should have absorbed so little attention. It is a disease of not infrequent occurrence,



the most frequent prodromal stage of phthisis; whereas croupous pneumonia is a very exceptional condition, either as a complication or as an initial stage of phthisis. In all books it is stated that catarrhal pneumonia is an extension of bronchitis, but to this statement I must take objection, if it be meant thereby that this form is simply a continuation downwards of disease travelling along the bronchial tubes.

It is essentially a disease of irritation; it arises from the effect of irritating (often damp) air on the alveolar walls, and that bronchitis often precedes only indicates (to my mind) that the tubes really filter out noxious ingredients from the air, and are defensive outworks calculated to protect the alveoli. By the frequent repetition of bronchial attacks the respiration becomes more forced, and consequently eventually the irritating particles affect the alveoli. I have watched catarrhal pneumonia, commencing in a scrofulous patient with all the acute symptoms of croupous pneumonia, passing from the stage of fine crepitation through consolidation in three weeks' time, terminating fatally after the space of two months; the alveolar tissue being thickened and invaded by cell-growth, and breaking down without any sign of croupous exudation.

**Symptoms** may then closely simulate croupous pneumonia, but in the ordinary run of cases the disease is more insidious, and follows previous attacks of catarrh and bronchitis. The main symptoms are troublesome cough, dyspnoea, and expectoration, with rapid pulse, often an absence of febrile signs, but loss of flesh and health are invariably present.

**Pathology.**—The process is one of irritation of the air surface of lobules, and whereas croupous pneumonia is lobar, catarrhal pneumonia is lobular; indeed, it is sometimes seen (post-mortem) affecting lobules here and there; intervening lobules being untouched, and forming a marked contrast by their grey appearance to the red colour of the irritated lobules.



Hyperæmia is the initial condition followed by active proliferation of the epithelium, the air-sacs being filled more or less with the pinkish-white or light red secretion. The condition may resolve, but it has a marked tendency to progress, inasmuch as the lung that gives way to such irritative disease is unable to throw it off, and is very apt to succumb, probably from delicacy of structure. If this takes place, then the alveolar walls become invaded by the cellular growth, and ultimately obstruction takes place, constituting the condition of phthisis.

**Signs.**—In acute forms of the disease, especially when several lobules are attacked, the signs may simulate very closely the early stages of croupous pneumonia; fine crepitation, rather more liquid, not so high-pitched, less rattling, may be heard, but the more usual signs result from a very insidious and chronic form. If, for example, a patient comes for examination with a history of previous colds and winter cough, we are very apt to find imperfect expansion of the lung, deficient vesicular murmur, which is impaired and more or less silent. On forced inspiration, or after the cough-breath, we hear quite at the end of inspiration a little stick, almost a graze or rub, of ill-defined character, and not amounting to a râle. This is an extremely suspicious sign, and indicates retention of catarrhal products.

If the disease progresses it may do so quite insidiously, and at the next visit (if it be after two or three weeks' interval) we may be surprised by hearing an explosion of sounds after cough, or the sound may become more liquid and lead up to the crepitant râle.

Inspection under such conditions shows imperfect expansion.

Percussion detects some diminution of resonance.

In cases of previous disease, where the lower parts of the same lung or the opposite lung become involved, peculiar patchy dulness may be detected, intervals of distended lung being clearly made out.



Auscultation then may reveal no more than a few scanty viscid clicks in the parts affected, with absence of respiratory murmur.

Prognosis in great measure depends upon the heredity of the patient, the present condition of health, and the amount of mischief. As far as signs are concerned, fluidity is unfavourable, and the pitch of the sounds helps towards forming a notion of the condition of the alveolar tissue; if there be much thickening, much consolidation, the pitch is raised, and the sounds assume a rattling quality.

If the disease retrogrades and the lung recovers, the sounds die away and the vesicular murmur gradually becomes restored, passing through a condition of imperfect elasticity, indicated by feeble vesicular murmur of a peculiar dry, and even harsh quality, in which the pitch of the sound is slightly raised.

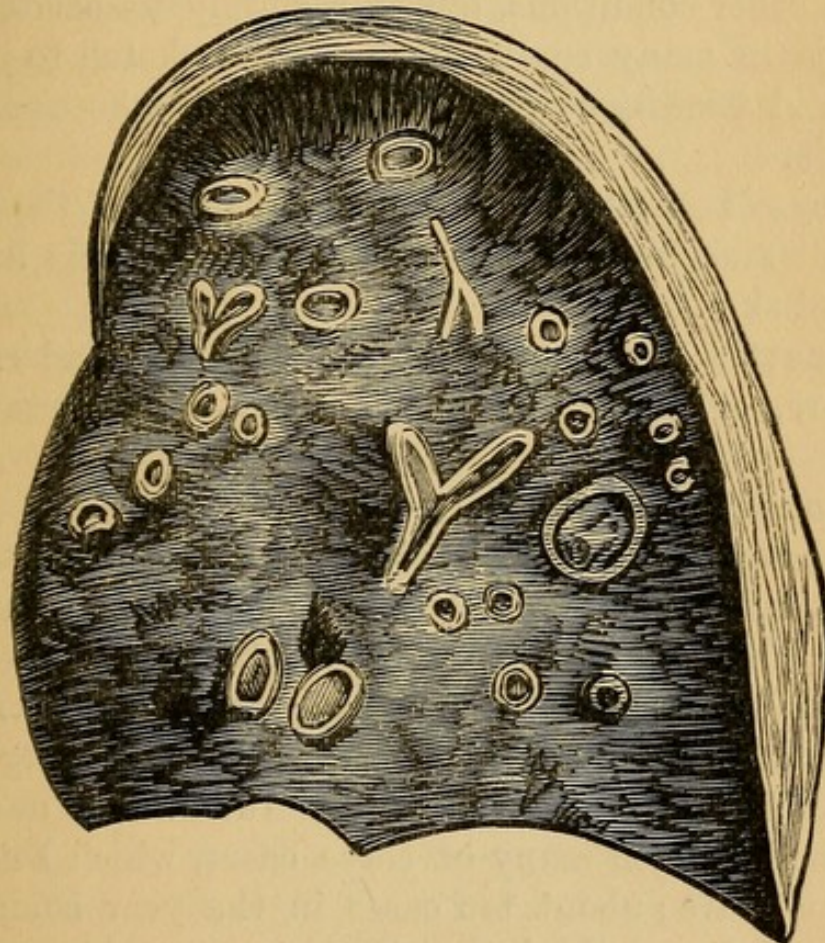
Pneumonia of this form is very apt to assume a special character in accordance with the type of constitution which it affects. In the young, cell-growth is a peculiar characteristic in all forms of disease and absence of fluidity: the air-sacs become choked with cells, which are very apt to give little indications of their presence to the ear, and, moreover, they are not expectorated; this is still more marked in scrofulous types of constitution, of which large cell-growth is a distinctive characteristic. These prevent fluid sounds, and result rather in silence than sound. On the other hand, in the adult, and especially in broken-down constitutions, the exudations have a tendency to become more fluid, and there is more sound and more expectoration. It is impossible to do more than indicate the varieties of signs under such altered conditions; they should, however, absorb much attention on the part of the student.

**Interstitial pneumonia** is a condition of a very chronic character, and, as a rule, is secondary to other inflammatory processes affecting the alveolar tissue, the bronchi, or the pleura. It includes a variety of diseases,



and the symptoms are those simply of annulled respiratory function; dyspnoea, especially on exertion, and cough. I have already described under the term fibroid disease the symptoms and signs which are to be looked for in this affection of the lung. I shall, in this place, only draw attention to a peculiar form which is due to syphilis, and which may be called **syphilitic pulmonary disease**. To a great extent this disease has escaped notice, but, after the experience of ten years, I must say that I look upon this as a specific lung disease, which is associated with no other condition except chronic syphilitic cachexia, and that it has distinct symptoms and signs of its own.

FIG. 18.



Pigmented Fibroid Lung.

**Symptoms.**—Marked dyspnoea, especially on exertion, the patient being sometimes unable to walk except on the level; more or less cough of a ringing character, with or



without expectoration, but generally there is much bronchorrhœa. As a rule, hæmoptysis of no great quantity is present from time to time. There is sometimes emaciation, but this is proportionate to the amount of cachexia. It is invariably associated with the syphilitic bronzing of skin and with tenderness on touch over the clavicle or upper ribs. It is more common in women than in men, and consequently primary symptoms are not always to be made out; but tenderness of the scalp has been, in my cases, seldom absent.

**Pathology.**—Marked increase in the connective tissue of the lung, which is most apparent in the inter-lobular septa about the bronchial vessels and the tubes. It results in the iron-grey induration, which may be found also in other conditions, but is certainly associated with syphilis in many cases. I append a sketch to indicate the solid condition which I believe may be ascribed to syphilis.

**Signs.**—Inspection gives no indication of the pulmonary disease, but suggests the general cachexia from the bronzed skin.

**Palpation**, with rare exceptions, detects tenderness on pressure, or on percussion over the bony parts of the chest.

**Percussion** gives a more or less dull note.

**Auscultation** detects a peculiar, dry, papery rustling, indicating thickening and want of elasticity in the air-sacs. This sound is pitched high, and is generally diffused over the infra-clavicular region. In marked cases there is also a high-pitched, tubular, blowing sound, derived from thickened bronchial tubes. I have now examined a good many of these cases, which I consider to be not rare; about ten cases in the year come under notice. I do not think that the signs resemble any other form of pulmonary disease, and the condition to which this syphilitic affection comes nearest is perhaps the early condition of inflammation affecting the bronchial tubes. If the condition of the two forms of disease be considered,



it will be seen that the two groups of signs are likely to resemble each other, inasmuch as in the syphilitic disease we have to deal with thickening of the connective tissue of the alveolar and bronchial walls.

In support of these observations, I must first state that they coincide with some made by Dr. Robinson, of the Fusilier Guards, from an independent point of view. He tabulated all the cases of syphilis which came under his care (and the records are most carefully kept), and noted the pulmonary symptoms and signs which followed. I have sifted out a class of cases of pulmonary disease which appear to me essentially distinct, and I find that the signs are invariably associated with symptoms of syphilis. Moreover, unless these cases are treated with anti-syphilitic remedies they never improve.

The pathognomonic distinctions are these:—The absence of moist sounds, for, although the patient often suffers from profuse bronchorrhœa, I have never yet detected fluid sounds; the general diffused condition; the absence of the usual signs of phthisis, emaciation being present only as a result of profound cachexia; the superficial tenderness of the thorax, and the bronzed colouring of the skin.

The condition remains unaltered for a considerable time, and if treated by appropriate remedies the patient soon improves, and the signs of induration disappear. But if the patient suffers from reduced circumstances (a condition not unusual in the class especially affected by this form of pulmonary disease), the fibroid induration may soften, liquefy rapidly, and give way to the usual conditions of phthisis. Until this happens, life is sustained with little else but the inconvenience of dyspnœa, arising from this affection; but if the lung is excavated, the powers of life rapidly fail, and the patient dies speedily.

It is then only under such circumstances that the opportunity is afforded of examining the lungs after death; and I believe it is only because of the carelessness



and want of interest with which cases of phthisis are ordinarily inspected, that the condition has escaped notice. The remaining tissue which surrounds cavities should be carefully inspected for indications of the primary condition of the lung, and in these syphilitic cases fibroid induration will be found to be present.

**Infective pneumonia**, or pythogenic pneumonia, is a form of pulmonary disease arising from blood-poisoning, from sewer gas and imperfect house-drains.

It has generally been presented to my notice in labourers employed in the sewers, or engaged in night-work, the emptying of cesspools, and the like.

That it is a distinctive disease I have no doubt, and characterised by peculiar physical signs.

**Symptoms** indicate the condition which is closely analogous to typhoid fever or septicæmia; rigors, sometimes repeated after an interval, heat of skin, rapidity of pulse, brown furred tongue, pungent surface, great prostration, and depression accompanied by emaciation, and a peculiar sallow look characteristic of blood-poisoning, have been observed.

I have now a case of this kind under my care, in which the symptoms and course of the disease have been very prolonged, lasting over some months, with repeated remissions and exacerbations after intervals of about a fortnight.

**Pathology.**—I have not had an opportunity of examining such a case, but the conditions observed are reported to be those of pneumonic congestion of a lobular (and not lobar) character.

**Auscultation** elicits subcrepitant râles scanty and scattered, sometimes at the base, sometimes at the apex; they appear to be formed in the terminal or peripheral lobules; they are irregular, and occur during inspiration and expiration, and have a peculiar viscid quality, suggestive of hæmorrhage when the blood is thick.

The signs, I believe, are pathognomonic, and accompany symptoms equally distinct.



**PHTHISIS.**—This disease has been kept until the last, designedly placed out of its alphabetical order, inasmuch as it must not be looked upon as a single disease, beginning invariably in the same way and continuing in one uniform progress, but must rather be looked upon as the terminus to which any pulmonary disease may converge, the vortex in which all may end. Clinical experience shows that bronchitis, congestion of the lungs, catarrhal pneumonia, pleurisy, fibroid disease, syphilitic pulmonary disease, hæmorrhagic fibrin in the lungs, may all terminate in destruction of tissue resulting in excavation and the formation of cavities. Secondary to the cavities, in my opinion, comes the infection of tubercle, which as a sequence of pulmonary disease is localised and limited to alveoli, sometimes infecting the lungs generally, as in acute tuberculosis.

Phthisis must then be defined as the disease in which the tubercular infection plays an important part, and the characteristic feature of which is the formation of cavities.

The predisposition to phthisis appears to be a special structural delicacy which is inherited from the parent, and which may be exhibited in various ways. It is not necessary, for example, that all the lung structure should be delicately constructed; it may be exhibited only in parts, as in asthma, or mainly in the lymphatics and glands, as in scrofula; or it may be shown in a special tendency to hæmorrhage without proneness to pulmonary inflammation.

The old notion, which still finds many supporters, was that phthisis began with the tubercular nodule, which, grouping together, accumulated and broke down, resulting in excavation; but the arguments that may be briefly advanced against this theory are the following:—

From pathology it can be shown that cavities form in tissue which has undergone previous inflammatory processes. Grey induration, pneumonic and syphilitic, may terminate in excavation; hæmorrhagic nodules soften, and form cavities; congestion of the upper lobe may



soften, liquefy, and form cavities. We can judge in many cases how the cavity has been formed, and we know that in the tubercular process the chief characteristic feature is the obliteration of bloodvessels; consequently softened tubercular matter does not show signs of unaltered blood.

From clinical experience it can be shown that the most frequent prodromal stage of phthisis is catarrh of severe type, frequently repeated bronchial catarrh extending to and involving the alveoli, or in more acute cases catarrhal pneumonia. The evidence of the old theory has been in great measure derived from physical signs supposed to indicate the deposit of tubercle—that is to say, by signs of consolidation, or of solid matter deposited in the upper lobe. This evidence is by no means trustworthy as a support of the theory, inasmuch as it can be shown that early deposits of tubercle (crude tubercles) cannot be detected, and do not give the signs attributed to them; nor can it with any probability be suggested that a minute nodule, or groups of nodules, when covered by a thick layer of uninjured lung, can be detected.

If it be urged that all cell action of a destructive character is to be considered tubercular, then in what does the definition of tubercle consist? This, indeed, seems to be the phase of opinion which is gradually gaining ground now—that tubercular processes may be diffuse; but to me the special feature of tubercular action is not destruction, but chronic irritation of a very slow character, in which the vessels are invaded, obstructed, and gradually obliterated. Even the breaking down of tubercular districts, which by all observers is allowed to be due to so-called necrotic processes, is not the characteristic feature of tubercle, and is in very great probability due to the same kind of septic changes which induces destruction of non-tubercular tissue. I am extremely unwilling to put forward any theories, but I cannot admit the received notions regarding the nature of phthisis, and I am bound to indicate the teachings of clinical experience, and put forward the facts which have been elicited by many years' practice.



The initial or prodromal stage of phthisis must then be considered as very variable. It may begin with bronchitis, congestion, pleurisy, catarrh, or (most frequently of all initial stages) it may begin with catarrhal pneumonia. It must not then be expected that the preliminary stage will always be represented by dulness of percussion, wavy breathing, increased vocal resonance, &c. All these may be present without phthisis, any and all of them may be only the signs of previous inflammation and induration. Such a condition indicates rather a quiescent state, and affords no evidence of any active process going on. At the same time, such a condition may yield and soften if the constitution of the patient be so enfeebled as to encourage such destruction.

No one sign, or group of signs, can be relied upon to indicate this first stage. They are variable, according to the tissue which is first attacked; if the bronchial tubes be first affected, the signs due to such conditions will prove the first stage, if phthisis follow; and the same is true of congestion with its respective signs, with pleurisy, and any other form which alters the lung and so prepares it for future destruction.

The preliminary condition necessary to prepare the lung for phthisis is an unhealthy or morbid alteration of the pulmonary (alveolar) tissue. When this has taken place, from any cause whatever, the soil may be said to be prepared for the seeds of phthisis. The sole exception that need be made to the above general law is the condition of emphysema, which is in many cases, but not invariably, proof against the destruction of phthisis. If previous attacks of disease, although successfully resisted by the constitution of the patient, so far damage the lung as to leave behind them marks or traces of their action, that lung may, under certain conditions of health, become the victim of phthisis, beginning first in the affected localities, and spreading to the rest of the lung.

If we examine a number of patients who have previously suffered from pulmonary disease of an inflammatory type, but were in a condition of health, we should find those



signs which have been generally ascribed by writers to the first stage of phthisis—signs which may be briefly described as those of indurated or cicatricial tissue, with impaired vesicular elasticity, and (in many) pleuritic adhesions. These signs are notably dulness of percussion, imperfect expansion of lung, high-pitched, slightly blowing breathing, and imperfect delivery or wavy respiration.

The following Table, taken from Pollock's very careful and accurate work on "The Elements of Prognosis in Consumption," will illustrate this statement. It contains the signs of eighty cases of incipient phthisis, observed with special care, and it represents the relative frequency of occurrence of the physical signs :—

Dulness of percussion observed in all the . . .	80
Deficiency of respiratory murmur in . . .	69
Bronchial voice . . . . .	48
Rough inspiration . . . . .	47
Expiration prolonged . . . . .	47
Mobility of chest-wall lessened . . . . .	41
Flattening of chest-walls . . . . .	38
Tubular quality of respiration . . . . .	32
Dry crepitation . . . . .	17
Wavy inspiratory sound . . . . .	15

According to the view of phthisis presented in these pages the first stage of the phthisical process is that in which destruction of tissue is threatened, if not actually started, in the tissue; but inasmuch as it is convenient to keep the nomenclature and the divisions into stages which have been sanctioned by long use, and, indeed, meet the exigencies of the subject very well (I only object to the interpretation of the signs), the subject will be considered under its three stages, the signs being classified accordingly.

The student must be forewarned that the signs of the prodromal, or first, stage of phthisis are not uniform, but that they vary according to the exact nature of the previous disease, and according to the tissue (bronchial, alveolar, or pleural) which has been injured.



1. **First Stage.**—This must then be declared variable, and not one disease, and consequently no single group of signs can be accepted as invariably preceding phthisis.

The chief pathological condition which excites suspicion is the viscid nature of the secretion, accompanied by thickening of tissue, indicative of (not a convalescence) a morbid indolence in the repair of tissue and in the removal of effete products. The distinctive features leading to an unfavourable opinion depends upon constitutional disturbance; by signs alone it would be impossible to determine from a single examination whether the viscosity was due to a healthy drying-up of fluid secretions, or a morbid stagnation and caseation; but if coupled with this condition, we find a further extension in the amount of lung involved, increased difficulties in respiration and cough, and the patient loses ground; the condition is evidently full of danger.

It becomes, then, a task of great difficulty to determine, for example, after repeated attacks of bronchitis in a patient, whether the tissue of the lungs is yielding to the disease or not.

When bronchitis is progressing unfavourably, such a downward course is not indicated by increased secretion, but rather by the presence of viscid clicks and crackles, heard only occasionally and after a forcible cough-breath, a little air having detected some too quiescent collection of material.

When phthisis follows attacks of bronchitis, the first suspicious indications are—

**Inspection**, imperfect expansion of the upper lobe, due to the unhealthy and inelastic condition of the tubes.

**Auscultation** indicates impaired vesicular murmur, which is slightly harsh and dry, and a click, crackle, or even subcrepitant r le after the cough-breath in one or two places, deep-seated.

**Catarrhal pneumonia** is so frequently the preliminary stage of phthisis that it must always be viewed as of most serious import. Inasmuch as it may occur without



proceeding to phthisis, it has to be considered as an independent condition; but if it occurs in patients debilitated by disease or by heredity of structural delicacy, the physician knows that there will be no boundary line between it and the commencement of phthisis, and will form his prognosis accordingly. In a few instances, under careful nursing, and perhaps from treatment, I have known this condition end, not in destruction but induration, and so the patient has recovered, even with phthisical antecedents; but these cases are rare. The physical signs have been already described under the heading Catarrhal Pneumonia.

**Inspection** shows also in such a case, as in the previous, imperfect expansion: the tissue is impaired, and offers more resistance to the entry of air. If expansion of the corresponding part of the chest was forcibly induced, the respiratory action would be fuller; but the ventilation of this lobe being due to a secondary current derived from the action of the diaphragm, the force is not sufficient to overcome the resistance.

**Auscultation** indicates the presence of exudation into the air-sacs by the very fine crepitant sound, moister than the fine crepitation of pneumonia, not so moist as a râle, although often inseparable in practice, which is heard at the end of inspiration—that is to say, at the time of the unfolding of the air-sacs.

This may progress onwards without a stage of demarcation to the second stage of phthisis, in which actual alveolar destruction takes place; or the secretion may become more viscid, and the sounds of crepitation may dwindle down to a crackle or click, or even an almost imperceptible stick (if I may be allowed the expression); all these become of serious import if on forcible ventilation of the parts affected by the cough-breath the air-sacs are detected in retaining secretion. Especially an unfavourable opinion must be suggested if the pitch of the sound is high, as this is a decided indication of thickening of tissue, and therefore (by conjecture) of alveolar invasion;



a most serious condition excellently described in Dr. Green's work on "Pathology."

When the preliminary stage is the relic of a pneumonic process, to which is generally added adhesion (to more or less extent) of the pleural surfaces, the signs will be those of simple induration and adhesion.

**Inspection**, as before, shows imperfect expansion; fixity of the chest, if the adhesions are thick and firm.

**Palpation** will give want of resilience, hardness of tissue, and more or less increase of vocal fremitus.

**Auscultation** indicates thickened tissue by absence of vesicular murmur and blowing breathing, the pitch of which is higher than natural.

The delivery of the sound may be jerky or impeded; the expiratory sound may be prolonged.

If hæmorrhage precedes the development of phthisis, the condition which will be found indicates either a diffused thickening of the alveolar tissue from inhaled blood, or a localised patching with fibrinous nodules.

In the first case, a high-pitched, tense, harsh sound will be heard (without any tubular respiration), not distinctive unless interpreted by the history; or, in the second case, circumscribed patches of dulness, with impeded expansion of a jerking or hitching character; high-pitched and localised tubular respiration of a feeble kind, with increase of voice sounds.

The sounds here indicated as occurring previous to the establishment of phthisis are those which are usually comprised in the descriptions given by most authors regarding the first stage of the disease. I only differ in attributing them to the results of previous conditions of disease, and in objecting to receive such signs as proof of the presence of tubercle.

With regard to the signs of the second and third stages of phthisis and their interpretation there is no difference of opinion.

2. The **second stage** is that of **softening**, which is characterised by the well-known sound which is accepted



by all observers as the sound of incipient destruction; it is the **crepitant rale**. (Walshe, moist or humid crackling.)

This sound I consider to be produced by liquid within thickened alveolar tissue, which is the incipient condition of phthisis; and is most usually found in advanced conditions of catarrhal pneumonia. If it be considered that catarrhal pneumonia is the acute form of that disease of which tubercle is the chronic form, then it will be correct to consider the crepitant r le as tubercular in character; but if it be stated that the crepitant r le is always due to a softened mass or group of tubercular nodules, this can be easily disproved by dissection. To this point I have paid the greatest attention, carefully examining the condition of the lungs; and, comparing them with the careful notes of competent observers, the most frequent pathological condition associated with crepitant r le is advanced catarrhal pneumonia; especially that form which so often attacks the previously unaffected portions of the lungs, and ushers in the fatal termination of the disease.

But it is not only with catarrhal pneumonia that this sound is associated; it may be found in any condition in which softening of indurated alveolar tissue is progressing: in congestion, in advanced capillary bronchitis, spreading downwards. In practice it may not always be distinguished, but in the majority of cases it is pathognomonic and distinct.

Second Stage.—**Symptoms.**—Emaciation and cough; the expectoration of frothy but tenacious mucus with puriform specks. In this stage the sputa should be carefully examined by the microscope for elastic tissue; the sputa being boiled with an equal bulk of caustic soda in solution (twenty grains to the ounce), and the sediment (after free solution) being examined.

**Pathology.**—The exudation products break down within the alveolar walls, and liquefaction and complete disintegration takes place. The macroscopical appearance



are those of solution of the lung tissue, minute cavities being in process of formation.

**Signs.**—Inspection shows imperfect expansion; if the process is being established after much previous disease, there may be actual flattening and depression below the clavicle.

Palpation gives no distinct indication.

Percussion gives more or less dulness; dependent upon the amount of lung involved, and the chronicity of the process. Resilience of tissue diminished.

**Auscultation** detects the crepitant râle, which is more audible after the cough-breath. Vocal resonance increased; indistinct buzzing bronchophony.

The special region for this sound is the supra-spinous fossa, where it is even more frequently detected than in the infra-clavicular region. The back regions should never be omitted, inasmuch as all moist sounds love to congregate there.

Third Stage.—**Excavation** of the lung, the continuation and extension of the process of destruction, followed by the evacuation of the contents, the result being the formation of cavities of various forms and sizes.

This stage is characterised by the **gurgling** râle, with or without the cavernous sound, according to the size of the cavity. If the cavity be large and completely empty the cavernous sound only is heard, but this occurs only when the disease is arrested and the cavity is not secreting, or has been recently emptied.

The **gurgling** râle has a sharp rattling quality, of more or less high pitch, according to the density of the surrounding tissue; it is due to air passing through a viscid fluid (derived from the walls of the cavity) and consists of a few bubbling râles of some size; more audible and sometimes only heard during or after cough. This sound may be heard during inspiration or expiration, or during both acts. It has more or less intensity, according to the size of the cavity and the quantity of air the cavity contains. It may be absent from the evacuation of the



contents of the cavity by expectoration, and it may be rendered indistinct and only audible after cough from the accumulation of a large quantity of secretion, which especially happens during the sleep of the phthisical patient.

Diarrhœa and profuse sweating have a very marked influence in diminishing the intra-pulmonary secretions; often no sounds at all are to be heard after a patient has suffered much from purging. Very small cavities may be detected by this sound—*e.g.*, rather less than half an inch across. The only condition which is at all likely (and that very rarely) to give this sound is a dilated or pouched bronchus, situated in the lower part of the lung.

The **cavernous sound** is only produced in cavities of a certain size; the smallest which I have as yet detected by its means was as big as a thumb-nail, three-quarters of an inch across, the diagnosis being verified by post-mortem examination. The cavernous sound is a hollow one, of which the special characteristic is the low pitch of the expiratory sound. This distinguishes it at once from the tubular sound, as I have already explained. Moreover, this sound (in a cavity) appears to come back towards the ear, while the expiratory sound of tubular breathing recedes away from the surface of the chest.

**Symptoms.**—All those usual in phthisis are found aggravated and intensified; emaciation, dyspnœa, cough, with more or less expectoration, according to the progress of the disease; sputa puriform, nummular, viscid, in which fragments of vessels even and small bronchioles may be visible under the microscope; anorexia, fever, night-sweats, and, if the secretion be excessive, diarrhœa, especially if the patient is not extremely careful to reject all the matter that comes into the mouth.

**Pathology.**—Cavities vary in size; they start from small beginnings, and may involve the whole of a lobe, which is their usual extent, or they may even advance beyond and include the divisions of the lobes and involve the whole



lung. It is not very rare to find the whole of one lung converted into a simple bag, from which all the tissue has been ejected, leaving nothing but a thickened pleura with remains of tissue at the edges.

**Signs.**—Inspection shows fixity of the chest-wall with marked flattening of the corresponding parts, the intercostal spaces being depressed.

**Palpation** gives no special indication.

**Percussion** gives a variable note, according to the actual condition of the cavity and the amount of air or secretion it contains.

With thick, dense walls, and a large amount of secretion (the cavity being sometimes quite full, especially in the morning) a dull note will be present. If, on the contrary, the walls be thin, and there be much air and little fluid, the note will be resonant, tympanitic, or amphoric.

The cracked-pot sound may also be elicited.

**Auscultation** gives the gurgling r le, the cavernous sound; sometimes bronchophony, often pectoriloquy; and the pectoriloquy, it must be added, has a cavernous sound—it is hollow and low in pitch.

Vocal resonance is also increased in this case.

The above description applies to the generic form of phthisis, which as a simple process of distinction may be considered as one and indivisible; but, as I have already urged, the prodromal varieties converging towards phthisis (in which all their primary features are engulfed) are extremely numerous, having distinctive characters as regards clinical history, symptoms, signs, and pathology. If the primary features of these diseases are not known and recognised, prognosis can be made on no firm basis; and if the phthisical proclivity is from the first overlooked the ultimate restoration of the health of the patient is likely to be doubtful. I must therefore call attention, first, to the varieties of cavity which belong to different forms of phthisis; and, secondly, to varieties of phthisis which are of great clinical importance.



**VARIOUS FORMS OF CAVITY.**

All cavities may be considered from that point of view in which the condition of their secreting walls is concerned. They may secrete profusely—a bad sign, as apt to terminate rapidly; and this rule may be laid down with regard to phthisis generally, that the rapidity of the case is likely to be proportionate to the amount of the secretion. The converse of this is, however, not true; a favourable result cannot be prognosed in all cases in which the secretion is scanty.

**Secreting cavities** are recognised by their gurgling râles, especially after cough; but if the secretion so accumulate as to fill the sac, absolute silence may reign over the cavity with dulness of percussion; this may be especially noticed in the morning after the accumulation during night.

**Dry Cavity.**—The fluid sounds diminish, and give place to air sounds, cavernous respiration, cavernous pectoriloquy, and tympanitic or amphoric percussion note. The amphoric note especially characterises the elastic walls of young persons.

**Contracting Cavity.**—The fluid sounds are few, and the air sounds are notable for a peculiar high-pitched whispering quality, which is easily recognised when once pointed out, and is quite characteristic of the condition. It is extremely difficult to convey any notion by words, but it may be compared in some measure to the rustling of stiff silk. Other signs there are besides this and the general improvement of the patient—*e.g.*, the retraction of the surface, the flattening of the chest-wall, and the upraising of the heart. Pectoriloquy may give way to bronchophony under this condition.

**Healed Cavity.**—This is by no means so rare a condition as many believe; it takes place most completely in the young. The chest is flattened, the thorax immovable, the shoulders may be depressed, the heart upraised and displaced, and the expiratory sound may be completely suppressed. Dull percussion will be found in some degree,



although there is occasionally an emphysematous dilatation of the neighbouring parts, which will give the high-pitched muffled note.

**Multilocular and trabeculated cavities** are remarkable for absence of sound: the cavernous sound may be damped by the diaphragmatic partitions between the cavities or the trabeculæ which run across them. Such conditions are diagnosed only with great care.

It is well known that all hangings and curtains, and even carpets, materially modify and obliterate the resounding properties of a room, and the reverberation of echo has been stopped by carrying a string from one end of the room to the other.

Trabeculæ, which are long strings formed of vessels, bronchial tubes, or interlobular tissue, play exactly the same part in a cavity; they damp sound, and obliterate echoes.

They have the property of vibrating with cough, and thus producing sound. I have heard intense twang produced by the vibration of trabeculæ, and this sound is very characteristic.

**Isolated cavities**, separated from the principal disease, are frequently the result of the liquefaction of blood residues, and may be recognised by their peripheral contraction and their pigmented walls. I have already indicated the localities which they frequent.

#### **CLINICAL FORMS OF PHTHISIS.**

**Acute, or galloping phthisis**, was recognised and described by Hippocrates. The symptoms given by him are briefly these—fever, with rigors and constant sweats, extremities cold, bowels deranged; sputa small, dense, concocted, and brought up rarely and with difficulty; fauces painful from first to last, having redness, with inflammation; the patient soon wastes and gets worse, becoming delirious near death. It would be indeed difficult to supersede this account, which is as accurate as it is graphic.



It occurs in early life, attacking young girls preferably, and constitutions tainted by the heredity of phthisis. It may run its course in twenty days (a rare occurrence); more often it takes six to twelve weeks to complete its work of destruction.

**Symptoms** are those due to an acute blood-poisoning, concentrated particularly on the lungs.

The invasion of the disease is ushered in by rigors, followed by continuous fever, and there may be crops of sudamina; pain in the chest, dyspnoea of an aggravated nature, cough, often paroxysmal and extremely distressing, sputa scanty and viscid, gastric disturbances, irritable fauces, and red tongue; restlessness, wandering, and delirium close the fatal scene.

**Pathology** is of a very distinct kind, and is not to be mistaken for any other condition. To give a concise idea and definition of the macroscopical appearance, I would say that the disease appears to belong to the type of catarrhal pneumonia, attacking the lungs universally throughout, starting from innumerable foci, spreading rapidly and destructively, and finally terminating in suppurative ulceration, forming a number of minute cavities in all parts of the lung.

**Signs** are chiefly characterised by their universality; ordinary phthisis is confined, for some time at least, to the upper lobe of one side. Acute galloping phthisis starts from many points, and the signs are scattered all over the lung, but they are localised in spots or patches. Consolidation yields the first group of signs; feeble breath, of a high-pitched, ringing character, eventually becoming tubular and metallic; dulness of percussion, insulated spots of clicking, or the crepitant r le rapidly giving way to metallic gurgling rattle, and increased consolidation with bronchophony. The pleura is generally involved.

Such are the features of one of the most terrible and acute forms of phthisis, which are pathognomonic of the disease.

**Laryngeal phthisis** is another distinct form of disease



in which the larynx is primarily affected, the lungs being often secondarily involved. It appears to me to begin with an inflammatory affection of the larynx, which is followed by a rapid and general outcrop of tubercles in the larynx, pharynx, and even the tongue and the lungs.

**Symptoms.**—Catarrhal inflammation of the trachea and larynx, accompanied by dryness of the throat and hoarseness. On examining the throat it is found to be red and swollen, of a dark livid colour, and secreting a nasty viscid fluid. The hoarseness progresses and aphonia follows, with pain on deglutition and cough—and marked tenderness on handling the larynx; subsequently the development of tubercle becomes apparent, and they can be seen in the fauces, and sometimes on the tongue. The lungs give very little indications, at first, of being involved, and the sputa throughout are scanty.

**Pathology.**—The above conditions must be considered as rare so far as it concerns the outcrop of tubercles, and the pathology may be confined to rapid scrofulous ulceration. The disease generally attacks young persons; principally young men.

**Signs** are chiefly laryngeal, and the pulmonary signs are very scanty, and, according to my experience of the disease (examples are not very common), imperfect expansion, harsh, dry, high-pitched respiratory sounds are the chief signs.

**Scrofulous forms** of phthisis and pneumonia are invariably characterised by large cell-growths, which form plentifully and accumulate in the districts affected by inflammation. The marked characteristic symptoms of true scrofula are the invasion by cell masses and the absence of fluidity. Consequently the **pathological** appearances are those of caseation and proliferation rather than of softening and liquefaction. These conditions are accompanied with the history or presence of enlarged glands.

**Signs** are characterised by the sounds of solidity rather than of fluidity.



In scrofulous pneumonia scattered clicks and dull percussion may only be heard over lungs which are completely invaded in all parts by the scrofulous growths, and the appearance of the patient only indicates the insidious process which is undermining his life.

**Trabecular Destruction of the Lung.**—This variety (to which I have given a name which will sufficiently indicate what is intended) is another clinical variety of a peculiar and destructive kind. I have seen several examples of it, and invariably in persons of the lower orders, whose constitutions have suddenly broken down under bad-living, want, and the miseries of a poor and hopeless life; it has appeared especially to favour those who having, during the period of active vital growth, given up their bodies to all kind of excesses, by sudden immersion in the depths of poverty are unable to supply the stimulants to which they have long been habituated, or even to obtain the support necessary for life.

**Symptoms.**—Dyspnœa cough, the expectoration of abundant viscid, purulent, often very foetid sputa; rapid emaciation, night-sweats and prostration; symptoms of acute pleurisy, with pain on respiration. The disease will run a rapid course in two or three months.

**Pathology.**—The disease affects in a remarkable and most complete manner the whole of one lung, and generally it is the left which is rapidly cleared out, leaving long cords of trabeculæ, and a thickened pleura as the only remnants of what was once a lung. It attacks patients not affected by heredity, and appears to be a pneumonic process of an ulcerative type. I have given an illustration of a case in which the whole of the left lung was completely destroyed in three months. (See Fig. 19.)

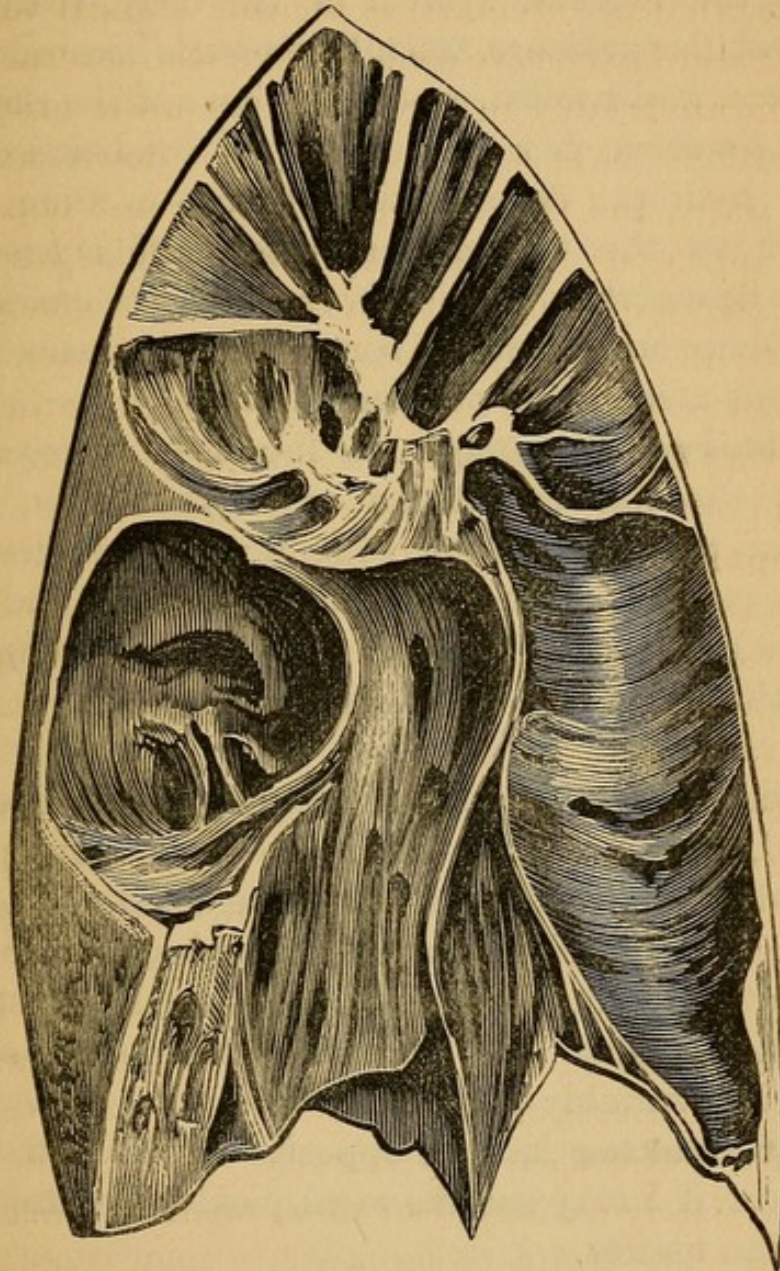
**Signs.**—The incipient signs are in some cases referable to the bronchial tubes; sub-crepitant râles scattered about, eventually becoming more fluid, and developing into bubbling or crackling rattles, very generally diffused.

In more advanced conditions we can hear no respiratory murmur or there be a slight in-and-out sound of which



the expiratory sound, such as it is, is lower in pitch. If the trabeculae be many the cough may be twanging in character, and with the action of the chest-walls, which is very imperfect indeed, a rolling up-and-down, viscid crackling sound caused, I believe, by the viscid secretions moving over the surface of the sac.

FIG. 19.



Trabeculated Excavation of Lung.

Palpation gives an increase of vocal fremitus.

Percussion gives a dull tympanitic note, and bronchophony is present.

Although the signs are few and negative in kind,



diagnosis may be made with certainty, and there is no other condition that can be well mistaken for it.

**Alcoholic intemperance** has a very distinct effect upon the condition, not only of the body generally, but also specially of the lungs.

**The beer-drinker** (of either sex) who indulges in copious potations pottle-deep, adds a large quantity of fluid to the vascular system of the body, increases the intra-vascular pressure, and all the vessels become enlarged, prominent, and swelled, especially those of the extremities. The consequence is a general sloppy condition of all the organs, hydropic degeneration (to use a more scientific term) of all the tissues and vessels, and a tendency to profuse hæmorrhage, which is especially characteristic of this intemperance, and in some cases imitates in all its symptoms a condition of hæmophilia.

**Pathology.**—The lungs are found in a condition of general vascular congestion and hyperæmia, and the traces of previous hæmorrhage are to be seen in old caseous patches in the midst of pigmented and more or less thickened lung tissue. When such lungs break down the secretion is likely to be profuse and the destruction rapid.

All the abdominal organs are found congested, enlarged, softened, flabby, full of blood, the heart soft and much enlarged, the liver fatty and congested, the kidneys large and flabby with adherent capsules, and the spleen large and diffuent. The sputa are, as a rule, in these cases sanguinolent, and the tubercle that is subsequently formed is invariably pigmented.

**Spirit-drinking** has an opposite effect, and tends to harden (or, if I may use the word, pickle) the lung and the rest of the tissues.

The symptoms are those of a constitutional character, and the effect is most marked on the nervous system—tremulous fidgets, insomnia, and general excitement.

**Pathology.**—A hardened fibrotic condition of all the tissues and organs is the result of spirit-drinking, and



the lung is included in the list of altered organs. It becomes hard and fibrotic; if cavities form they are often no larger than a nut, and scattered around the periphery; between them the tissue is firm and hard and of a fibroid character.

If tubercle result, it occurs in very close, compact, but large masses, sometimes assuming the form of bands traversing the lung.

**Signs.**—A hard, high-pitched, crackling r le is the sound most frequently heard in and indicative of alcoholic fibrosis, with hard, high-pitched, vacuous breath sound.

[A caution is here necessary to those who, recognising the source of the disease, think to check the further progress of the malady by stopping the supply of liquor—in other words, by turning off the tap. My experience upon this point is that such cases very rapidly deteriorate. If this is not done with great judgment, it would be better for the patient to continue his vicious habits than to discontinue completely and suddenly all supply of spirits.

This is not my advice, however, with regard to beer-drinking; the immoderate imbibition of fluid should be at once stopped and spirit substituted for beer.]

**Dislocation of heart** or displacement as a result of adhesive pleurisy and a contracting fibroid condition of the lung, with or without cavity, is a very common and important sign.

The heart is uncovered, the lung receding from its surface and leaving it completely exposed, the beat being perceptible over the pr cordial region, in the second interspace if the heart is drawn up, while the apex assumes various positions in accordance with the mechanical action (from the retraction of the left or the right lung) which causes an alteration in its situation.

It may be drawn upwards and over to the left axillary region, the apex beating in the side, or it may be drawn up towards the right side; the base in this case being as high as the first interspace, and completely over to the



right of the midsternal line, the apex beating near the middle line under the sternum in the lower sternal region. Dislocation to the left is usually more marked than on the right side, inasmuch as contracted conditions of the lung are also more frequent on the left side.

**Dislocation of cavities** may be observed after some lapse of time, the cavity having a tendency to slide with the progressive retraction of the lung; so far as my observation goes, cavities in the anterior of the lung move towards the humerus, cavities in the posterior part of the lung move towards the spine, the firmest and thickest adhesion being situated here.

**Advancing phthisis** is marked by the extension of sounds and the increase of their fluid quality; as a rule, the more abundant the secretion the more rapid the destruction.

**Arrested phthisis** and its progress towards recovery is marked by the absence of fluidity in the sounds, and their gradual assumption of viscid, and subsequently dry, qualities, together with the general improvement in the patient's health and the diminution of expectoration. The tissue hardens and the signs become harsh, rough, and dry, assuming that whispering or rustling high-pitched quality which I have already attempted to indicate in the description of the sounds caused by a contracting cavity.

The student must be advised that patients suffering from the last stage of phthisis present themselves for examination when no signs can be detected; this often follows diarrhœa or profuse sweating, and is, according to my experience, more commonly the case with women than men.

**Decubitus** in phthisis is usually on the sound side: cough being increased by the accumulation of secretions in the diseased lung if the patient lies on the affected side.

**PNEUMOTHORAX**, or the presence of air in the pleura, arises from perforating disease of the lung, or



traumatic injury causing a direct opening through the chest-wall, or laceration of the lung from contusion and fracture of the rib.

Perforation of the lung from disease takes place from disease within the lung, destroying the pleura, this being the usual condition of pneumothorax, or from an empyema boring its way upwards into the lung, and establishing a communication with the air-passages.

The disease is generally accredited to tubercular action; but although it is a consequence of phthisis, perforation is caused by the corrosive action of a purulent, almost gangrenous, ichor, which burrows a way, sometimes by very small channels, right through everything that comes in its way, including the pleura, when it is not protected by adhesion.

I fail to comprehend on what grounds this peculiar action is ascribed to tubercle, the essential character of tubercular action being chronic irritation and cell growth. It is rather due to a pneumonic process of the suppurative type, and the perforation may take place in caseous patches which are suppurating, and in some cases have a distinct circumscribed appearance, which suggests the notion that they are really morbid matter planted in parts of the lung by insufflation. A more mechanical and less striking result is caused by the perforation of a lung from an empyema. It is to the first class of case that acute and striking symptoms belong.

**Symptoms.**—Acute pain in the affected side, of a most poignant and sudden character, accompanied with severe dyspnoea and prostration. The patient is often conscious that something has given way. Orthopnoea is the position generally assumed at the moment, but a variety of postures may be tried, at one time the patient being supine with the head raised, at another lying over towards the sound side.

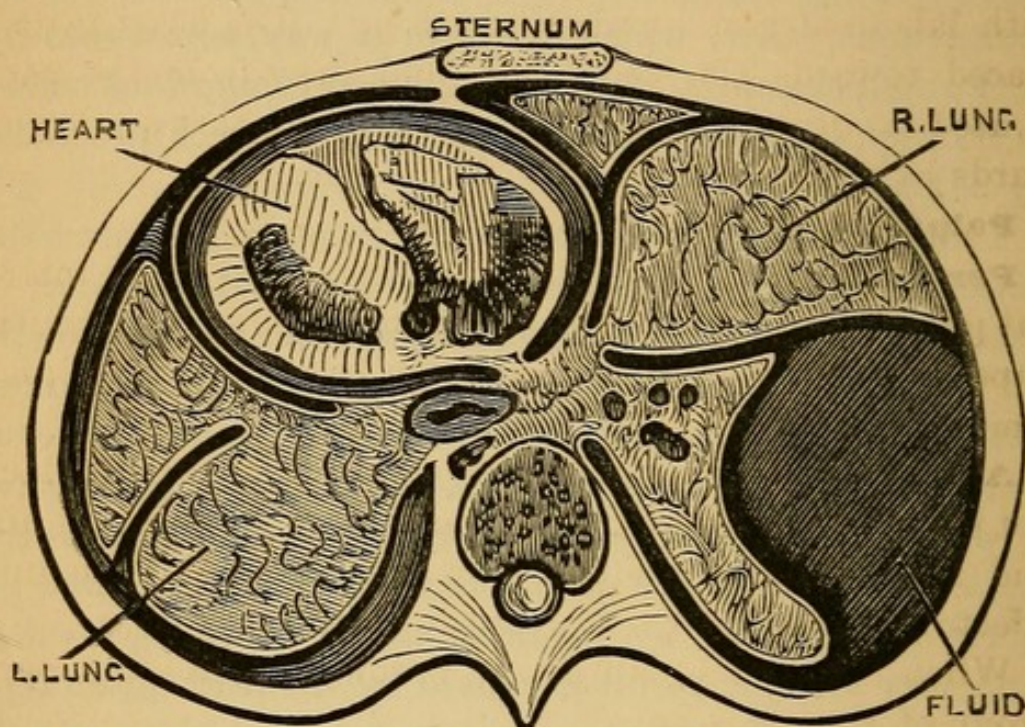
Palpitation is generally a marked symptom; the pulse-rate being greatly accelerated, and respiration also remarkably hurried. Great anxiety is depicted on the coun-



tenance, and the lips and face become livid, the voice being reduced to a whisper, and the surface clammy.

As an exception to the rule, Walshe mentions a case (and I could give a similar instance) of a man in the third stage of phthisis, coming to him one day, with a little more anxious face than usual, complaining of slight pain below the nipple of some hours' duration. He had walked without difficulty more than a mile, although the heart was pushed over into the left axilla; the man died in two weeks.

FIG. 20.



Transverse Section in Right Pyo-pneumothorax. (Pirogoff.)

**Pathology.**—On opening the chest more or less air usually escapes; this will depend upon the size and patency of the opening from the lung into the pleura. The lung is more or less collapsed. Perforation occurs in the posterior axillary region, or in the mammary region, in front below the third rib. I have seen as many as four openings in the lung. Perforation is most frequent on the left side. All the abdominal organs are gorged with blood, which is forced upon them in consequence of the pressure upon the vessels of the wounded lung.

**Signs** vary according to the size of the opening from



the lung to the pleura, and the free or impeded communication between the air within the pleura and the large air-passages.

If the opening be small and easily closed, formed at the termination of a small and tortuous sinus, air collects within the pleura, and (expanding) causes much distension of the chest with pain.

**Inspection** in this case shows marked bulging of the affected side with obliteration, and sometimes convexity of the intercostal spaces. There is no movement of that side of the thorax, the sound side heaving frequently with laboured and hurried breathing. The heart is displaced towards the sound side, and away from the perforated lung, and the abdominal organs are thrown downwards by the pressure on the diaphragm.

**Palpation** detects little fremitus.

**Percussion** is high-pitched and tense, and if the internal pressure be very great, the vibrations may be so much impeded that the note of percussion becomes muffled and almost dull.

**Auscultation** gives negative signs when the air does not enter freely; there may be no to-and-fro sound at all, and all the sound audible may be the sound of the unaffected lung transmitted over the compressed lung.

When the opening is free and patulous, then a different group of sounds is heard.

**Inspection.**—The distension of the affected side may not be great; depression of the intercostals may actually be observed with the inspiratory action of the thorax.

Palpation detects rather more fremitus.

Percussion gives a noisy, resonant, amphoric note.

**Auscultation** detects the sounds of loud to-and-fro amphoric respiration which may have a metallic tone. Vocal resonance may be intense and diffused and metallic in quality.

In the fistulous opening into the lung from an empyema there is usually free communication, and for two reasons—one is, that the opening is conical and large,



not formed by the trickling downwards of a corrosive fluid, but by the boring pressure upwards of a large body of pus; and the other is, that as the opening is situated above the level of the root of the lung (the line of the main bronchus) no expansion of the lung takes place to close the opening. The rationale of this is very simple: the empyema is mechanically forced upwards by the pressure of the lower ribs and diaphragm, the locality of least resistance, the direction in which the abscess is forced to point being the upper part of the lung, hence the empyema burrows upwards and bores into the lung above.

In cases of pneumothorax from empyema the usual signs of pneumothorax may be found, and pectoriloquy also, the margin of the sac being thus exactly limited, while over its boundaries buzzing bronchophony only can be heard.

The bell sound is elicited over the air cavity of pneumothorax by placing a half-crown on the walls of the chest and striking it sharply with another coin, the ear being placed against an opposite part of the chest.

There is one remarkable point with regard to perforation and compression of lungs which deserves consideration.

Air can, I believe, pass across the mediastinum and burst into the pleura. Wilks and Moxon relate two cases of pneumothorax from tracheotomy, both lungs being found contracted in the chest, and the cellular tissue in the posterior mediastinum was filled with air, producing large bubbles which had burst through the pleura into the chest.

Some time ago a case of pneumothorax occurred, proving fatal in an hour. When I examined the body I found the left lung collapsed and compressed, and a small perforation in the right lung, which was universally adherent to the chest-wall.

Dr. Powell records a case of a kindred nature, in which a sinus had opened from an old cavity through the posterior mediastinum into the opposite pleural cavity.



Pneumothorax is usually accompanied with fluid, and then it strictly demands the term of **hydro-pneumothorax**, or if the fluid be pus, **pyo-pneumothorax**, the latter term being especially applicable to an empyema, which has formed a communication with the outside air through the lung, and is not yet emptied.

When fluid and air are present together, then the succussion sound or splash may be heard. This only occurs when the amount of fluid is not excessive; if it be excessive, the sounds due to air are lost and replaced by dullness.

The splash of succussion has generally a ringing, metallic, silvery quality, which also modifies the quality of the voice and breath sounds.

The only sound that is likely to mislead is the metallic sound of a distended stomach containing fluid, and this point must be decided by percussing out the position of the stomach.

In hydro-pneumothorax the patient assumes a variety of postures and changes his attitude frequently, sometimes on the affected, sometimes on the sound side, rarely assuming the upright position.

In one remarkable case that came under my notice, adhesion to the diaphragm had taken place previous to the perforation of the lung; consequently the sounds due to the presence of air were almost annulled, and the sign which indicated the condition was a metallic splash which could be heard at the back, the fluid audibly running along the posterior surface of the lung and descending when the patient's position was suddenly changed.

**INFECTIVE PHTHISIS.**— There is one other variety of phthisis to which attention must be directed, although observations on this are confessedly incomplete, but many marked instances have come under notice of such a kind as to compel belief in the infectivity (in some form) of phthisis. For some time the peculiar symptoms and signs of that form of pulmonary disease which especially attacks wives who have devotedly nursed hus-



bands who have died from phthisis, have attracted my notice, and the conclusion at which I have arrived is this—that there is a peculiar constitutional condition caused by the infection of phthisis simulating blood-poisoning of a chronic kind, in which the general disturbance of the system is out of all proportion to the amount of pulmonary disease, which is not established until some months afterwards: the condition is not a true phthisis, but rather a chronic infective pneumonia. I have now in my note-book details of fifteen cases, and they tend to show that this condition is only caused by close communication with the phthisical patient during the last months of life, and the phenomena of infection are these:—

**Symptoms.**—Rapid emaciation, anorexia; depression, with rigors repeated at intervals, profuse night-sweats, diarrhœa, and often vomiting. Cough is established late in the disease, and the sputa are frequently bloody. The patient is soon very much emaciated, and of a sallow hue; skin clammy, pulse small and rapid.

**Pathology.**—Hitherto only one case of the kind has been examined by me, but in this the appearances were very peculiar. The right lung had a cavity in the upper lobe which had apparently formed from the breaking down and evacuation of a congestive pneumonia, the surrounding walls being soft and red, and the contents of the cavity resembling the debris of congestion. One or two groups of quite recent grey tubercle were found in close connection with this cavity. The whole of the left lung was studded throughout with little patches of lobular congestion, some of which (of old standing) had caseated and lost their colour. Microscopical examination showed a congestive condition of the peripheral alveoli, and many of the vessels were found plugged with small blocks of fibrin. The connection between this condition and pyæmia was shown by the presence of a recent empyema of small size, and an erysipelatous phlebitis of both fore-arms.

So fully persuaded have I been as to the possibility of some form of infection, that guinea pigs were, at my



suggestion, exposed to the emanations of a number of phthisical patients. Out of eleven thus exposed in air-flues leading from the wards (the air being drawn off by a heating-apparatus) two died, one in eight, the other in nine weeks after exposure, and they were found affected with artificial tuberculosis of the lungs, liver, spleen, and glands. They were examined by Dr. Klein, whose name will be a sufficient guarantee for the accuracy of the observations. The most remarkable point about the infection is that it coincides in time with that necessary for the production of tuberculosis by the injection of tubercular matter.

These experiments are as yet incomplete, and only tentative; but they appear to lend colour to the theory that the exhalations of a phthisical patient are poisonous.

**Signs.**—The next step is to show that the pulmonary signs differ from those of ordinary phthisis, and they do so in two points: one is, that the lung disease does not account in any degree for the depressed and emaciated condition of the patient; the other is, that signs are frequently to be found on both sides. This is not invariably the case, but I have found it so in the majority of the cases (15) that have come under my notice.

**Inspection** shows deficient expansion and imperfect action.

**Percussion** gives a dull note.

**Auscultation** elicits the viscid sub-crepitant râle, dull in sound and slow in formation, which is to be heard in infective pneumonia, and is very much like the sounds heard from hæmorrhage into the lung. These signs, when put on paper, may seem to afford small grounds for diagnosis, but when the whole phenomena of the case are considered they appear to me to uphold the theory that a form of blood-poisoning may result from the infection of phthisis. If the lung disease be advanced excavations may be present.

I think it right thus to call attention to this point, as it may tend to induce those who nurse patients suffering



from phthisis to adopt careful hygienic measures, notably free ventilation, in order to shield themselves from harm.

**THICKENED PLEURA** is a condition which frequently occurs after chronic pulmonary disease of a fibroid character. Adhesion first takes place as a consequence of irritative diseases in the pulmonary tissue, and the frequent concomitant of the phthisical process. The agglutination of the pleural surfaces takes place without effusion, and is a gradual process, which creeps downwards from the apex to the diaphragm. During the course of the disease the lung, not being used so much as the sound lung, adheres in the quiescent state—that is to say, more or less in the condition of expiration. With the further contraction of the lung, due to the fibroid action, the adhesions are stretched, and as the traction is equal at all points on the surface, the intercostal spaces are pulled in and beneath the ribs, fatty tissue fills up the parts so as to form marked ridges on the surface of the pleura, and the thickness of the pleura is often great, sometimes as much as an inch. This adhesion of the pleura and the active contraction of the fibroid lung are the agents in the deformities which are so often observed in the chests of phthisical patients.

**Symptoms.**—Dyspnœa, with occasional pain on exertion.

**Pathology.**—Thickened, white, sclerous condition of the pleura.

**Signs.**—Inspection shows fixity of the thorax in proportion to the extension of the adhesion. If the adhesion has fixed the diaphragm, the shoulder is depressed, and the body inclines to the side affected. The intercostal spaces are much depressed.

**Palpation** indicates great hardness of the tissue.

**Percussion** will give a dull note on the surface with a slight blow, and occasionally the dulness is very marked.

**Auscultation** shows more or less diminution of breath sound. The characteristic sign of this condition I believe



to be marked transmission of the heart's sounds over the area of adhesion, their intensity being sometimes loud.

In one case which was under my care, and subsequently examined by Dr. Greenfield, the physical signs indicated the presence of a circumscribed tumour in a peculiar condition, the right infra-mammary region close to the sternum. The post-mortem examination showed a large circumscribed patch of very thick pleura in this position, with fibroid lung beneath.

**Cardiac sounds** arising from pleural adhesion.

The adhesion of the pleura to the pericardial surface causes traction upon and displacement of the heart. The systolic murmur which often follows this has been already alluded to: it is audible generally over the pulmonary vessel; sometimes there is a double exocardial crackle heard, which is probably due to the creaking of the adherent parts, especially if a cavity be closely contiguous to the heart, the heart sounds give rise to creaking of a cavernous quality, sometimes of a very noisy character, or the fluid contents of the cavity are audibly jogged by the heart's action.

The displacement of the heart from excessive accumulation of fluid within the pleura will, by the derangement of the form of the vessels, give rise to murmur over the base of the heart which disappears after tapping.

### **TUBERCLE.**

Whether tubercle is the incipient cause of phthisis or not, no one who has ever examined a phthisical lung will deny that it is found as a recent formation before death. Putting aside, then, the vexed question as to the commencing process in phthisis, I must endeavour to point out how the formation of tubercle is to be recognised by physical signs.

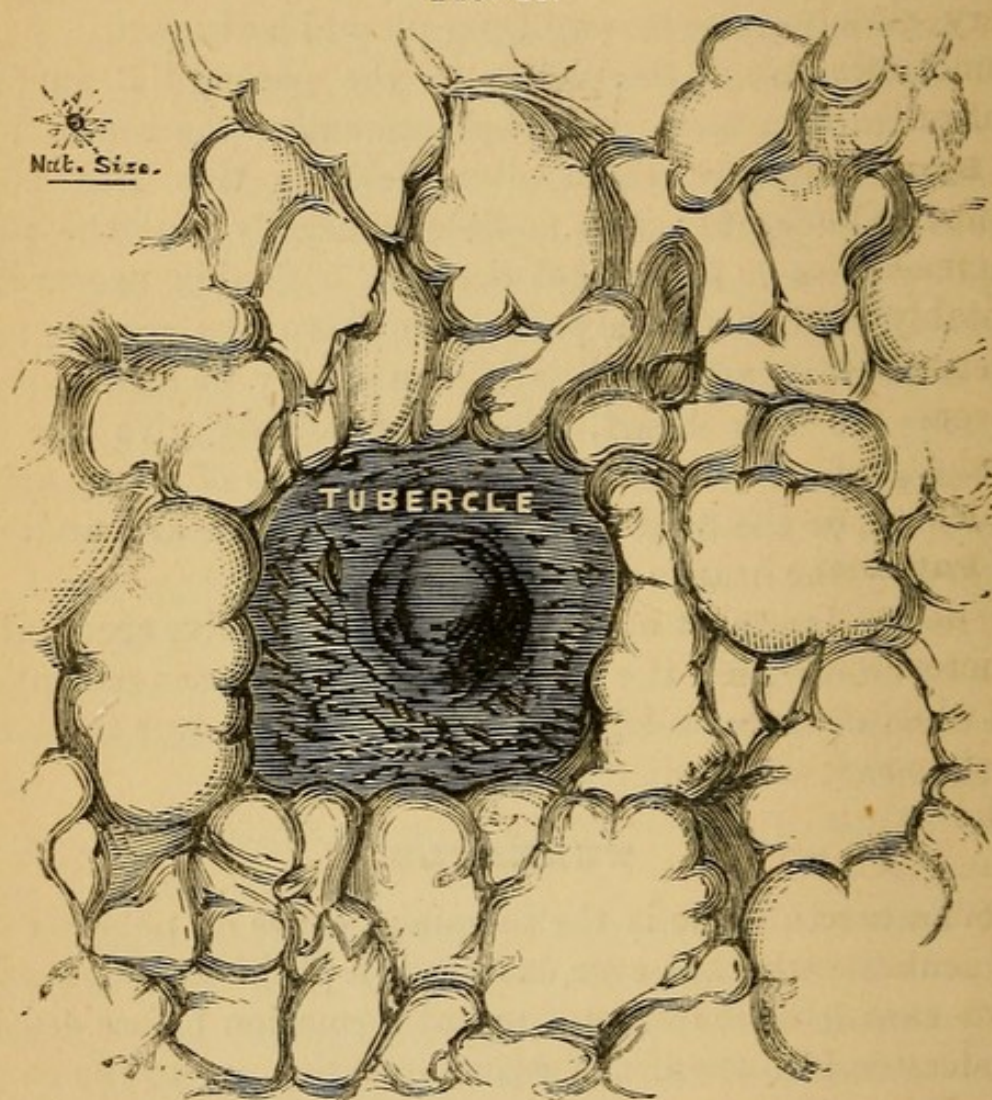
Tubercle must be considered from two aspects: it may be general, affecting both lungs, equally and uniformly (or, indeed, the infection may be carried still further throughout all the organs of the body), this must be



termed general tubercular infection, or **acute tuberculosis**, or it may be local, affecting only limited portions of the lung, in which case it may be called **localised tuberculosis**.

Buhl long ago pointed out that acute tuberculosis was a general infection, derived from the absorption of caseous matter; the channels by which absorption is effected being the lymphatic vessels.

FIG. 21.



Distension of Lung round Localised Scrofulous Tubercle,  
Secondary to Phthisis.

Localised tuberculosis appears to me (from the consideration of many pathological arguments which can be brought to bear on the subject) due to the limited infection (not affecting the whole system) of parts of the lungs



from the insufflation of septic matter into the lobules along the bronchial tubes, or from an eruption of tubercles in the neighbourhood of an infecting region from soakage into the tissue.

Tubercle, then, from my point of view, is invariably a secondary product due to implantation, general or local, of septic matter in tissue, capable of producing tubercle by connective tissue growth.

**Acute tuberculosis** in its symptoms approximates very closely to the conditions of typhoid fever, and is sometimes only to be distinguished by very careful observation.

**Symptoms** are very subtle, and chiefly those which point to blood-poisoning; rigors, night-sweats, diarrhoea, increased temperature, rapid pulse, great emaciation, and notably a marked pallor and anæmia of body. This is a distinctive characteristic between the exanthematous fevers and tuberculosis; in the fevers we find flushing and a general mottling of surface; in tubercle infection, paleness, and rapid emaciation.

**Pathology.**—The miliary eruption of tubercle has been so often described that there is no need to enter into microscopical details in this place. Two illustrations (one of which will be found on page 65) are given to show the distension of lung which takes place around tubercle. The two drawings are taken from different specimens, the one given here representing a nodule of localised tubercle from a case of scrofulous phthisis, the other from a case of acute tuberculosis: the actual process is different in the two cases, but the result is very similar. It must be understood, however, that catarrhal pneumonia is a condition which often precedes tubercle in localised form, and also, but not so frequently, the acute form of general infection.

The lungs in acute tuberculosis on removal from the body are distended, and of a grey colour, and their condition might easily be mistaken for that of emphysema. An experienced observer will, however, notice that the



inflated grey appearance is universal and uniform, whereas in emphysema portions of the lung are more affected than others, and an irregular bullatous condition is present.

Attention must also be called to the fact that the grey distension of the tuberculised lung is always in the anterior portions, while the posterior parts are compressed and condensed, and the tubercle often closely matted together. This is totally irrespective of the presence of catarrhal pneumonia or congestion, which will induce gravitation of the fluid secretions to the back part.

In most of the cases that I have examined I have found this marked distinction of distended lung to the front, and compressed lung to the back; a condition which I believe to be usually absent in emphysema.

On making section of the lung, we see minute isolated nodules of tubercle scattered uniformly through the lungs. I have been unable to detect any other peculiarity in the localisation of miliary tubercle.

This condition may occur with or without pleurisy, and in some cases pleurisy may take place during (I presume in consequence of) the infection.

This appears to me so important a distinction between this disease and emphysema (for which tuberculosis is too often mistaken, but which is incapable of pleurisy) that I referred to Dr. Goodhart, whose pathological experience is very great. Out of thirty-seven cases of miliary tubercle he found the symptoms of pleurisy in seven; while with regard to the disease in children he has formed a decided opinion that the pleuræ are very often adherent by somewhat recent lymph—miliary tubercle in children being more mixed up with cheesy products than in adults. Although, then, pleurisy is not frequent in the tuberculosis of adults, it is not absolutely precluded, as it appears to be in idiopathic emphysema.

**Signs.**—Inspection shows rapid breathing, generally abdominal, chest not bulged, extreme emaciation, generally



depression of the intercostal spaces, and fixity of the ribs ; skin very dry and rough.

**Palpation** gives no distinct indication, but the fremitus is more distinct behind than before.

**Percussion** gives a high-pitched, tense, muffled note in both superficial and deep percussions, and it sometimes has a diminished resonance, approximating to dull. In front, over all the anterior portion of the chest, this tense percussion note is most marked ; behind, the note becomes actually hard and dull.

**Auscultation.**—Vocal resonance in front of a very diffused, buzzing, indistinct character ; behind, high-pitched bronchophony marked. Respiratory murmur diminished and of a high pitch ; inspiratory murmur deferred ; expiratory shortened (this arises from the distension of the lung). Behind, indistinct tubular breath sounds may be heard.

If to this condition be added congestion or catarrhal pneumonia, scattered clicks, crepitant sounds will be heard ; sometimes bubbling râles of a high-pitched, rattling quality, which often mask the characteristic signs.

The distinction between tuberculosis and emphysema, and other diseases, is so difficult and nice that, although I have already twice drawn attention to the point, it may be convenient to give the chief characteristic features of the two diseases in the following Synoptical Table :—

<i>Acute Tuberculosis.</i>	<i>Emphysema.</i>
In early life. Initial fever and rigors. Diarrhœa often present. Pallor. Abdominal respiration. Chest not enlarged. Deep percussion muffled. Marked dulness behind.	In advanced life. No fever. Diarrhœa not a symptom. Cyanosis. Thoracic respiration. Chest bulged. Deep percussion resonant. Resonance behind.



**Localised tubercles** occurring in lungs excavated by phthisis are found in alveolar groups or clusters, and the bronchioles leading to them are in a condition of marked irritation.

**Symptoms.**—If they are forming in any quantity, as they are apt to do when the secretion is excessive, or the patient's strength so failing that the matter is not ejected by expectoration, dyspnœa of an aggravated form, with night-sweats, are observed, accompanied with that paleness which is a marked peculiarity in the aspect of the tubercular patient.

**Pathology.**—Alveolar groups of tubercle invariably assume special localities, and are to be found in the anterior and most expansile parts of the lungs; adhesions of the pleura discourage their formation, and the difference in the amount in one lung, which is completely adherent, and in the other when perfectly free is remarkable.

This form of tubercle, as a rule, avoids the back of the lungs, especially when the lumina of the air-sacs are already occupied by the secretions of congestion, and if an acute outcrop is developed (in a case of one-sided cavity) in a lung previously healthy, of which I have seen several examples, the tubercle is chiefly deposited below the level of the main horizontal bronchus.

In addition to these points (which appear to my mind very suggestive) must be added the observation that where the expectoration has been for some time back tinged and mixed with blood continuously the groups of tubercle are much pigmented.

**Signs.**—The chief indications are given in the dyspnœa: the respiratory murmur being feeble, with an occasional scattered subcrepitant sound, or if the secretion be very profuse, a plentiful crop of bubbling râles; very likely to be confounded with capillary bronchitis, and only to be distinguished by the high-pitched, rattling quality which belongs to the tubercular affection.

In chronic fibrosis of the lung with excavation occurring in old spirit drinkers tubercle is sometimes found



deposited in a broad, glistening, homogeneous band affecting the middle of the lung: this is of slow formation, and yields dull percussion, especially at the back, with absence of respiratory murmur, and an indistinct bronchophony. In all cases of localised tubercle, it is only by considering all the phenomena that a diagnosis, which is not simply conjectural, can be made.

### TUMOURS.

Pulmonary tumours may be classified and considered under three heads—glandular enlargements, malignant growths, vascular tumours: these are practically those which are presented to notice, but almost any kind of growth occurring in the body may be repeated, usually as a secondary condition in the lungs. The classification above is not intended to be scientific, inasmuch as all secondary growths in the lymphatics might be considered as glandular enlargements; but under this term it is convenient to consider two forms of tumour which are very similar, and in some cases extremely difficult to distinguish. They are Scrofulous Glands and Lymphadenoma.

**Scrofulous glands** are very common, especially in childhood, but they may be seen in the adult, and associated with scrofulous forms of phthisis.

**Symptoms** in children, dyspnoea and harsh, croupy, dry cough, no expectoration, the cough being harsh and ringing, and aggravated at night.

**Pathology.**—This condition is caused by enlarged scrofulous glands embracing the trachea about its bifurcation, and situated beneath the manubrium sterni.

**Signs.**—Inspection shows the upper part of the sternum thrown forward, if the enlargement is great and the case chronic, as in the accompanying illustration. Palpation detects nodular swellings and hardness in the supra-clavicular fossæ.

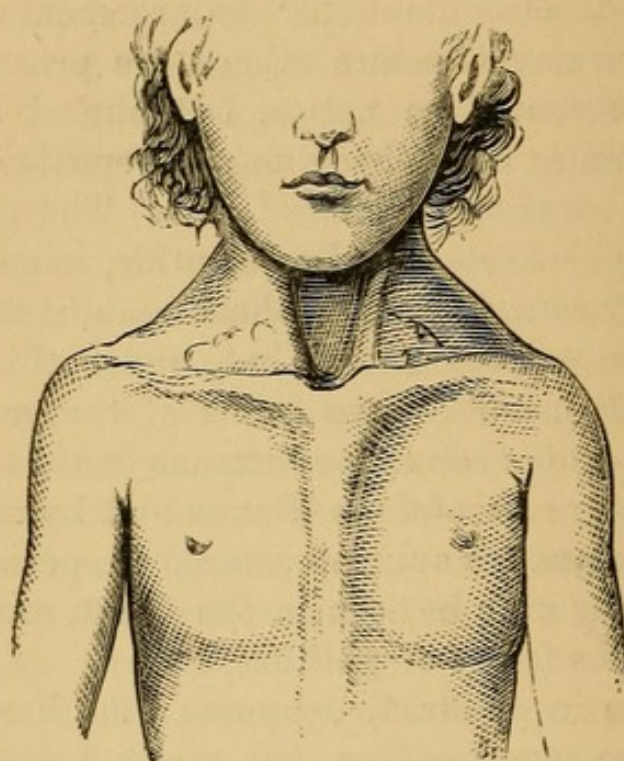
Percussion gives marked dulness over the region. Auscultation shows pressure on the trachea, a harsh tracheal sound, intense behind, over the tubes.



The second sound at the base of the heart is generally accentuated with a sharp clack, the valves closing under increased pressure, and there may be a systolic bruit heard over the aortic or the pulmonary, or over both valves.

I have given a drawing of the chest in a typical case, because this condition, which is a very common one with scrofulous children, is often overlooked.

FIG. 22.



Enlarged Bronchial Glands pressing forward the Upper Part of the Sternum.

**Lymphadenoma** sometimes occurs as a general affection of all the glands (Hodgkins' disease), or more locally it may affect the glands of the neck or the mediastinum. It is difficult to be sure of the exact condition : diagnosis is helped by the extreme anæmia of the patient, who has a sallow hue, and the glandular swellings are more regular than in scrofula, and generally show first in the supra-clavicular regions ; but this observation is made with great reserve, as the clinical distinction between the two is often impossible. When it occurs as a mediastinal



tumour it spreads into and encroaches upon the lung tissue.

The growth is found to consist of reticular meshwork containing lymphoid cells.

Mediastinal tumours of all kinds give similar symptoms and signs; of these, the following are the most characteristic:—

**Symptoms.**—Dyspnœa of a very aggravated kind recurring in spasmodic attacks; more or less orthopnœa, sometimes very marked; the patient being often obliged to sit with the head down to relieve the trachea of the tumour's weight; dreading to go to bed, suffering from wakeful and restless nights, cough with bloody sputa; and pain, and a sense of weight and constriction behind the sternum.

**Pathology.**—Tumours of various kinds, embracing the trachea and bronchi, and sometimes moulded firmly round the large vessels of the heart.

**Signs.**—Inspection detects the prominence of the sternum, the sternal and clavicular fossæ being very deep, the veins of the chest tortuous and enlarged; local bulging over the tumour; stridulous or aphonic voice.

Palpation gives annulled vocal fremitus if the tumour be next the chest-wall; if it be covered by compressed lung vocal fremitus may be increased.

**Percussion** gives distinct and circumscribed dulness; a very valuable sign.

**Auscultation** gives the signs of pressure on the tube; laryngeal stridor, and harsh, tubular, high-pitched respiratory sound, or no breath sound if the pressure be great.

Vocal resonance very indistinct from the tumour itself, but intense bronchophony or pectoriloquy if compressed lung cover it.

**Cancer** may assume two forms; it may invade the lung in isolated nodules, varying in size from a hemp-seed to an orange, in which case it is generally secondary to distant cancer, or it may originate in the bronchial



glands and mediastinum, or more rarely attack the lung as a primary growth.

**Symptoms.**—Chiefly pain and emaciation; the dyspnoea is not so constant, but will depend upon the amount of invasion and the region infected; cough is usually attended with bloody expectoration.

**Signs.—Inspection.**—Diminished respiratory action; flattened side, the axillary glands sometimes involved; malignant sallow hue of surface.

**Palpation.**—Vocal fremitus increased if the lung is compressed; annulled if expanded tissue intervene between the chest-wall and the tumour.

**Percussion** gives a dull sound proportionate to the extent of the growth.

**Auscultation** detects tubular respiration, or if a bronchus be compressed and obliterated by the growth the respiratory sounds may be annulled and silent.

In the nodular form, and when the growths interfere with the passage of the air, clogged respiration of a very coarse and marked kind may be sometimes heard.

Vocal resonance may be diminished or intensely increased.

**Vascular tumours** are considered in the next section, under the heading "Aneurisms."

The evidence of the presence of such a tumour as far as the pulmonary signs are concerned depend upon the pressure produced upon the lung. If the main bronchus be flattened, harsh tubular breathing will be heard; if it be occluded no breath sound will be heard over the tube, and only an indistinct, stridulous, deferred, respiratory murmur over the posterior base of the lung thus affected.



SYNOPTICAL TABLES

OF

THE SOUNDS IN DISEASES OF THE  
RESPIRATORY ORGANS.



*Synopsis of Sounds in Disease of the Respiratory Organs.*

## MORBID VESICULAR SOUNDS.

*Vesicular Murmur Altered.*

<i>Sound.</i>	<i>Significance.</i>
Increased or exaggerated	Distended lung
Diminished	Feeble action Adherent pleura
Suppressed	Obstruction of air-passages, pleural fluid, or tumour
Inspiratory sound short	Hampered expansion Adherent pleura. Pleurisy
Inspiration deferred	Emphysema
Expiratory sound short	Distended lung
Expiratory sound prolonged	Emphysema Diminished elasticity
Expiratory sound suppressed	Contracted cavity
Hesitating	Hysteria
Jerking	Adherent pleura
Cogged	Cancer. Tubercle
Harsh	Induration and thickening Fibrosis. Syphilis. Hæmorrhagic

## ADVENTITIOUS SOUNDS.

Fine Crepitation	First stage of pneumonia
Crepitant râle Viscid crackle Occasional click	Early stage of phthisis
Subcrepitant râle	Congestion Brown induration Pulmonary apoplexy
Bubbling râle	Capillary bronchitis Œdema of the lung

## MORBID BRONCHIAL SOUNDS.

Sonorous rhonchus	Large tube bronchitis
Sibilant rhonchus	Small tube bronchitis
Tubular } Bronchial } Respiration	Consolidation of lung Compression from fluid, or tumour



## SOUNDS OF DESTRUCTION.

<i>Sounds.</i>	<i>Significance.</i>
Gurgling râle	Softened lung tissue
Cavernous respiration	Pulmonary cavity
Amphoric respiration	Pneumothorax Pulmonary cavity

## PLEURAL SOUNDS.

Friction	Roughened pleura
Metallic sounds	Hydropneumothorax
Splash	Hydropneumothorax

## MORBID VOICE SOUNDS.

Increased vocal resonance	Dense pulmonary tissue
Diminished vocal resonance	Interposition of fluid, solid, and air between the chest-wall and lung.
Bronchophony	Solidification } of lung Compression } and in other conditions
Pectoriloquy	Air-containing cavity but also in other conditions
Ægophony	Interposition of a vibrating sur- face between a rigid tube and the chest-wall
Metallic sounds	Hydropneumothorax

## MORBID COUGH SOUNDS.

Intense	Consolidation Cavity
Diminished	Effusion into pleura Tumour near chest-wall
Cavernous	Pulmonary cavity
Metallic	Hydropneumothorax
Brassy	Bronchial irritation and pressure.



## THE PHYSICAL EXAMINATION OF THE HEART.

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### CHAPTER XVI.

#### CLINICAL ANATOMY OF THE HEART.

THAT portion of the external surface of the chest which covers the heart is called the **præcordial region**; it extends from the middle of the second intercostal space to the lower border of the fifth intercostal space, and is bounded on the right side by a vertical line situated a finger's breadth to the right margin of the sternum, and on the left by a vertical line a little within the left nipple line.

The exact size and position of the heart varies in individuals, but an average may be struck by comparing a number of cases; and the upper margin of the heart may be placed, as above, in the middle of the second interspace. The condition of the auricle varies with the quantity of blood contained, and if distended they may reach to the second rib.

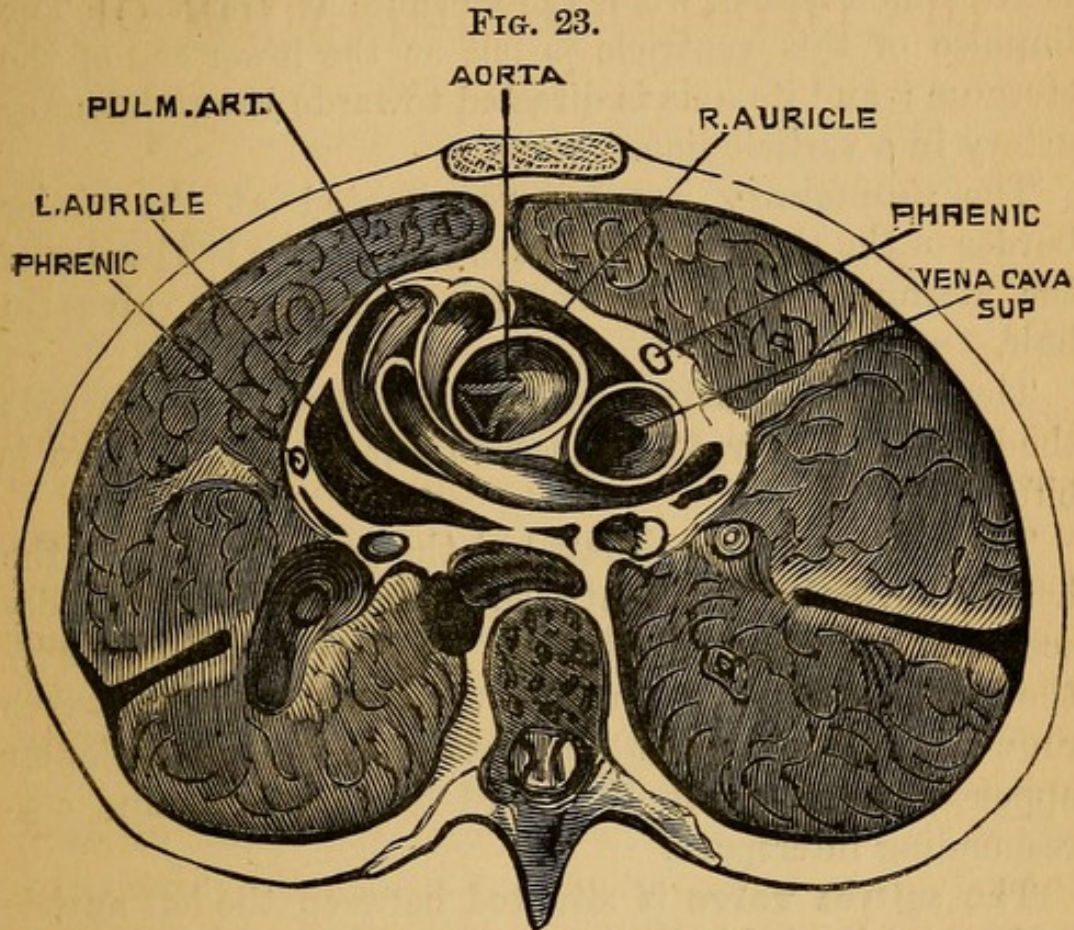
Beneath the præcordial region between the sternum and the spine lies the heart. It has an irregular conical shape, the base facing upwards, backwards, and towards the right shoulder; the axis being directed downwards, forwards, and towards the left.

The **apex** is situated in the fifth intercostal space, between the fifth and sixth ribs, about midway between the line of the left nipple and the left border of the sternum; three and a quarter inches to the left of the mid-



sternal line; two inches to the left of the left border of the sternum; one and a half inches below the left nipple; one inch to the right of the left nipple.

The heart has four chambers—two ventricles and two auricles; one ventricle and auricle on each side.



Transverse Section of Thorax showing Position of Valves of the Heart. (After Braune.)

**Right auricle** lies in front of the right half of the left auricle, above the right ventricle, behind the third right cartilage—*i.e.*, between the lower border of the second right cartilage and the lower border of the fourth, extending into the right mammary region an inch (sometimes more, sometimes less) beyond the right parasternal line. It is, as a rule, covered by the right lung.

The **tricuspid** valve is situated between the auricle and ventricle, between the third left and the fifth right cartilage.

**The right ventricle** occupies the lower sternal region,



extending from the third cartilage to the sixth, and transversely from the right parasternal line to the junction of cartilages and ribs, between the third and the fifth ribs on the left side.

This portion of the heart forms the exposed front of the heart, which is seen when the sternum is removed. The impulse of this ventricle is felt at the lower end of the sternum; and its axis is directed towards the pulmonary artery in a vertical line.

The **pulmonary valves** are situated at the upper border of the third left cartilage, close to the left border of the sternum, which divides the line of the valves in half.

The pulmonary artery ascends from the valves towards the left side as far as the second left cartilage, where it bifurcates.

The **left auricle** is situated at the posterior part of the heart above the left ventricle, resting behind on the descending aorta and œsophagus; and covered in front by the large vessels, and partly by the right auricle, it curves round towards the left and forwards, and its auricular appendix lies beneath the third left cartilage and corresponding interspace.

The **mitral valve** is situated between the left auricle and ventricle; behind, and to the left of the tricuspid; about level with the upper border of the third left cartilage, close to the left edge of the sternum; its lower border being level with the fourth cartilage, just behind the midsternal line.

The **left ventricle** lies below the left auricle, to the left of the spine, extending towards the left, and forwards to the apex situated in the fifth intercostal space; it forms the convex left portion of the heart, and extends from the third to the fifth left interspace; its furthest left border reaching close to the left nipple line.

Its axis is horizontal, and its cavity, which is conical in shape, is narrowest at its greatest distance from the aorta.



Its apex is the apex of the heart, which beats at the fifth interspace, where its impulse is felt.

The **aortic valves** are situated at the orifice of the ventricle and the root of the aorta; below, and to the right of the pulmonary valves; behind the sternum, at a place corresponding to the lower border of the third cartilage.

A circle half an inch in diameter would include all the valves, one of only quarter of an inch would include all but the tricuspid.

The position of the heart and its valves having been given in their relation to the anterior of the chest, their position with regard to the back must be described. The exact position varies in individuals very much, as will be seen on comparing the results obtained in the frozen sections of Pirogoff and Braune.

The following account may be taken as indicating the average position of the heart and its valves:—The heart lies between the fourth and eighth dorsal spines, the top of the arch of the aorta being level with the third dorsal spine, the top of the pulmonary artery on a level with the fourth dorsal spine. The pulmonary valves are opposite the fifth dorsal vertebra; the aortic valves are opposite the sixth dorsal vertebra; the mitral valve is opposite the sixth and seventh or eighth dorsal vertebra; the tricuspid valve between the sixth and eighth dorsal vertebra.

The **superficial cardiac region** is usually four-sided, but sometimes triangular in shape; is chiefly formed by the right ventricle, which is exposed to view uncovered by the lungs; lies between the fourth cartilage to the upper border of the sixth rib. It is bounded above and at the sides by the right and left lungs, below by the diaphragm. It measures vertically two inches, horizontally two and a half inches. This uncovered space would be contained within three lines forming a triangle; the midsternal line meeting one line drawn horizontally from the apex, and another drawn upwards from the apex to the level of the fourth cartilage.



The lungs from their upper part descend together, and meet to form a covering for the great vessels of the heart, and they part generally on a level with the fourth cartilage; the left lung receding, and leaving the heart exposed, by a double curve, at first convex, and then concave, when it curves forward again to form a very peculiar tongue, which laps over the apex of the heart and passes downwards, as will be seen by reference to the woodcut on page 57.

They modify by their presence the dull note of percussion, and consequently over all the præcordial surface, excepting the region of superficial dulness, deep-seated or diminished dulness is elicited by percussion over the confines of the heart.



## CHAPTER XVII.

## THE SOUNDS OF THE HEART.

THE heart moves smoothly and noiselessly in its pericardial sac, and in a normal condition no sound is produced by this movement; but when the ear is applied to the præcordial region of a healthy individual two sounds are heard—one corresponding to the impulse or systole, which is called the systolic sound; a second one which follows this, and is called the diastolic sound.

Each of these sounds is followed by a short interval of silence.

The two sounds differ in duration, intensity, pitch, and quality.

The two silences differ in duration.

As regards the relative duration of the sounds and the silences, if an entire period from the beginning of one systolic to the beginning of another be divided into 10 parts—

- 4 will be occupied by the first sound (systolic).
- 1 by the following silence (post-systolic).
- 2 by the second sound (diastolic).
- 3 by the second silence (post-diastolic).

The sounds of the heart are chiefly due to the forcible tension of the valves.

The first sound being caused by the closing of the auriculo-ventricular valves, the tricuspid and the mitral.

The second sound being caused by the falling to of the sigmoid valves, aortic and pulmonary.

(For practical purposes these causes for the sounds of



the heart are sufficient, but it may be well to state that other causes help, viz. :—

For the first sound { The shock of the heart's apex on  
the chest.  
(The bruit of muscular contraction.)

The sounds vary relatively at apex and base.

The first sound, as compared with the second at the apex, is longer, louder, and lower in pitch; strongly accentuated.

It is dull, accentuated, and booming in quality, sharply defined at its beginning, not so much at the end.

The second sound is consequently at the same point shorter, less loud, higher in pitch; it has a sharp clacking quality.

The sounds are slightly different when the ear is placed over the base of the heart.

The first sound is not longer than the second; it loses its booming quality, and the accent is passed to the second, which increases in intensity and ringing sound.

The auricular systole immediately precedes the impulse; in health this contraction gives no sound.

The ventricular systole causes the impulse and the first sound.

The arterial contraction, after the impulse of the heart, causes the second sound by the pressure of the blood on the closing sigmoid valves.

This is followed by a period of rest.

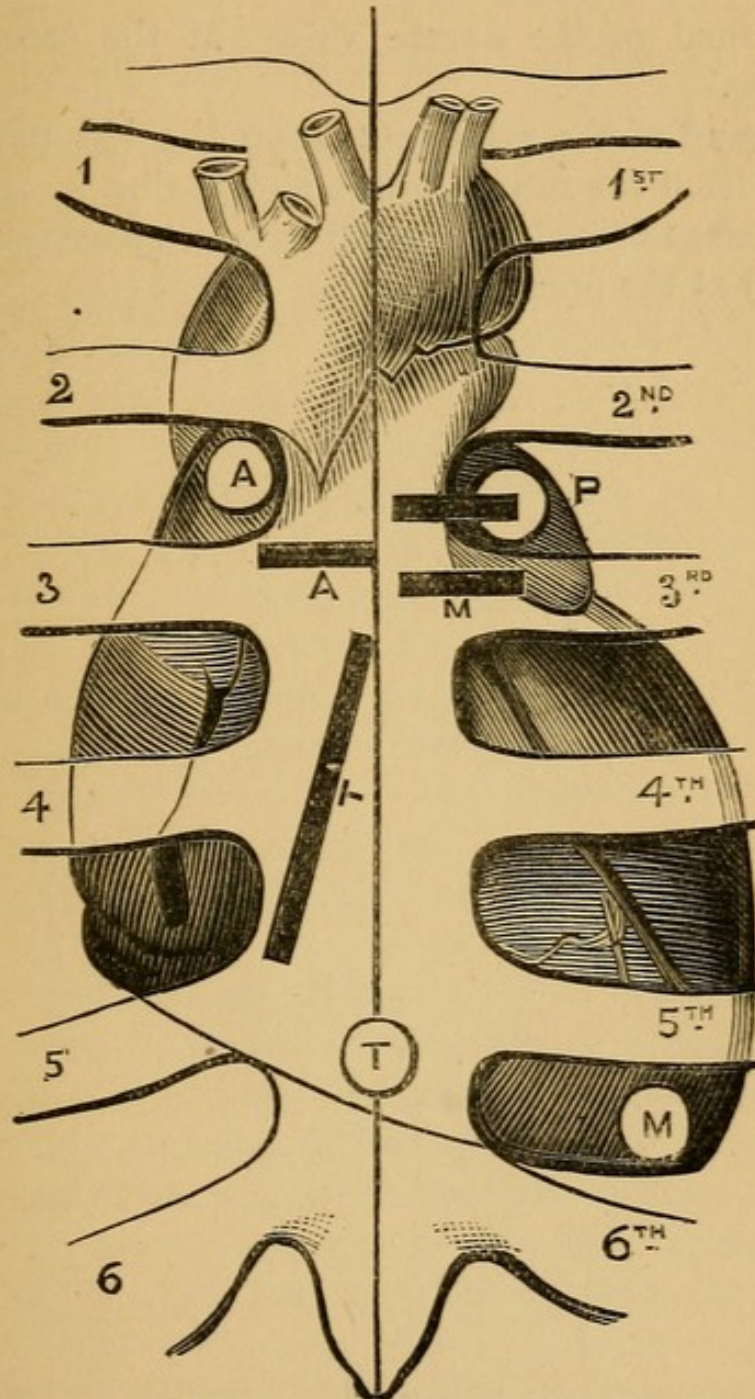
1st Sound....	{ Tricuspid ... Mitral..... }	Shut.....	Ventricular Contraction.
2nd Sound...	{ Aortic..... Pulmonary.. }	Shut.....	Arterial Contraction.
Silence.....	{ Tricuspid ... Mitral .....	Open... {	Auricular Contraction during the last part.

The impulse of the heart is sometimes difficult to determine in relation to the sounds. In health there is no such difficulty, but it may be necessary to decide by other means when the impulse takes place. If the apex beat can be felt the sensation of the impulse will give a ready



means for the recognition of the first sound, but if the apex beat is from any cause indistinct, the finger must feel for the pulse in the carotids, where it is synchronous with the first sound, the radial pulse being perceptibly postponed.

FIG. 24.



The Position of the Heart beneath the Sternum. The Bars indicate the Position of the Valves. The Circles the Sites for Auscultation.

But it must be remembered that the heart has four chambers which empty and refill, and it may be requisite



to discover whether these chambers are acting properly. This is to be done mainly by remembering that sounds produced by a fluid in motion are transmitted in the direction of the current.

The sound of the **pulmonary** valves is best heard at the second left interspace.

The sound of the **aortic** valves at the second right interspace.

The sound of the **tricuspid** along the left part of the sternum from the fourth to the eighth ribs.

The **mitral** being best heard over a space (an inch in diameter) at the apex beat.



## CHAPTER XVIII.

## THE ACOUSTICS OF BLOOD MURMURS.

THE modifications of sound resulting from the passage of air through tubes of different calibre have been considered with reference to lung sounds, the production of sounds in fluids also undergo modification which are made use of in clinical medicine to detect alterations in the heart and its vessels.

Formerly it used to be thought that the sounds heard in heart disease during the circulation of the blood resulted from the friction of the blood-current on the walls of the heart or vessels through which the blood was travelling. Laennec concluded that the abnormal sound (which he likened to that produced by a pair of bellows when in use), and called "bellows sound" (*bruit de soufflet*), was produced like the muscular murmur by muscular contraction or spasm.

This theory was, however, challenged by Corrigan, who substituted another, the gist of his observations being found in the following quotation:—"When an artery is pressed upon, a small stream rushes from a narrow orifice into a larger one; the rushing of the fluid is combined with a trembling of the artery, and the sensation to the sense of hearing is the *bruit de soufflet*."

This is quite in accordance with the experiments of Savart on the alterations in fluids as they pass through small orifices.

If water be allowed to pour, for example, from the top of a shower-bath, or from a cullender, the streams which descend will be observed to be clear and continuous close to the holes, while below they break into a succession of drops, and this liquid vein, *veine fluide*, alternately swells



and contracts in its course, like the bulgings of a vein, forming a vibrating column of water, which will produce a musical sound if the drops be caught on a membrane. The revolution of the fluid into drops begins to take place soon after the fluid passes through the hole, becoming more definite lower down until the drops become completely formed and separate. If under pressure these drops succeed each other with sufficient rapidity a musical note may be heard.

This vibration of fluid passing through a narrow orifice, which is similar to the vibration of air passing through the mouth of an organ pipe, is the cause of sound in fluids; the subject has been of late years further investigated by Chauveau in some experiments on animals. The artery of a horse was torn inside in several places, and the vessel thus roughened to the current of blood, but no *bruit* or sound was produced; but when the vessel was narrowed by a thread tied round it so as to make a constriction in its course, the bellows murmur was heard distinctly, and the vibrations caused in the current were felt. It is easy enough to make a murmur of this kind by pressing upon the jugular vein with the stethoscope, and it is sometimes difficult in ordinary practice to avoid making that very sound artificially, for which the observer is searching, as indicative of disease.

The explanation of the sounds which are heard in anæmia, given by Chauveau, is very interesting and simple; the quantity of blood circulating in anæmia being reduced, the heart adapts itself to the required condition and becomes also reduced in size; this was shown in his experiments on horses, who were the subject of anæmia; the cavities and orifices of the heart under these circumstances became lessened in size, while the large arteries maintained their normal size, being unable to alter in consequence of the rigidity of their walls; hence the blood passing out of diminished orifices into these large vessels is broken up into a liquid vein, and sounds are produced.



In a somewhat similar manner the humming sound produced in the veins, which the French have called the *bruit de diable* (from the resemblance to the noise of a toy which bears the name), may be explained; the size of the jugular vein at the base of the neck (the position which this murmur affects) is kept rigidly constant under all circumstances in consequence of the adhesion of the walls of the vessels at this part of the cervical fascia; when the calibre of the vein above is altered a murmur is produced by the current passing into this rigid dilatation. With regard to musical murmurs in some cases it can be shown that they depend upon the rapidity of the heart's action; a frequent murmur of a whistling character is one heard in nervous and hysterical patients whose heart, when they are being examined, is beating with a surprising rapidity. This murmur abates considerably when the patient becomes more tranquil. This is not, however, the case with all musical murmurs, some of which are persistent and so intense as to be audible some distance from the patient and yet no organic disease may be detected. Walshe, however, states that a musical quality is sometimes given when prominent spiculæ of a vibratile character project into the current. Rapidity of the heart may be used for bringing out the quality of a murmur; and if any doubt exists on such a question it is useful to make the patient pace up and down the room smartly for a short time. The rationale of musical murmurs is a question not yet decided, nor can we at present diagnose their causation. It is necessary therefore to warn the student that cardiac disease is not to be considered as proportionate to the intensity of the murmur, and not seldom it is found that a most dangerous cardiac disease is present which gives no audible sound at all; while one condition of the heart may give rise to murmurs of varying quality, sometimes soft, at another time loud and musical, so that in this department of auscultation also the student must not be misled by trusting solely to his ear.



## CHAPTER XIX.

## INSPECTION, PALPATION, AND PERCUSSION IN HEART DISEASE.

IN the physical examination of the heart, as in that of the lungs, the first method to apply for the determination of its condition is **Inspection**.

Attention is chiefly directed to the position of the apex, which can generally be seen beating, but palpation must aid the eyes to find the precise locality. As long as the apex is beating beneath an intercostal space, eyes and fingers are sufficient to establish its position; but when it is beating beneath the ribs or sternum, and the eyes and the fingers cannot detect its whereabouts, the stethoscope must be used and so moved about as to discover by comparison of the relative intensity of the sound at various points where the sound is loudest.

Pulsation must not be mistaken for the true apex beat, as the apex may be forced from the surface of the chest, and the pulsation of the right ventricle may (for example) be visible.

The apex in health beats in the fifth intercostal space, but it is driven out of place by various conditions of disease.

**The apex is lowered** in enlargement of the heart, and may be depressed down to the eighth rib. Walshe mentions a case in which the apex was  $3\frac{3}{8}$  inches to the left of the nipple.

It is thrown down also by aortic aneurism and those pulmonary diseases which force down the diaphragm: these are emphysema, asthma, and chronic bronchitis, pleuritic effusion, and (in some cases) mediastinal tumour.

**The apex is raised** by all abdominal diseases, tending



to force the diaphragm upwards—*e.g.*, by ascites, distension of the stomach and abdominal tumours—the most frequent cause of its elevation is the adhesion of the pleura and the contraction of a fibroid or phthisical lung.

Pericardial effusion forces the apex of the heart upwards so that it may beat as high as the fourth interspace.

**Lateral displacement** of the apex takes place from the diseased and towards the sound side in effusions of all kinds and the admission of air into the pleural sac from the presence of intrathoracic tumours from growth and collections of matter or hydatid cysts in the liver; and from the sound towards the diseased side in contracting lung the result of fibrosis or arrested phthisis. In rare examples the displacement is congenital (congenital transposition of the viscera).

The heart is thrown forward by tumours of the posterior mediastinum, aneurism and cancer, and thrown backward by tumours of the anterior mediastinum, cancer and abscess, and by effusions into the pleura and pericardium.

The apex changes its position according to the posture assumed by the patient, but permanent displacement of the heart may take place solely from decubitus on one side. A most remarkable instance occurred lately under my observation.

A woman who had suffered from sacro-iliac disease and abscess of the left side for some months died after nineteen months' illness. She had been compelled to lie always upon the right side. After death the heart was found lying over to the right side of the mesial line, the apex being situated under the sternum, and the axis of the heart being directed upwards towards the right shoulder. The apex was on a level with the fifth interspace, and the heart was not turned, the right ventricle being in its proper place. There was no disease of the lungs further than this, that the right lung was in a condition of slight hypostatic engorgement.

The right lung weighed 18 ozs.; the left,  $9\frac{1}{2}$  ozs.



*Synoptical View of Displaced Heart.*

Apex Depressed	Emphysema Bronchitis Hypertrophy of Heart Aneurism Pleuritic Effusion
Apex Raised	Diseases of Abdomen Fibroid Phthisis Pericardial Effusion
To the Left of Nipple	Fibroid Phthisis Pleuritic Effusion } of the Right Side Pneumothorax } Intrathoracic Tumours
To the Right of Nipple	Right Fibroid Disease Left Empyema Left Pleuritic Effusion
To the Right of Sternum	Right Fibroid Disease Left Hydrothorax Left Hydropneumothorax.

**Pulsation of the veins** is a sign which must attract attention during inspection.

Plethora of the right auricle, preventing the free return of blood to the heart, is indicated by waviness in the jugular current. If the systolic impulse be distinct, the tricuspid valve is incompetent.

Marked pulsation occurs in obstruction in the pulmonary artery, notably in pulmonary thrombosis and embolism. It occurs also as a result of old-standing emphysema and mitral disease.

A plethoric condition of the veins is found in beer-drinkers—from the copious imbibition of fluid; and this condition is very conspicuous in the veins of the extremities.

**Pulsation of the arteries** is marked in atheroma, and in regurgitation through the aortic valves.

Inspection must also appreciate any bulging or alteration of the chest-wall. In early life this is caused sometimes in a very marked degree by cardiac hypertrophy, the ribs being forced forward by the enlarged heart.



Retraction of the skin over the apex beat after the impulse of the heart has been delivered has been considered diagnostic of adherent pericardium, but this may follow the pressure of the atmosphere acting from without inwards, and cannot be depended upon as characteristic solely of that disease.

It may be mentioned here that in displacement the heart is often rotated in such a manner that when the apex is pushed over to the right the left ventricle comes forward, the right ventricle turning backwards, and when it is pushed over to the left the right ventricle comes to the front.

It is a common notion to look upon the movement of the heart to the right side taking place, as if the heart was pivoted above, and the apex moved to its allotted place like the end of a pendulum; but although this does occur in extreme cases (of extensive effusion, for example), yet the most usual form of dislocation is the movement of the base upwards, the apex being situated under the sternum, the push being exerted upwards towards the right shoulder.

**Palpation** is used to estimate the impulse, ascertain the position of the apex, and detect abnormal pulsations and thrills.

The impulse is increased in functional excitement, in angina pectoris, cardiac asthma, pericarditis, endocarditis, hypertrophy of the heart, by sexual vice and alcoholic intemperance, and in reaction after hæmorrhage.

The impulse is weakened by debilitating influences, prolonged illness, fevers, dilatation of the heart, fatty degeneration, and the deposit of fat about the heart, effusion into the pericardium and emphysema.

Pulsation of an abnormal character may be due to some other cause than the apex beat. It is frequently to be seen in the epigastric region from dilatation of the right ventricle, and is a common sign of emphysema.

Sometimes this form of pulsation is such as to induce pulsation of the liver throughout the whole right hypo-



chondriac region; a sign also visible in aneurism seated beneath the liver.

Pulsation of a very extended nature may be often observed when the heart is uncovered by retraction of the lung; this is an ordinary result of phthisis with adherent pleura, and occurs most frequently on the left side.

It is difficult in some cases to determine the exact nature of the pulsation, and how it is caused, and whether it be systolic or diastolic.

To solve this question a bristle carrying a piece of paper (as suggested by Balfour) may be attached by a bit of wax to the place, and its movements compared with another placed over the apex.

**Thrills** are caused by the palpable vibrations of the liquid vein or sonorous blood-current, or by the friction of pericarditis. They have the same relation to blood sounds that vocal fremitus bears to vocal resonance, in being due to palpable vibration.

They are caused by the transmission of vibrations from the blood-current to the containing walls, and, according to Bergeon, are due to rough murmurs in a thin and elastic tube.

As far as my observations go, they are usually present when there is much dilatation with thinning of the walls of the heart, and may be felt over the auricles as well as the ventricles.

**Percussion** is valuable for defining the size of the heart or the presence of foreign matter in the pericardial sac.

Hypertrophy of the heart is detected by the increased area of dull percussion which extends chiefly in the direction of the left axilla.

Pericardial effusion gives rise to an extended area of dulness on both sides, and the region which must be specially examined is the fifth right interspace.



## CHAPTER XX.

## SOUNDS OF THE HEART IN DISEASE.

THE sounds of the heart may by disease be increased or diminished in intensity, altered in rhythm, or modified by the presence of adventitious sounds, termed **murmurs**.

**Increased intensity** of sound is caused by increased action of the ventricular walls, which may simply be functional, the result of excitement, and is of frequent occurrence in nervous and hysterical patients. When it occurs with a bumping, irregular impulse of the heart it suggests sexual intemperance and vice. It also occurs from a dilated condition of the ventricles, with or without hypertrophy.

In these forms of disease it is the first sound which is specially intensified and accentuated.

Increased intensity of the second sound indicates excessive arterial tension, which may be caused from within the vessel by obstruction to the circulation, or it may be due to pressure outside the vessel, as in tumours embracing the vessel. This accentuation of the second sound is found in children with scrofulous glands; in adults with aneurism, of which the sharp clacking sound with which the valves close affords an early sign.

**Diminished intensity** of the sounds is caused by weakening of the muscular walls of the heart from adynamic diseases, fevers, cancer, and those diseases which cause emaciation and atrophy generally. Fatty degeneration of the muscles, or the accumulation of fat about the heart, are important causes of this modification.

**Distance of the heart sound**, the sound being indis-



tinct and heard at some distance from the ear, is caused by the interposition of fluid, as in pericardial and pleuritic effusions, and by concentric hypertrophy of the ventricular walls of the heart.

**Irregularities of rhythm** are evinced by reduplication of the sounds, or by intermittence.

**Reduplication** of the sounds is caused by the non-synchronous action of the two sides which no longer work in unison.

The first sound is doubled when the tricuspid valve is closed behind time, and the second sound is doubled when the aortic valves are shut before time.

Sounds are reduplicated at any age, and in health as well as disease.

A very common cause of reduplicated apex sounds is a distended stomach, which sometimes occasion intermittence; the same condition is induced in many individuals at the time of an approaching thunder-storm.

Reduplication may be simply functional, or it may be caused by organic disease, and indicates a non-synchronous irregularity in the filling and emptying of the cavities of the heart.

**Intermittence** in the sounds are due to two classes of causes, one being extra-cardiac, and the other intra-cardiac.

Under the first heading belong nervous conditions from overwork and electrical states of the atmosphere, especially during the approach of a thunder-storm, distension of the stomach, and dyspepsia, and gouty irritation.

Under the second is included those severe forms of mitral stenosis of which intermittence is a marked symptom.

These two classes of conditions must be differentiated by special attention to the character of the pulse, and the general signs and symptoms of the case.

**CARDIAC MURMURS.**—Adventitious sounds which accompany the action of the heart, and are heard over



the præcordial region, are called **cardiac murmurs**; they are caused by some altered condition affecting the passage of the blood, or by roughening of the pericardial surfaces.

In the first case they are **endocardial**.

In the second case they are **exocardial**.

**Endocardial murmurs** are the result of organic or functional disease, and are due to—

Obstruction from disease.

Obstruction from imperfect valvular development.

Dilatation from pressure, morbid and mechanical.

Rupture of valves from injury.

Constitutional conditions causing hæmic murmurs.

**Exocardial murmurs** are caused by disease affecting the pericardium.

**Endocardial Murmurs.**—Obstruction generally arises from endocarditis, frequently the result of rheumatism, or more rarely of previous laceration of the valves; in all cases the growths which are formed on the surfaces of the valves are largely fostered and increased by attrition with the opposite parts of the valve and the blood-stream.

Obstruction from imperfect valvular mechanism may be a congenital malformation, or as appears very probable it may be due to an arrest in development, or to the prevention of growth from structural peculiarity, whereby the heart fails to meet the requirements of the growing body.

In such a case the valve, or valves, which is quite adequate for the vital requirements of childhood, becomes quite incapable of conforming to the rapid development of the whole frame, which sometimes takes place with a surprising rapidity, and hence symptoms of cardiac disease become developed during the early period of adult life.

Dilatation of the heart's cavities, valves, or vessels arises from excessive and prolonged strain—as, for example, running, carrying weights, and those exercises which call into play the muscles of the lower extremities.

Rupture of valves occurs usually from sudden exertions,



lifting weights, and momentary strain of an extraordinary nature.

Hæmic murmurs are generally found in anæmia, in fevers, and occasionally in endocardial irritation from rheumatism.

Exocardial murmurs are caused by roughening of the pericardium, the result of rheumatism, and other forms of pericarditis, of cancer, and of tubercle.

The quality of cardiac murmurs may be expressed in various terms—*e.g.*,

Soft blowing bellows murmurs (*bruit de soufflet*).

Rough grating sawing sounds (*bruit de râpe de scie*).

Musical murmurs (*bruit de soufflet musical*).

Of these qualities, the first is common, the last rare.

The important characteristics to note in any particular murmur are—

1. The time it occupies as regards the beat.
2. The site at which it is most intense.
3. The direction in which it is transmitted.

1. As regards the period to which a murmur belongs, all endocardial murmurs may be considered as included in the three following:—

Systolic, formed during the ventricular systole.

Diastolic, „ „ „ „ diastole.

Presystolic, „ „ „ auricular systole.

2. As regards the site at which they are heard with the greatest intensity.

The area for aortic sounds is very extended, and a murmur created at the valves may be heard more or less distinctly over the sternum. The maximum intensity is heard in the second right interspace, or over the second right cartilage. It may be heard at the xiphoid cartilage.

The area for mitral sounds is much more limited, and confined to the position of the apex, from which it seldom travels far.

The area for pulmonary sounds is a small space in the second left interspace, close to the sternum.

The area for tricuspid sounds is above the xiphoid



cartilage, and, according to my experience, extends over to the right side of the midsternal line.

3. As regards the direction in which the sound is transmitted, that will be influenced in great measure by the direction of the current, but the aortic murmur, whether systolic or diastolic, is usually propagated along the aorta.

Exocardial murmurs are caused by the action of the heart, and their position, as external to the heart, is the chief point which demands diagnosis.

Murmurs due to disease of the aortic valves are simple in cause and easily understood; they may therefore be fitly considered first.

They are due to vegetations, contractions, or rupture of the aortic valves, and may be systolic or diastolic.

**Aortic Systolic Murmur.**—A direct onward murmur produced by narrowing of the aortic orifice is formed during the contraction of the left ventricle.

It is therefore **systolic**.

**Site.**—This murmur is heard with greatest intensity at the base of the heart over the second right interspace or the second right cartilage.

**Transmission.**—It may be traced upwards along the course of the aorta, and sometimes extends for a considerable distance from its origin. It may be heard in the secondary arteries.

**Causes.**—In organic disease it arises from inflammatory contraction and thickening of the aortic valves, from rheumatism or following rupture of the valves, from congenital malformation, and from aneurismal dilatation above the valves.

In functional disease it is heard when no permanent alteration of the valves is present, as in anæmia.

It may be heard in endocarditis during an early stage of rheumatism.

This murmur must be looked upon less unfavourably than aortic regurgitation. Aortic obstruction without regurgitation is a rare form of heart disease; and it must be pointed out that hypertrophy of the ventricle takes place in proportion to the obstruction.



As a rule this murmur occurs in company with that of regurgitation, or with an alteration of the second sound, which is either muffled, or feeble, or strongly accentuated with the clack of a stiffened valve. If the presence of aortic aneurism be suspected and the amount of hypertrophy be great, prognosis must be made with caution, and the murmur may be removed from the category of least unfavourable to that of most dangerous.

The student must bear in mind that, notwithstanding the general rule which may be applied to the aortic direct or systolic murmur, isolated cases occur in which very extensive disease of the aortic valves has been found after death which has been indicated during life by only a direct systolic murmur, and the disease has been tolerated by the patient without proportionate symptoms within a short time of death. Cases of this kind will be found recorded by Graves, Stokes, and Peacock.

**Aortic regurgitant murmur** occurs with the second sound of the heart, and is therefore **diastolic**. It is caused by failure of the valves to stop the back current of the blood, the valves being incompletely blood-tight. In quality it varies, generally low in pitch and blowing, but it may be loud, hissing, whistling, or musical, and it may be accompanied with thrill.

**Site** is not uniform, its maximum intensity is usually over the second right interspace, but it may be heard over a large area, and may be even louder at the apex than the base.

**Transmission.**—It has an extensive area through which it is conducted, and it may be heard downwards along the sternum as far as the xiphoid cartilage. It may be heard all over the præcordial surface or lost at a short distance from the valves.

**Causes.**—It is due to permanent patency of the aortic valves, from ossification or ulceration, from rupture, from stretching of the aorta.

This murmur is a most important one and must be looked upon as always dangerous. It is never functional,



and therefore when heard organic alterations are always present, and the liability to sudden death is very great.

Regurgitation induces dilatation of the heart.

The murmurs of the pulmonary valves are formed in the same way as those of the aortic, but they are far less frequent and not so formidable.

**Pulmonary systolic murmur** is a direct onward murmur and accompanies the systole. It is therefore a systolic murmur. It is seldom rough or loud.

**Site.**—It is more superficial than the aortic murmur, and is most intense at the second left interspace close to the sternum on the left side.

**Transmission.**—It takes an upward direction to the left towards the middle of the left clavicle.

**Causes.**—In consequence of the freedom from permanent injury which these valves enjoy in rheumatism and their non-liability to rupture, the results of inflammatory disease are very rare. The chief causes of this murmur are (if organic and permanent) of congenital origin, the orifice being narrowed.

But the more frequent causes are inorganic or external. Hæmic murmurs from anæmia and at the end of an attack of rheumatism are common in this artery, and murmurs from pressure or displacement.

It is common in children (it may be artificially produced by the pressure of the stethoscope) as the result of the pressure of an enlarged gland, and it is by no means infrequent in cirrhosis of the lung with displacement of the heart and contracting phthisis.

**Pulmonary regurgitant murmur** is extremely rare, and its description need not be attempted.

One case of the kind was reported by Dr. Wilks, and another has been published by Dr. Begbie.

A diastolic murmur, not aortic, has been heard at the base of the heart and on the left side in cases of aortic aneurism opening into the pulmonary artery.

The murmurs just described are sometimes termed basic murmurs, because of their site of maximum intensity.



We now come to another class, the apex murmurs, which are more difficult and complicated.

The student must bear in mind that the auriculo-ventricular valves are closed in the systole of the ventricles, and consequently that a murmur caused by the systolic action of the ventricles drawing blood back through the unclosed mitral or tricuspid valve is a regurgitant murmur, although it is systolic.

**Mitral regurgitant or systolic murmur** is one of the most common murmurs, and is heard during the systole of the ventricles; it is therefore termed systolic, but, inasmuch as it is due to the backward current of the blood through the mitral orifice, it is regurgitant. It is generally soft and blowing in quality, but sometimes musical.

**Site.**—The apex of the heart is the situation of this murmur.

**Transmission.**—The area of this murmur is sometimes very limited, but it is to be heard conveyed along the ribs to the lower angle of the left scapula.

**Causes.**—It is a frequent result of rheumatic endocarditis, and sometimes (rarely) of rupture, but this murmur may occur without disease of the valve, and it may result from the dilatation of the ventricular wall, or from simple dilatation of the auriculo-ventricular opening.

**Tricuspid regurgitant or systolic murmur** is formed in the same way as the mitral regurgitant sound, by the systolic action of the right ventricle forcing the blood through the imperfectly-closed tricuspid valve.

**Site.**—This systolic murmur is heard over the xiphoid (ensiform) cartilage and towards the right side. It is seldom heard very high up. It is generally of a blowing character.

**Transmission.**—It appears to me to be generally transmitted towards the right side, and this especially occurs if the right auricle and ventricle be out of place either from distension or from dislocation.

**Causes.**—The principal cause is distension from intra-



pulmonary pressure, and it is rare to examine fatal cases of prolonged pulmonary disease without finding dilatation of this valve.

It is found in emphysema, bronchitis, and cirrhosis of the lung, and, according to my experience, is not an infrequent result of prolonged walking, lifting of weights, &c., as induced by certain occupations.

**Mitral presystolic or direct murmur** is caused by the systolic auricular contraction during the passage of the blood from the left auricle into the right ventricle. It occurs just before the systole, and is never heard unless stenosis or narrowing of the mitral valve is present.

It is often very harsh in quality, and accompanied by thrill.

Its name, presystolic, was first applied by Gendrin in 1841. It precedes and runs up to the first sound of the heart, but it is often difficult to time its exact period as regards the heart beat; it is necessary, then, to check off the heart beat by putting the finger on the carotid pulse, which occurs with the beat; the radial being postponed or felt some time after. It is not a very rare murmur, and is always of unfavourable import.

**Site.**—This murmur is very circumscribed in area of extension, and is confined to the apex of the heart, not travelling back to the left shoulder-blade as in the case of mitral regurgitation.

**Transmission.**—The characteristic condition of this murmur is its non-transmission, the area being very limited.

**Causes.**—It is generally produced by rheumatic inflammation of the valve, leading to agglutination and stiffness; the result being usually what is known as a button-hole mitral, a small opening in a membranous valve, scarcely admitting the tip of the little finger.

Besides this there is another condition, the funnel-shaped valve, which is long and contracted, but suggests, not inflammation, but rather imperfect growth, dependent upon congenital malformation.



The button-hole mitral is a very dangerous form of heart disease, and the presystolic murmur always serious.

**Tricuspid presystolic murmur** has been heard, but it is extremely rare in ordinary practice.

With regard to the frequency of murmurs, it may be said that murmurs of the left side are common; of the right side, rare; as regards single murmurs, the following represents their order of occurrence—regurgitant murmur of the mitral, aortic, and tricuspid, obstructive murmur of the mitral and aortic.

The relative danger of the different murmurs can only be estimated approximately, and they may be put in the following order:—

Regurgitant murmurs on the left side are serious, and may be followed by sudden death.

Obstructive murmur of the mitral valve is also very serious; of the aorta less dangerous.

The worst murmurs are no doubt the aortic regurgitant and the mitral presystolic.

**Pericardial Sounds.**—The chief and most frequent sounds caused by diseases of the pericardium are those of friction, which are imitated by placing the stethoscope in one hand and rubbing the back of that hand with the finger of the other.

As a rule these sounds are superficial—that is, they seem to be heard directly under the ear.

They may accompany the systole and diastole together or singly.

Moderate pressure generally intensifies them; strong pressure prevents them.

They may also have their site altered by change in the position of the patient; this is a very important diagnostic point. The sound sometimes disappears when the patient sits forwards, this probably depending upon the intervention of fluid between the surfaces.

They are accompanied with fremitus, which can hardly be mistaken for thrill.

These sounds vary much in intensity, and in slight



degree may only be heard as a slight grazing; in greater degree they become creaking, grating, and scratching.

These sounds are produced by pericarditis, fluid in the pericardium, attrition from intense pressure, tubercle and cancer of the pericardium, which conditions are rare, but which certainly give rise to friction sounds.

## SYNOPSIS OF CARDIAC MURMURS.

*Systolic Murmurs.*

<i>Site.</i>	<i>Transmission.</i>	<i>Cause.</i>	<i>Valves Injured.</i>
2nd Right Interspace. Base.	Along the Aorta to the Right.	Obstruction.	Aortic.
2nd Left Interspace. Base.	Towards the Left Clavicle.	Obstruction.	Pulmonary.
Apex.	Left Axilla and Scapula.	Regurgitation.	Mitral.
Lower Sternal Region. Apex.	Right of Sternum.	Regurgitation.	Tricuspid.

*Diastolic Murmurs.*

2nd Right Interspace. Base.	Down the Sternum.	Regurgitation.	Aortic.
2nd Left Interspace. Base.	Downwards (?)	Regurgitation.	Pulmonary.

*Presystolic Murmurs.*

Apex.	Not Transmitted.	Obstruction.	Mitral.
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**Pneumopericardium** is a very rare condition, occurring after perforation of the pericardium, and is recognised



by a churning splash or gurgling sound with the heart's action. Dr. Stokes describes the sound as a mixture of various attrition murmurs, with a large crepitating and "gugling" sound, while to all these phenomena was a distinct metallic character.

It is an extremely rare condition.



## CHAPTER XXI.

## THE PHYSICAL SIGNS OF CARDIAC DISEASE.

HAVING now described the murmurs which accompany the heart's sounds from valvular and other diseases, I shall proceed to group together the physical signs which enable us to form a diagnosis on any special form of disease presented to notice, and as in the case of pulmonary signs, so here it will be convenient for reference to adopt the alphabetical order, and conditions will be included which are connected with or bear upon the subject.

**Anæmia.**—This condition is easily recognised by inspection, the pale pearly-white skin not being mistakable, this being the ordinary colour of simple anæmia.

Anæmia perniciosa, however, is generally characterised by a pale primrose colour, sometimes of a very sallow hue.

**Symptoms.**—Debility with dyspnœa on exertion, and a tendency to faint.

**Pathology.**—Pallor of the organs is the only sign; the blood having a very scanty proportion of red corpuscles.

**Signs.**—The murmurs heard in anæmia are generally soft, of a slight blowing quality, sometimes made very harsh and whistling with exertion or excitement.

They are invariably systolic basic murmurs, and are either heard in the aortic or in the pulmonary area.

They are accompanied by various murmurs in the jugular veins, and have a tendency to alter.

The pulse gives no indication of disease.

**Angina pectoris** is a disease capable of producing the most acute anguish, terminating in sudden death.

**Symptoms** are derived entirely from the description of



the patient; the chief symptom being pain of a most intolerable character, felt at the lower sternal region, with pain referred to the middle of the arm and numbness. To this is added a sense of impending death, due to oppression and a sense of suffocation, but it is not accompanied with difficult action of breathing.

**Pathology.**—Various conditions are associated with these symptoms, flabby dilatation, ossification and rigidity of the coronary arteries, aneurism, and valvular disease; but, inasmuch as none of these are constantly present, and they are found without angina, the condition appears to be a neurosis (an anæmia it may be) of the cardiac plexus.

In John Hunter's case the muscular tissue of the heart was very pale, and the coronary arteries were converted into long rigid tubes.

**Signs.**—There is no sign special to this disease, and cases occur in which auscultation is quite at fault; no anomalous sign may be present; still disease may be detected, and the physician must carefully examine for feebleness of pulse, irregularity of action, and especially to the percussion of the upper sternal region for the detection of aneurism. Gairdner is of opinion that small aneurisms very near the heart are more apt to induce angina than larger tumours at a distance.

**Aneurism of the heart** is a localised sacculated pouching of a left cavity of the heart. The right cavities are never affected in this manner. No special symptoms or signs can be attributed to them. In some of the cases the condition was only detected after death, in others (22 out of 27) palpitation, tumultuous heart action, irregularity of pulse are reported; and the presence of valvular murmurs. The condition may occur at a very early age, this being due to its connection with rheumatism; it is usually found as a pouch at the apex, varying in size from a nut to a hen's egg, or even larger. Aneurisms of this kind also are found involving the left auricle and the aortic and mitral valves.



**Aortic Disease** (Valvular).—The diseases which affect the aortic valves are known as obstruction and regurgitation.

**Aortic obstruction** simply, without regurgitation, is not very common, and may be present, even in excessive extent, for a long period without inducing distress. This has indeed occurred when the opening was not larger than a pea.

**Symptoms** are due to insufficient filling of the systemic arteries, but it may exist for some time without causing constitutional disturbance, unless the other valves, notably the mitral, give way under pressure.

**Pathology.**—Inflammatory roughening of the valves from rheumatism and chorea; it also occurs from congenital malformation, the valves being blended together to form a diaphragm or septum, perforated in the middle. Hypertrophy of the left ventricle is an invariable result of this form of disease.

**Signs.**—A systolic murmur, heard with greatest intensity at the second right interspace, and transmitted along the course of the aorta.

If hypertrophy of the heart be present to any extent, the apex beat may be thrown down, the impulse excessive, and the sounds generally indistinct.

The **pulse** is small, and would be indicated by the sphygmograph with a sloping upstroke and a round top.

Percussion must define the area of the heart, and discover any extension in size from hypertrophy. A systolic thrill is often present.

The simple obstruction murmur is not common, and comparatively with other cardiac murmurs deserves a less unfavourable prognosis, but it is frequently associated with the regurgitant murmur, a complication of very grave anxiety.

The systolic murmur is often heard in cases of anæmia and rheumatic endocarditis, in pericardial effusion and aneurismal or altered conditions of the aorta above the valves from displacement or pressure.



The diagnosis of organic disease is materially aided by the diagnosis of hypertrophy.

**Aortic Regurgitation.**—This is one of the most dreaded diseases of the heart, inasmuch as it leads frequently to sudden death. The general symptoms are severe palpitation, induced by the slightest exertion, or by distension of the stomach, dyspnœa on exertion; the capillaries are evidently scantily filled, and the general complexion is sallow and bloodless. Neuralgia and angina are frequently associated with this disease, together with sleeplessness and much agitation. Dropsy and cyanosis are generally absent.

**Pulse** is sudden, abrupt, flipping, or jerking in quality, the beat being immediately followed by a sudden collapse. It has been called a water-hammer pulse; the sphygmographic upstroke being very high, followed by a rapid fall, the valves not being competent to support the column of blood.

**Pathology.**—It may result from rheumatic ulceration of long standing, or more frequently from calcareous degeneration, and from congenital malformation. In men it is a condition following strain, rupture of one or more of the valves not being uncommon.

**Physical Signs.**—A diastolic murmur at or below the level of the aortic valves, heard along the lower part of the sternum, sometimes most intense at the xiphoid cartilage. It is transmitted above the valves to a considerable distance.

This murmur is generally preceded by a systolic murmur. If the diastolic murmur be inaudible in the carotid arteries, it is accompanied by a systolic murmur, heard best in the aortic area. This signifies much obstruction, less incompetence.

If, on the contrary, the diastolic murmur be audible in the carotids, it is preceded by a loud systolic murmur there, and this condition indicates much incompetence, little obstruction. (Balfour.)



In advanced cases the beat is hypertrophied and dilated, and the mitral valve incompetent.

The usual result of regurgitation is dilatation, to which may also be added hypertrophy; so that in this state of the valves an enormous amount of blood is thrown at each impulse into the arteries. Hence the visible pulsation of the arteries of the neck and head, the vessels bounding up under the skin; the arteries are also lengthened by the action, and hence the term "locomotive pulse." In the brachial and radial arteries the pulse is much increased by raising the arm, and the same effect is produced in the lower arteries by the patient lying down.

The form of valvular disease is one of the most serious, and is that from which sudden death occurs, and it has been known to take place from the most trivial excitement, even from speaking, eating, or walking.

**Asystole** is a term which was first suggested by Beau (*asystolie*) to indicate the inability of the ventricle to completely close and empty its contents into the aorta. This may occur in cases of valvular disease, but dilatation is its most frequent cause; not only is the cavity increased and the weight to be moved greater (from the greater quantity of the blood received into the enlarged cavity) but the muscle is thin, and less able to cope with its task.

The physical signs of this impending condition are diminished intensity of heart sound, hampered action, accumulation in the right auricle, with a tricuspid murmur and a feeble wavering pulse.

**Atrophy** of the heart is generally a result of wasting disease, and may be found in emaciation after cancer, and some forms of phthisis. As far as the pulmonary disease is concerned, the heart is enlarged and increases in size, but atrophy of the heart appears to be proportionate to the emaciation. On looking over my notes of 178 fatal cases of phthisis and other chest diseases, I find sixteen in which the heart weighed less than eight ounces and a half,—all, with two exceptions, being



associated with phthisis; but out of these sixteen, eleven cases showed signs of amyloid, some of them to a very great extent, the heart weighing from eight ounces and a quarter to five ounces and three-quarters. In the other cases of phthisis the heart weighed nine ounces or over.

**Bronchial compression** is sometimes caused by dilatation of the left auricle. Cases have been recorded (first by King) in which the left bronchus was almost obliterated by such pressure.

In a case reported by Friedreich, a loud humming sound was heard with inspiration and expiration over the whole left side of the chest.

**Cheyne-Stokes respiration** is a peculiar form of dyspnoea, first described by Cheyne in 1818, and subsequently described by Stokes in the following terms:—“It consists in the occurrence of a series of inspirations increasing to a maximum, and then declining in force and length until a state of apparent apnoea is established. In this condition a patient may remain for such a length of time as to make his attendants believe that he is dead, when a low inspiration, followed by one more decided, marks the commencement of a new ascending and then descending series of inspirations.” This form of breathing is found in fatty heart, valvular disease, dilatation, and aortic atheroma.

**Chorea** is a disease closely associated with rheumatism, and a frequent cause of inflammatory disease of the heart. Out of 180 cases of acute rheumatism (recorded by Sibson), twenty-one had chorea; fifteen of these had pericarditis; fourteen had endocarditis; pericarditis and endocarditis being together present in thirteen.

**Dilatation** of the heart is caused by the yielding of weakened walls to excessive pressure, and arises from various debilitating influences, which weaken the muscular tissue of the heart—*e.g.*, anæmia, fevers, obesity from pulmonary obstruction—*e.g.*, emphysema, cirrhosis, &c., from valvular regurgitation, and from prolonged strain.

**Symptoms.**—Palpitation and liability to fainting; rapid



action of the heart from trivial emotions and excitement, and by distension of the stomach. Passive congestion of all the parts supplied, which pour their blood into the dilated cavity, leading to enlarged veins, cyanosis, and dropsy. Congestion of the lungs inducing dyspnoea; congestion of the liver, leading to jaundice and renal congestion with albuminuria. Piles and diarrhoea are frequent symptoms.

**Pathology.**—All the heart's chambers (or only some) may be dilated. It is generally accompanied with some increase of the muscular wall. The walls are often flabby, and the tissue pale. The orifices of the heart are also distended.

**Signs.**—Percussion shows extended area of dulness, and a circular form is assumed by the heart. The area of dulness has been known to extend as high as the first, as low as the seventh rib.

Impulse diffused over a large space and weaker than normal; occasionally causing undulations. Heart's sounds are weakened; the first sound being short and raised in pitch.

**Pulse** feeble and quick, especially under exertion. If the amount of dilatation is excessive, and includes the valves to any extent, soft blowing murmurs may be heard and thrills localised according to the valve area. If the tricuspid be much enlarged, which is common enough in pulmonary obstruction, the jugulars become obstructed and pulsate.

**Embolism** occurs from the removal of small pieces of fibrin from a ragged or inflamed valve surface, the clot passing away into the blood current and being impacted in some vessel.

As regards the heart, this condition occurs from disease of the mitral or aortic valves and the embolus may pass into the lungs by the bronchial arteries and into the brain by the cerebral arteries, giving rise in the latter case to right hemiplegia with or without aphasia.

**Pathology.**—Wedge-shaped blocks are formed in any



organ whose blood-supply is thus plugged. When an artery of the extremity is occluded gangrene follows.

**Fatty degeneration of the heart** results from fatty alteration of its muscular fibre. The most frequent causes of this disease are old age, alcoholism, gout, and wasting diseases of all kinds.

**Symptoms.**—Syncope, vertigo, and dyspnœa are the most common. As regards respiration it is in this form of cardiac disease that the Cheyne-Stokes dyspnœa occurs.

Surface pale and sallow, disposition to melancholy, and great indisposition to exertion of any kind.

**Pathology.**—The muscular tissue of the heart is pale, flabby, and soft, sometimes so friable and greasy that the finger can go through it with very little pressure. Under the microscope fat globules are seen in abundance filling the muscular fibres. Sometimes the muscles are pale and streaky, resembling the markings on a partridge's breast.

**Signs.**—Percussion gives no indication. Impulse weak and often indistinct, a feeble, very dull short first sound, and a feeble but accentuated second sound are the chief signs that indicate this important lesion.

Pulse very small, compressible, and feeble; often irregular.

This is a dangerous condition, as it leads in many cases to sudden death by syncope, or (rarely) by rupture of a ventricle and oozing of the blood into the pericardium.

**Foramen Ovale open** is a condition leading to admixture of the blood in the right and left sides of the heart. It is a cause of cyanosis or morbus cœruleus, but unless the valve be permanently open this condition does not arise.

**Symptoms.**—Blueness of surface more or less marked, the hue being of a mulberry colour, especially marked in the extremities and lips, and increased by cold.

**Signs.**—A double murmur, of a soft blowing quality, loudest at the base of the heart, and heard half-way down, but inaudible at the apex. The pulse gives no indication.



**Fœtal Heart** is rarely heard before the fifth month. It may be heard like the indistinct ticking of a watch; generally it is best heard between the fifth and sixth month. The site over which it is heard is variable, according to the position of the fœtus, generally on the left side, sometimes in the umbilical region, sometimes in the right side. The number of pulsations ranges from 120 to 180.

The placental murmur is variable in character, and resembles the sounds produced by pressure on a large vessel. It occurs first usually at the fourth month. It has no special situation.

**Gout** leads to irregular and feeble action of the heart, often of a spasmodic kind, associated with dyspepsia, fatty degeneration of the heart, and disease of the liver and kidneys.

Intermittence of heart's action, paroxysms of pain with symptoms of angina and occasionally sudden death are the result of the degenerative changes in the heart and vessels from gout.

Irritable asthma and a peculiar form of bronchitis are also caused by gout, and if the diathesis be not recognised and the proper remedies exhibited the disease may persist for some time.

**Hypertrophy of the heart** is the result of healthy reaction against stress of work, increased work demanding increased growth. It results from valvular disease, especially aortic obstruction, from kidney disease as a result of obstruction in the arterioles, consequent upon a diseased state of their walls and in pulmonary obstruction.

**Symptoms** from hypertrophy alone are by no means marked, and those generally given are derived from the primary cause, or result from hypertrophy and dilatation combined.

**Pathology.**—Great enlargement, thickening and hardness of the muscular walls of the breast, so that a heart may weigh twenty-four ounces.

In renal disease the hypertrophy is confined to the



left ventricle, the right side sharing very little in the increase.

**Signs.**—Inspection shows often a bulging of the præcordial wall, but chiefly in early life. Palpation detects a very forcible and heaving action of the apex stroke due to the fact that an extended area of ventricle delivers the stroke, also with increased force. Percussion marks out by dulness an increased area, sometimes as low as the seventh left interspace. This area of dulness may be found behind; the heart, pressing upon the lung, preventing respiration, and thus pleuritic effusion may be simulated.

The sounds of the heart are altered, the first sound being dulled but prolonged, the second sound is generally loud. Reduplication of the first sound is also met with occasionally.

Pulse: full, hard, prolonged.

**Hydropericardium**, or dropsy of the pericardial sac, occurs as a sequence of general dropsies, or is the result of pressure upon the pericardial veins from aneurism or tumour.

The symptoms are discomfort and a sense of weight in the region of the heart, with dyspnœa and feeble pulse. The physical signs are those of effusion into the pericardium described below.

**Mitral diseases** are regurgitant or obstructive, the former arising from imperfect closing of the valve, the latter from narrowing of the orifice.

**Mitral regurgitation** may follow dilatation of the orifice from extreme pressure, from prolonged exertion, or it may be a result of endocarditis from rheumatism or chorea.

**Symptoms** are those due to pulmonary obstruction: dyspnœa in exertion being the most constant. Patients suffering from this form of heart disease may, if careful, enjoy fair health, but eventually dropsy, œdema of the lungs, obstruction of the liver follow, with cyanosis and general anasarca.

**Pathology.**—Imperfect closing of the mitral valve,



which may be demonstrated after death by the putting a ligature round the aorta, cutting off the left apex, and pouring a stream of water upon the ventricular surface of the mitral valve.

Dilated hypertrophy is the common result of this condition.

**Signs.**—Palpation detects irregularity of rhythm and impulse. Percussion shows extension of area in proportion to the hypertrophy.

Auscultation detects a systolic murmur at the apex. It is not extended upwards towards the base, but may be heard in the left axilla and at the angle of the left scapula.

With this sound is generally associated a marked intensity of the pulmonary second sound, heard over the second left interspace.

**Pulse** generally small, running, easily compressible, and irregular.

Mitral regurgitation is a frequent result of rheumatism, to which this form of heart disease is usually to be traced. It is difficult to give an opinion with regard to the relative duration of life in such disease, but patients may have a murmur indicating regurgitation, and show no general symptoms for years, and I believe with care, time, treatment, and rest, the murmur may diminish, and the heart recover.

I have had cases under my care in which the murmur was due to severe strain or prolonged exertion, and after treatment with iron, and rest, the heart has recovered and lost its murmur.

**Mitral Obstruction.**—This condition results in palpitation and dyspnoea, the symptoms from pulmonary congestion being marked, and œdema and dropsy being the ordinary sequelæ of this form of heart disease.

**Symptoms.**—The general appearance indicating mitral obstruction is peculiar: a flushed hectic colour of the cheeks, with or without cyanosis, and there is frequent œdema, or general dropsy.



The effect on the lungs is shown by congestion and pulmonary apoplexies.

**Pulse.**—Small and running, weakened and irregular, often intermittent.

**Pathology.**—This is the frequent result of rheumatism. It may also be caused by endocarditis after laceration, and it would seem to result from a congenital imperfection, the valve remaining of small size in spite of the general growth of the body.

The worst form of mitral stenosis, or obstruction, is that in which a button-hole opening, scarcely admitting the tip of the little finger, is found in a membranous diaphragm stretched across the valvular orifice. This is the condition which gives rise to the presystolic murmur, and is the most dangerous of mitral diseases, ranking next to aortic regurgitation in fatality. Another form is the funnel-shaped opening, often of small size.

Besides this, vegetations may occur on the auricular surface of valve, or on the free flap on the ventricle. From strain, one or two of the strings which hold the valve down, give way, and may be found loose from their attachment.

**Physical Signs.**—A murmur heard over the mitral area at the apex, just preceding the systolic sound. This is called the presystolic murmur. It follows the diastolic sound, and runs up to the systolic.

The diagnosis is to be made sometimes only by placing the finger on the carotid pulse.

It is often accompanied with a purring thrill or vibration, distinctly perceptible on placing the fingers over the apex: and a heavy impulse or thump of the apex.

This murmur is, however, very apt to disappear, and the student must not be deceived with regard to the presence of mitral disease because there is no murmur. In the worst conditions of the mitral, and when the heart is most embarrassed, the murmur may be absent; the apex beat is very irregular, often thumping and intermittent, the general condition, and especially the character of the pulse, indicating great danger.



It is general to find in mitral stenosis pulsation on the third interspace, due to the condition of the left auricle, and a systolic murmur is sometimes heard in this region.

**Myocarditis**, inflammation of the muscular tissue of the heart, a result of rheumatism as an extension of pericarditis, and of pyæmia.

**Symptoms.**—Dyspnœa, debility of heart action, vomiting, collapse, with delirium and coma. The average duration (according to Friedreich) is four days, so that the disease runs a very rapid course.

**Pathology.**—Muscular tissue of the heart is inflamed, swollen, and softened. It becomes grey and yellowish, breaking down under the formation of minute abscesses.

**Signs** are very indistinct: impulse of the heart weak, and a systolic murmur has been noted; the first sound of the heart being dull and feeble. Pulse frequent, weak, and irregular.

The diagnosis depends upon the presence of acute symptoms indicating sudden collapse of heart power.

**Morbus Cæruleus**, or the blue disease, is recognised on inspection by the deep mulberry tint of the surface of the patient. It is due to malformation of the heart or transposition of vessels, modifying the usual distribution of arterial and venous blood.

**Symptoms.**—Purple discoloration of the skin, more intense with palpation, dyspnœa, and, especially in cold weather, bulbous fingers. Cough and bronchial congestion are common.

**Pathology.**—Open foramen ovale; defective septum between the ventricles; pervious ductus arteriosus; irregular distribution of the aorta and pulmonary arteries; the aorta arising from the right ventricle, or from both ventricles.

**Signs.**—In those cases which have come under my observation I have detected a soft purring murmur below the base. Walshe states that systolic basic thrill and murmur are to be heard over the pulmonary area.

**Pericardial Diseases.**—**Pericarditis** is an inflammation



of the pericardial sac associated with acute rheumatism, pyæmia, and disease of the kidneys; or it may be a result of the presence of aneurism or tumour.

**Symptoms.**—Pain is a characteristic symptom (occurring in 70 per cent. of Sibson's cases), but it may be absent. Tenderness is present, and pressure in the epigastrium causes distress.

The usual position assumed by the patient, according to my experience, is the supine (on the back), the shoulders being low, but the head raised high, at an angle with the line of the body.

**Pathology.**—The ordinary and well-known signs of inflammation of a serous membrane are observed: injection of the vessels on the surface; fibrinous exudation followed by effusion of serous fluid into the sac.

**Signs.**—The preliminary sign of the onset of pericarditis is exhibited in a state of excitement in the heart's action, turbulent impulse, irregular rhythm, reduplication of sounds and rapid pulse, accompanied by pain and tenderness in some cases.

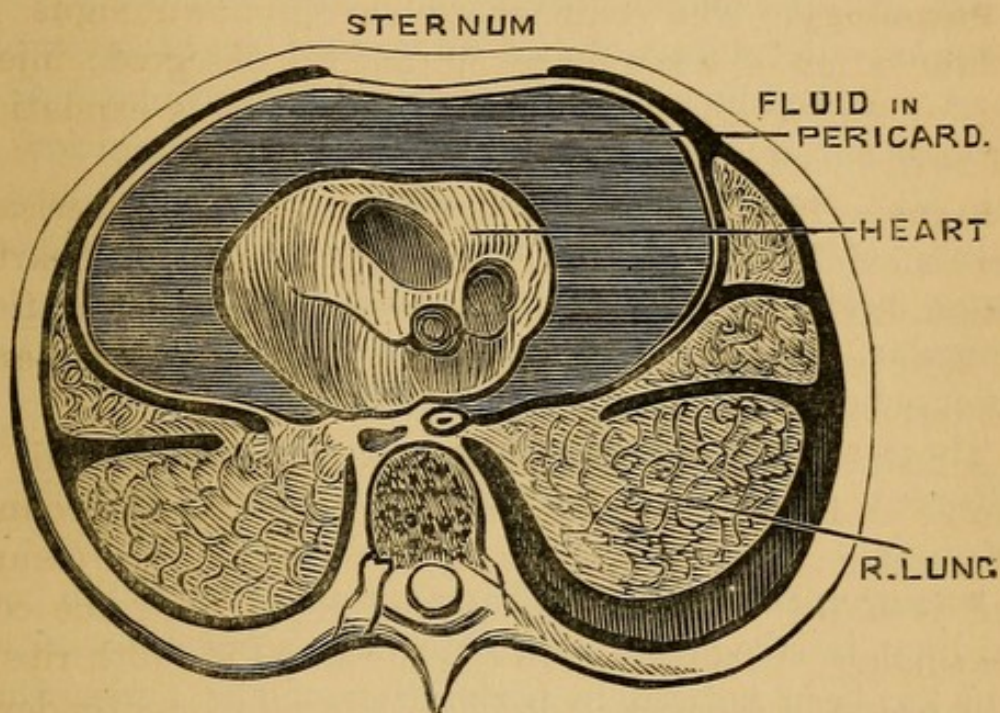
A friction murmur, which may be single and systolic or double, may be the prelude to the friction-rubbing sound. This sound is distinguished by being increased by pressure, and is heard in the lower sternal region and the corresponding left side between the third and the fifth ribs.

The true friction rub (to-and-fro) is subsequently developed, sometimes in a very short time, lasting occasionally for a short period, being then lost by effusion, although this is not the rule. This rubbing sound is superficial, harsh, and of a to-and-fro quality, with or without thrill. As a rule the friction sound is at first limited to the position and surface of the right ventricle; but as the disease progresses it extends to the left side, and the friction sound is heard over the apex. It may also extend to the large vessels, and be heard under the manubrium sterni, and over the right auricle. Thrill, when felt, is generally present in the second left interspace, or over the apex.



**Effusion into the pericardium** is detected chiefly by percussion. The fluid effused finds its level under the force of gravity, and falls to the lower portion of the sac; as it increases it depresses this portion, and forces the heart upwards, the sac being gradually enlarged on either side, before and behind, pushing the diaphragm and the abdominal organ downwards and the lungs on one side. The accompanying diagram, from one of Pirogoff's frozen sections, will show what takes place.

FIG. 25.



Transverse Section in a case of Pericardial Effusion. (Pirogoff.)

The effusion increasing, the lower part of the sac extends on either side, and the whole form of the pericardium is pear-shaped, the stalk end being above; and the area of dulness extends across the front part of the chest from the sixth right cartilage to the sixth left, and the fifth right interspace should be examined carefully and frequently where pericarditis is suspected. The heart is thrown to the top of the fluid, and is sometimes raised very high, the impulse being felt as high as the second interspace, the apex reaching the fourth rib.



The usual course is about eight or ten days from the beginning to the end of the effusion.

As the fluid is absorbed the heart regains its normal position, and the rub of pericarditis, usually on the return of the sound, grating and harsh, is again heard over the surface.

When the effusion is great the area of dulness may extend over the anterior surface of the chest, from the clavicle to the ninth right and left cartilages. (Sibson.)

**Pericardial adhesion**, to a limited extent, cannot be detected; but if the adhesions are extensive, as, for example, when they completely envelop the whole surface of the heart, the following signs may be looked for:—

**Signs.**—Retraction of the intercostal spaces over the heart, followed by a diastolic shock or back-stroke. Area dulness not diminished, and especially no alteration in this sign, nor in the impulse when the patient assumes a different position. The action and impulse are sometimes feeble, and generally tumultuous, hampered and irregular, and in some cases violent action has been observed over all the front part of the chest.

These are signs which have been noticed in cases of adherent pericardium, but the condition is difficult to diagnose.

**Pneumopericardium.**—The presence of air in the pericardium is recognised by a churning or gurgling splash, and has been noticed in perforation of the pericardium from artificial puncture (paracentesis pericardii), accidental puncture of the œsophagus from a juggler's knife, traumatic injury, fistulous opening from hepatic abscess, communication with a pulmonary cavity and cancerous ulceration.

The condition is extremely rare, but the signs are always described as peculiar and striking.

Tubercle and cancerous nodules may affect the pericardium secondary to a general infection, and give rise to rough friction sounds. Cancer, according to my experience, may give rise to extremely rough to-and-fro sounds.

**Pulmonary obstruction** gives rise to systolic



murmur, and this murmur is heard not infrequently from pressure or traction upon the artery; it is frequent with adherent pleura and cirrhosis of the lung or contracting phthisis, and is not uncommon in children from enlarged glands pressing upon the artery. It also occurs from congenital malformation.

**Sign.**—A systolic murmur over the second left interspace, transmitted upwards towards the middle of the left clavicle in the direction of the vessel.

**Pulmonary regurgitation** is a very rare disease. One case has been reported by Dr. Wilks; in this a double bruit was heard, more distinct on the left side; the pulse of aortic regurgitation being absent.

**Pyæmia** may cause pericarditis and endocarditis; and abscess of the septum has been found in rare cases.

**Rheumatism** is one of the most frequent causes of heart disease, endocardial and exocardial, in this country, but it is necessary to point out that rheumatism is absent in many cases, and inflammatory agglutinations of the valves may follow other diseases. From my own experience, I believe that diseases from injury are numerous, more so than is the general opinion.

Pericarditis occurs in about twenty per cent. of cases of acute rheumatism; coming on with the onset of the disease, and generally at the end of the first week; but it may be postponed until three weeks, or even more. Effusion progresses for three days after the mitral symptoms, and in eight days afterwards declines, giving altogether twelve days for the stage of effusion. These data are taken from Sibson's elaborate paper in "Reynold's System of Medicine." As regards the frequency with which the valves are attacked, the same observer gives the following data:—In 107 cases of endocarditis (out of a grand total of 325 cases of acute rheumatism) fifty-seven had mitral murmur, thirteen had tricuspid murmur, ten aortic, and nine mitral and aortic combined.

**Rupture of the heart** is a consequence of fatty disease, especially partial or localised, so as to produce



weakening of limited areas. It occurs in old age, generally above sixty; and the rupture has been induced by slight exertion, even by straining at stool.

**Symptoms.**—Intense and sudden pain in the præcordial region, with collapse and faintness, followed by very speedy death. Vomiting is an occasional symptom, and also diarrhœa. Patients have lived forty-eight hours after the rupture, and even as long as a week.

**Pathology.**—The pericardium is found full of clot, and a rent is seen on the surface of the heart, generally single, but sometimes more than one; most frequently on the anterior wall not far from the apex. The left ventricle is usually the part ruptured, but the right auricle and right ventricle may suffer.

**Signs.**—Death is so sudden that examination during life can seldom be made. Extended area of percussion dulness, and the absence of palpable impulse are the chief signs.

**Rupture of valves** is due to overstrain from excessive muscular action.

**Strain** upon the heart is induced in three ways. It may result from sudden momentary exertion, as the lifting of weights, jumping, or the like, in which the upper muscles of the thorax are called into play; or from prolonged continued action of the lower muscles of the trunk, as in walking and running; or from the combined action of all the trunk muscles, as in walking or running with heavy loads.

In the first case, the valves of the heart may give way and rupture from their attachment; the aortic valves being most frequently torn; the mitral valve suffering next in frequency, and the tricuspid the least often.

The symptoms are severe and sudden, and the patient is generally aware of something having given way; pain at the heart is generally severe, extending to the spine and shoulders; syncope is the chief symptom when the aortic valves are torn, suffocation and oppression, in rupture of the mitral valve; hæmoptysis may occur soon after the injury.



**Signs.**—A loud double murmur, with occasionally a flapping sound over the area of the valves affected.

Prolonged exertion of walking or lifting may lead to strain upon the cavities of the heart, producing dilatation of a very marked character of all or only of some.

Two fatal cases of the kind have come under my observation, the symptoms of which were very peculiar, the pain being referred to the lower part of the body, the patients being collapsed, cold, and with blue extremities. Blood-spitting was present to some extent.

In these cases the signs were due to dilatation, and soft double purring murmurs with thrills were heard over the heart's area, which were transmitted along the aorta, into the axilla, and to the right of the sternum. Cases of a similar kind have come under examination, occurring especially in hammermen or men engaged in foundry work; the right side of the heart in these cases being extremely prone to dilatation. With complete rest and tonic treatment the heart recovers and the murmurs go.

Aneurism is another condition which results from exertion, to be described in the next chapter.

**Tricuspid Murmurs** are those due to obstruction or regurgitation—the result of endocarditis, or more frequently of dilatation from pressure. The area of murmur is at the lower sternal region and towards the right.

**Tricuspid Regurgitant or Systolic** murmur is by no means rare, and is a sequela of pulmonary pressure, whether it be from disease of the lungs or from regurgitant disease of the mitral valve. Among diseases of the lung, emphysema, chronic bronchitis, cirrhosis, and fibroid disease of the lung may be considered as special causes of this murmur. Thrombosis and embolism also produce great strain upon the valve, with marked venous regurgitation.

**Tricuspid Obstruction** from stenosis of the valve is a rare disease. Dr. Hayden has recorded a case in which a presystolic murmur was heard in the fourth intercostal space close to the left side of the sternum.



Dilatation and obstruction of this valve produces great stasis of the venous system, throwing a large quantity of blood on to the abdominal organs, a condition likely to lead to dropsy.

**Venous Enlargement** must raise a suspicion of thoracic tumour, especially when local. In women the condition accompanies lactation.

**Venous hum** (*bruit de diable*) is a continuous murmur, not unlike the noise proceeding from a shell placed to the ear, heard generally in the jugulars under conditions of anæmia, but extending also to other veins, for example, the femoral. Their mode of formation has been explained already, and care must be taken not to produce murmurs artificially by the pressure of the stethoscope.

They vary in quality and intensity, according to the action of the heart.

**Venous Pulsation** indicates regurgitation through the tricuspid or pressure from tumour and aneurism. It occurs in most marked form when the obstruction to the pulmonary circulation is great, and is a characteristic sign of thrombosis and embolism.



## PHYSICAL SIGNS OF DISEASES OF THE AORTA.

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### CHAPTER XXII.

#### CLINICAL ANATOMY OF THE AORTA.

THE aorta, the great vessel of the systemic circulation, rises from the base of the heart in continuation with the conus arteriosus of the left ventricle, its orifice being guarded by three sigmoid valves. Opposite to each flap the root of the aorta bulges outward, the three dilatations being known as the sinuses of Valsalva. The pulmonary artery is at first in front of the aorta, but afterwards it passes to its left side. In its course upwards the aorta bends over to the left side, forming the arch of the aorta, which is divided into three portions,—the ascending, transverse, and descending parts of the arch.

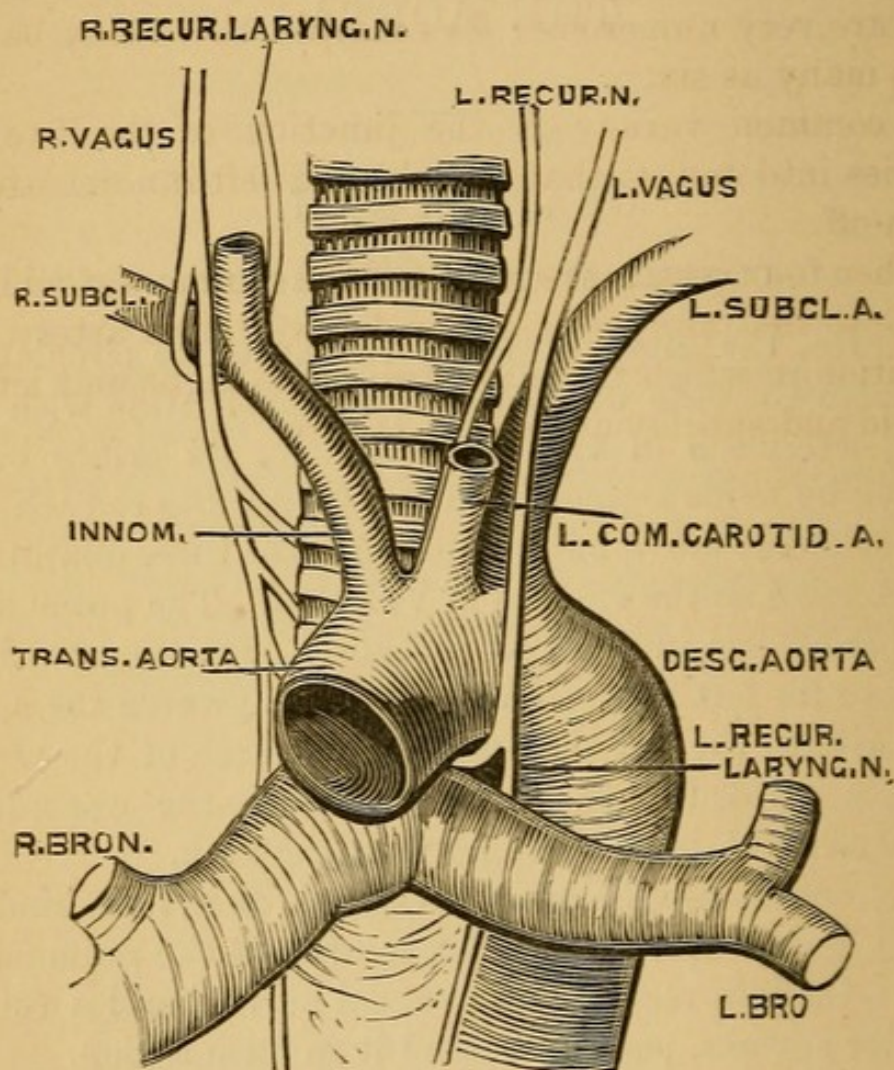
The trachea, œsophagus, and thoracic duct, lie behind it : while it arches over the left bronchus and the pulmonary artery : the left recurrent laryngeal nerve doubles round its under surface, passing behind it to form a loop.

The upper convex portion of the arch gives rise to the innominate, left carotid, and left subclavian arteries. It first touches the spine between the fourth and fifth dorsal vertebra : it then descends along the left side of the spinal column, passing through the opening of the diaphragm. The thoracic duct, the greater vena azygos, and the œsophagus (at first) are to the right side, but the œsophagus crosses over to the left in front, and is on the left side just above the diaphragm.



The topographical anatomy of the aorta has already been given at page 10 : but some account of the abnormal conditions which are frequently found affecting the position and distribution of the arch and the secondary arteries, is important with reference to the diagnosis of aneurism.

FIG. 26.



The Arch of the Aorta with Neighbouring Parts.

The arch may vary in size and position : it may reach as high as the top of the sternum, or may arch very low down, and be much reduced in size.

Lately, I examined a remarkable instance of the latter condition with diminished circulation of the left side. The patient had suffered from laryngitis and enlarged



gland: and the signs, consequently, closely simulated those of aneurism.

The aorta and the pulmonary artery may be transposed at their origin, so that the aorta circulates venous blood: an occasional cause of morbus cæruleus; or there may be intercommunication between the two, or both may be blended together in one stem.

The variations regarding the vessels springing from the arch are very numerous: for example, there may be one, or as many as six.

A common variety is the junction of the two left arteries into one, so that a right and left innominate are given off.

When four vessels are given off the commonest addition is due to the interposition of a left vertebral artery: the condition in which there are two pairs, right and left, of carotid and subclavian vessels, is rare.



## CHAPTER XXIII.

## DISEASES OF THE AORTA.

THE diseases to which the aorta is liable are those resulting from arteritis of an acute or chronic nature. These are, thickening of the walls, a condition to which the term *endo-arteritis deformans* has been given, and a degeneration commencing in the inner coat of the artery termed *atheroma*, leading to ossification. It is with the latter condition that we are at present concerned, as the cause of those saccular expansions communicating with an artery, which are called aneurisms. They may be considered under two forms : as dilatations of limited portions of the vessel, the fusiform or cylindrical aneurism, or as sacculated pouches, given off from one side of the vessel, to which the name saccular aneurism is given.

Mechanical strain is no doubt the chief cause of these sacs : women are very rarely the subjects of aneurism, men are very liable, especially those who are subjected to great exertion and labour in their trades and occupations : and aneurisms occur at the period of life at which men are most active in exertion, and in frames of body which are remarkable for a robust and muscular development ; and when a man in the prime of life, of vigorous strength and robust build, presents himself for examination with a history of cardiac and pulmonary symptoms, suspicion of aneurism should be at once present to the mind of the examiner.

**Symptoms.**—These are often very obscure, and depend chiefly upon the amount of pressure upon the adjacent organs and nerves.

The most frequent indications of pressure are derived



from the presence of dyspnoea, sometimes very severe, often spasmodic. Palpitation on exertion, inability to walk far without a feeling of being ready to drop, point to distress in the circulation.

Pain in the chest is felt over the aneurism, or in the neck and arms, in the sternum, and sometimes in the spine.

**Laryngeal Symptoms** are extremely important and distinct. A high-pitched raucous clanging cough and voice are characteristic of pressure, and often present in aneurism; but it must be pointed out that a very small tumour from the under-surface of the arch (where the recurrent nerve hooks round it) is sufficient to cause the symptom, and laryngeal stridor and spasmodic dyspnoea of a severe nature may be present without other signs, indicating aneurism. Gairdner relates an instructive case in which such a condition arose from a small aneurism of the innominate pressing on the right recurrent nerve. Complete aphonia may be present.

**Asthmatic attacks** of breathing may occur without laryngeal complications, from pressure on the left bronchus.

**Hæmoptysis** is not uncommon, although it generally occurs in specks and streaks, and may be overlooked. If in large quantities, the blood may come from direct communication of the sac with the air passages, and death may be preceded by copious gushes of blood from the mouth.

**Dysphagia** occurs from the pressure of the tumour on the œsophagus.

**Alteration** of the pupil, consisting in contraction of the pupil of the affected side, and immobility under altered conditions of light have been observed in many cases. Dilatation occurs in rare cases.

**Orthopnoea** is the position generally assumed by the patient, the exact attitude maintained being that which will enable the force of gravity to aid in the removal of pressure from the parts principally affected.



Gairdner has ably summarised the symptoms in aneurism, and the following is a *précis* of his remarks:—

1. When angina pectoris is present, the aneurism is probably situated in the ascending portion, near the cardiac plexus.

2. When laryngeal symptoms are present, the right or left recurrent nerve is involved from a sac in the innominate, or on the posterior and inferior part of the arch.

3. Bronchial asthma and orthopnoea indicate pressure of the pulmonary plexus of nerves from the descending part of the arch.

4. Alterations of the pupil are due to pressure upon the sympathetic from the upper and back part of the arch, or its branches.

5. Dysphagia is a result of pressure on the œsophagus or the pneumogastric nerve.

**Signs.**—Inspection detects local bulging and protrusion of the manubrium, the sternal fossa being deep and well marked.

The positions of bulging are the right infraclavicular region close to the sternum, the left infraclavicular and mammary regions, the upper sternal and the infraclavicular region, on both sides; sometimes the bulging (when the sac has made its way through the chest wall) is enormous.

**Palpation** detects pulsation, a very important sign of vascular tumour; a little behind the apex beat, the impulse of which is occasionally intense, with a heaving or pushing character, and a hard solid feel if there be much coagulum in the sac.

**Thrill** may be transmitted to the touch, due to the rippling current of blood in the sac. It is more common in fusiform dilatations than in sacculated aneurisms, sometimes it is only found in the most prominent part of the tumour, and it may be present at one time and disappear at another.

**Percussion** gives the earliest indication of arterial enlargement, and the region in which this is detected



depends upon the locality of the aneurism, and the part should be carefully examined by cross percussion, that is, by placing the finger of the left hand parallel to the sternum, and percussing from the right nipple-line to the left side; a circumscribed patch of percussion will be found at the second right interspace if the aneurism is situated in the ascending arch, or in the first if it should be in the innominate artery.

If the aneurism is situated under the sternum, the percussion note is more difficult to distinguish.

If it is in the descending part of the arch the left interscapular region should be carefully searched, and especially the part opposite the fourth dorsal vertebra.

Sometimes the dulness is found lower down, between the eighth and tenth dorsal vertebræ.

Percussion occasionally fails to establish the presence of aneurism.

**Auscultation.**—When the ear is applied over the seat of aneurism the result may be to show—

1. That there are no sounds at all.

The sac may be silent, a condition which is by no means uncommon, especially in sacculated aneurisms in the form of blind pouches, given off from different parts of the arch.

2. That there is a double sound,

a double murmur,

a systolic murmur with diastolic sound,

a diastolic murmur with systolic sound.

These sounds are due to aneurism uncomplicated with disease of the aortic valve.

As far as I know there is no practical point to be obtained from these different sounds, which vary from posture and other considerations.

As a rule I would say that noisy double sounds with free expansile pulsation are due to dilatation without coagulum.

Pulsations with thudding impulse and sounds only, or no sounds at all, are due to sacculated aneurisms filled with coagulum.



The exact location of an aneurism must be made by a careful consideration of the parts affected, by the position of the aneurism, and the careful investigation of the several pulses.

The secondary condition of the heart, resulting from aneurism, forms an important point in the group of physical signs. In early conditions, when pressure upon the aortic valves is marked, the second sound over the valves may be very accentuated, sharp and clacking; but this is not an invariable rule, and in some advanced cases the heart sounds are very indistinct and deadened.

The impulse of the heart must be accurately noted and the position of the apex ascertained. It is often displaced from hypertrophy, and sometimes in an extraordinary manner from pressure of the tumour encroaching upon and pushing down the base of the heart.

**Pulse** may be diminished or obliterated in a special vessel, or its beat postponed. Careful search should be made for inequality, by testing the corresponding arteries on each side with the hands. Diminution in the pulse of one side indicates pressure or weakening of the current from dilatation. The student must be cautioned that congenital peculiarities sometimes traverse this sign.

**Pulmonary signs** must be looked for especially over the large bronchi in the interscapular regions. Signs of pressure are often present, and are either stridulous breathing and bronchial respiration, or abolition of the respiratory murmur except at the base of the lung.

The frequency in which aneurisms occur may be given in the following order, derived from Sibson's figures:—

- Ascending.
- Transverse.
- Ascending and transverse.
- Sinuses of Valsalva.
- Descending.



## CHAPTER XXIV.

## HISTORICAL SUMMARY.

I CANNOT conclude these pages without giving a short sketch of the history of physical signs; how far they date back, and to whom they are due: one's memory should always have a place for those observers who have established one fact which enables us to detect the presence of disease.

The earliest sign for the detection of thoracic disease, known centuries ago to Hippocrates, was probably the splash of succussion; and the use of percussion for the diagnosis of fluid in the belly was known in his time.

It was not until rather more than a century ago, that the art of percussion was amplified and practised by Auenbrugger, who published his results in 1761.

It is due, however, to Corvisart that the discoveries of Auenbrugger, who had met with most undeserved neglect by his contemporaries, were brought to light, and the art was practised especially with regard to heart diseases. Corvisart, as Laennec, his pupil, distinctly states, never applied his ear immediately to the chest, although he was aware that the beating of the heart could be heard by holding the ear near the chest. To continue Laennec's account, Bayle was the first to listen directly to the chest.

It was not until 1816, that mediate auscultation was discovered by Laennec.

He was consulted by a young woman apparently suffering from disease of the heart. Her age and sex rendered immediate auscultation inadvisable. Laennec, remembering that sounds could be heard transmitted through wood,



conceived the idea of making use of this property of solids in the case. He took a roll of paper and applied one end to the præcordial region, on the other end he placed his ear, and he was not more surprised than gratified to hear the beating of the heart more clearly and distinctly than by immediate auscultation.

For the next three years he employed himself in elaborating this discovery by observation at the Hôpital Necker, and in 1819 he published his work on mediate auscultation.

To him we owe most of our knowledge of physical signs, and his observations have well stood the test of time.

Since his time additional observations have been made.

Piorry, in 1826, who elaborated the doctrine of percussion, supposed that each organ and substance had a special sound. He invented the pleximeter, and modified the form of the stethoscope.

In 1839, Skoda published his work on percussion and auscultation, and established a scientific basis for the interpretation of percussion notes.

To him is particularly due the discovery of the tympanitic note, which is often found above the fluid of an effusion, and hence is sometimes called Skoda's tympanitic note: his interpretation of the sound is open to challenge.

Among English observers and writers on this subject, Williams comes first, publishing his work on diseases of the lungs in 1828. After him came Walshe, whose book is a perfect library of facts, noted with the greatest accuracy, supported by pathological evidence, and detected with a nice musical ear.

Flint, of New York, devoted much attention to the conditions of sound involving an alteration of pitch, and to him are due the distinctive characters of solid and hollow sounds, which are clinically of the greatest value and importance.

To France must be attributed, not only the name given to the presystolic murmur by Gendrin in 1841, but the condition which it indicates, as first described by Fauvel.



In recent times, much has been done for the advancement of auscultation, especially with regard to the distinctive signs of cardiac disease, by Peacock in England, Stokes in Ireland, and Gairdner and Balfour in Scotland.

There are still obscure points to clear up, and fresh observations required with regard to the interpretation of sounds, which may exercise the attention and thought of all.

It would be difficult to find a more apposite conclusion to this brief account of the revelations of the stethoscope than that prophetic view of the employment of sound for the indication of the working of the animal frame, which is contained in the following quaint but striking words (which have been often quoted before) from the writings of Hooke, in the beginning of the last century.

“Who knows but that it may be possible to discover the motions of the internal parts of bodies by the sound they make; that one may discover the works performed in the several offices and shops of a man’s body, and thereby discover what instrument or engine is out of order, what works are going on at several times, and lie still at others, and the like. I could proceed further, but methinks I can hardly forbear to blush when I consider how the most part of men will look upon this, but yet again I have this encouragement not to think all these things utterly impossible, though never so much derided by the generality of men, and never so seemingly mad, foolish, and fantastic, that as the thinking them impossible cannot much improve my knowledge, so the believing them possible may perhaps be an occasion of taking notice of such things as another would pass by without regard as useless.”



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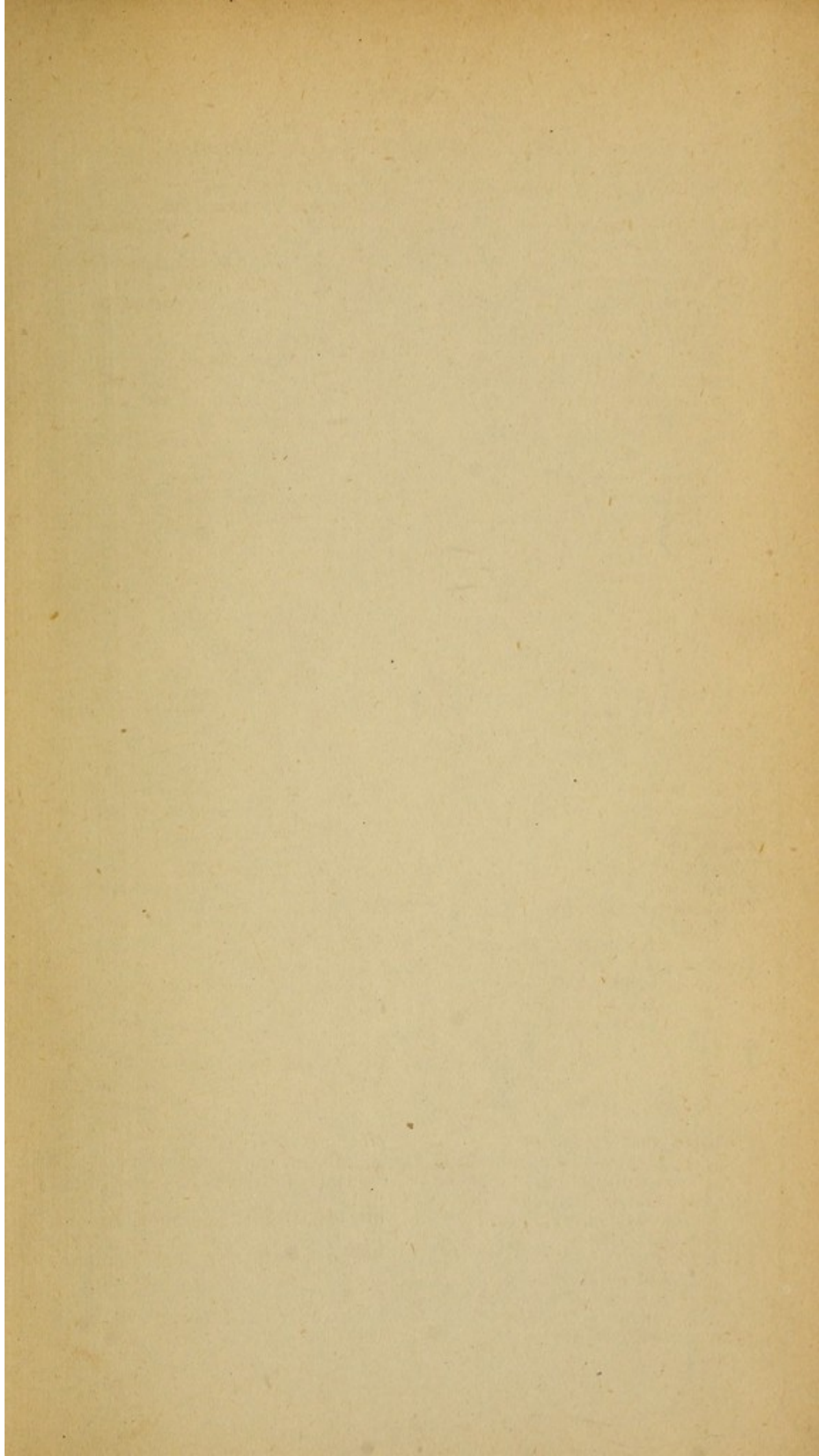


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