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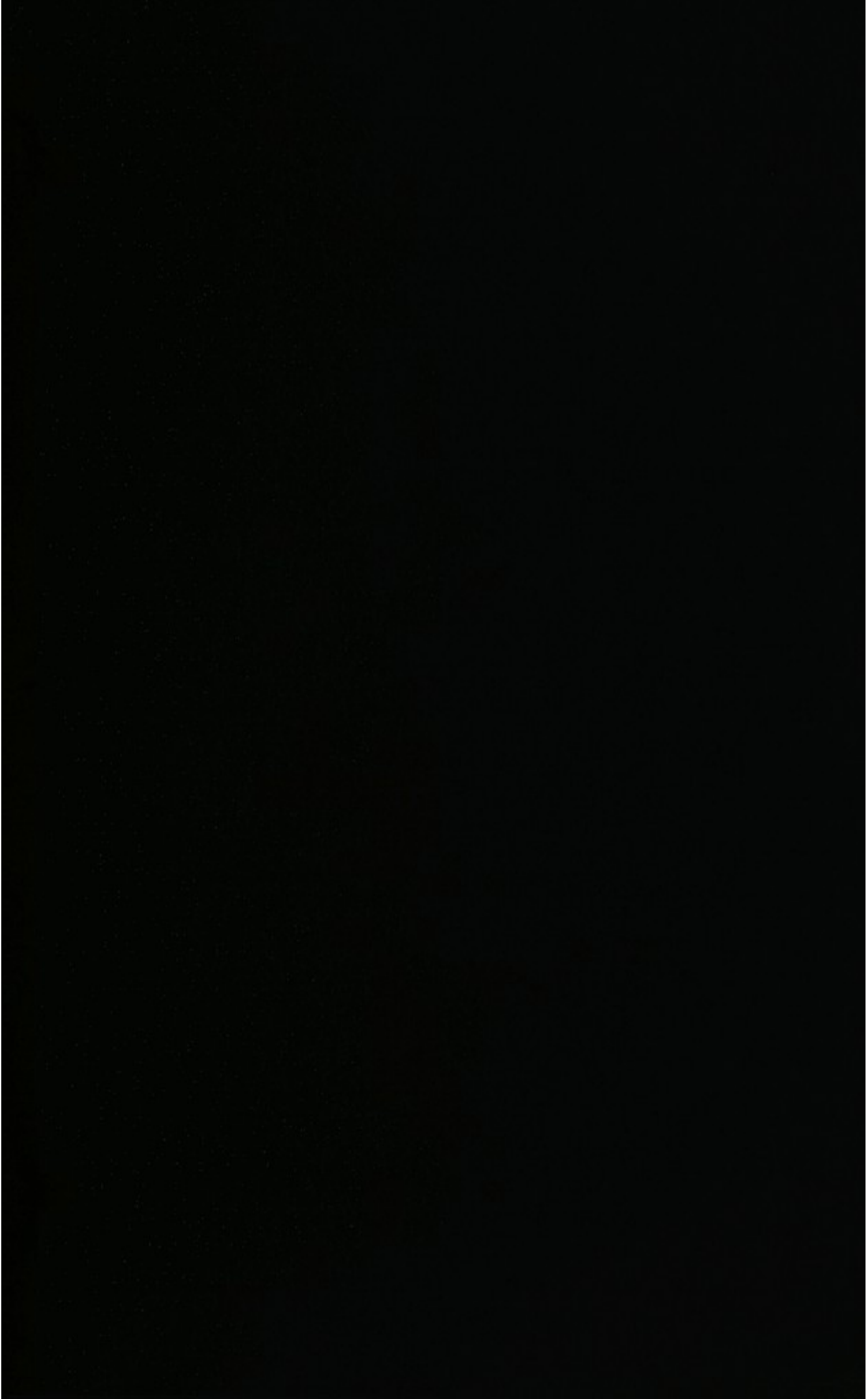
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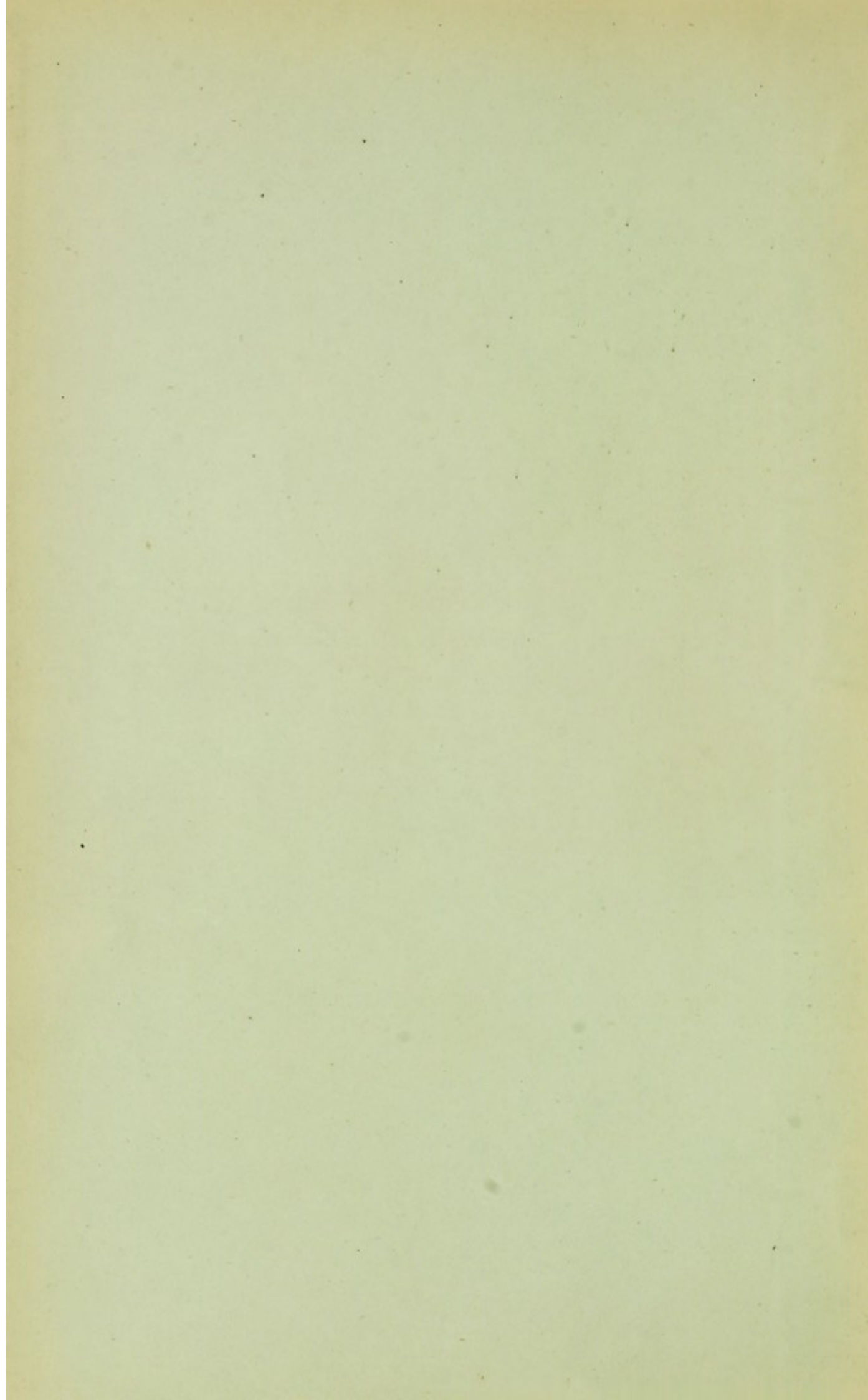


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CLINICAL LECTURES AND ESSAYS
ON
RICKETS, TUBERCULOSIS
ABDOMINAL TUMOURS
AND OTHER SUBJECTS

BY
SIR WILLIAM JENNER, BART., G.C.B.

M.D. LOND. AND F.R.C.P., D.C.L. OXON., LL.D. CANTAB. AND EDIN.
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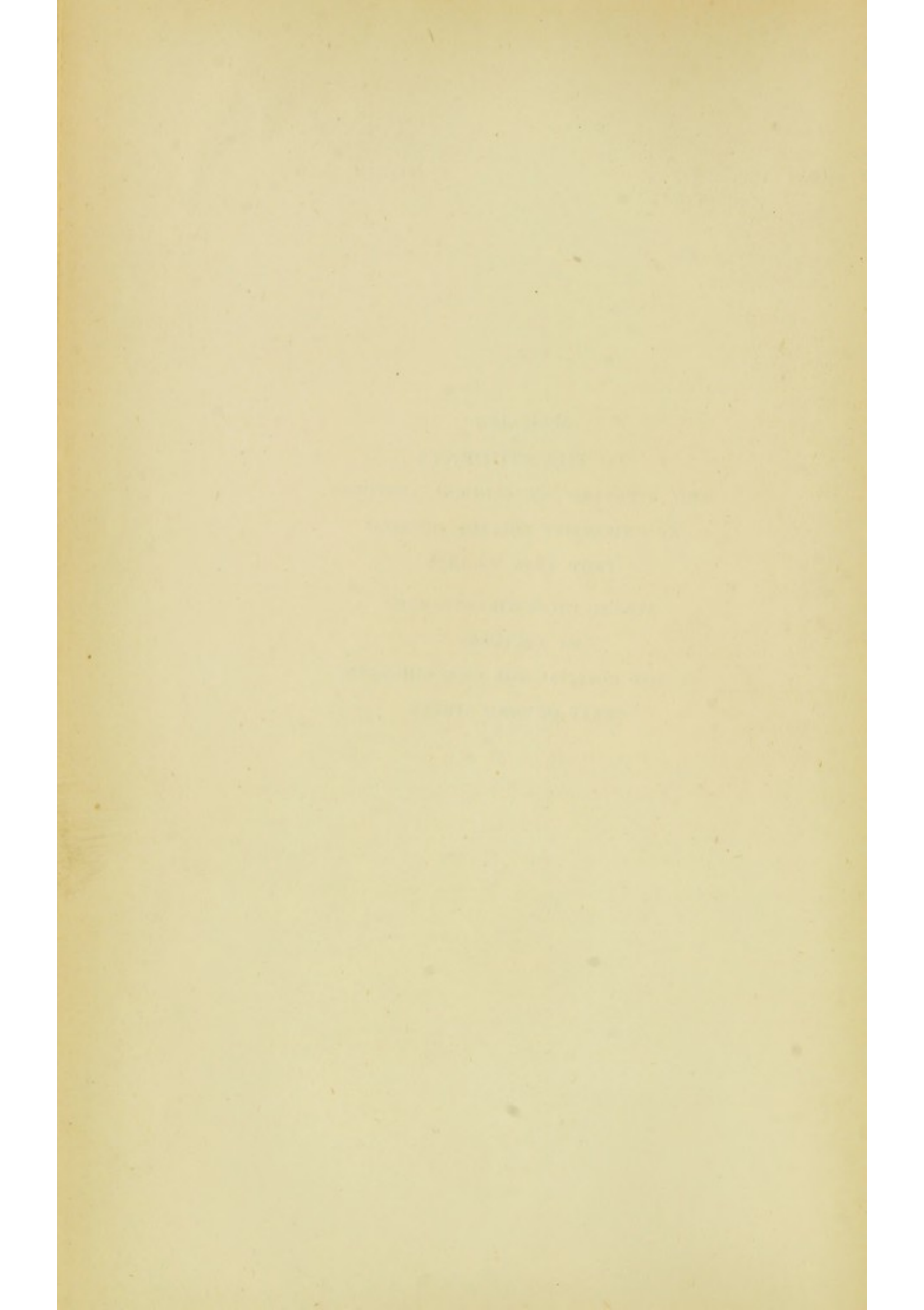
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1918.

DEDICATED
TO THE STUDENTS
WHO ATTENDED MY CLINICAL LECTURES
AT UNIVERSITY COLLEGE HOSPITAL
FROM 1850 TO 1878
ALSO TO THOSE WHO ATTENDED
MY LECTURES
AT THE HOSPITAL FOR SICK CHILDREN,
GREAT ORMOND STREET



P R E F A C E

IN the present volume I have reprinted my lectures on Rickets and other lectures and papers. I have followed the same rule as in the previous volume on Fevers, viz., to leave the statements made exactly as they were originally given, without attempting to embody the results of more recent investigations. For example, I have not altered the paragraphs relating to the difference between Scrofula and Tubercle. For although recent histological researches have shown the identity of their ultimate pathological products, yet I still hold that the constitutional states which precede the local manifestations are of a different type in these two disorders, and deserve separate consideration. I still adhere to the distinctions which are set forth in the lectures.

The lectures on abdominal tumours were reported for me by my able friend and former pupil, Dr. W. R. Gowers.

I thank my friends Dr. T. Barlow and Dr. Sidney Coupland for all they have done for me.

WILLIAM JENNER.

GREENWOOD, BISHOPS WALTHAM,

Sept. 1894.

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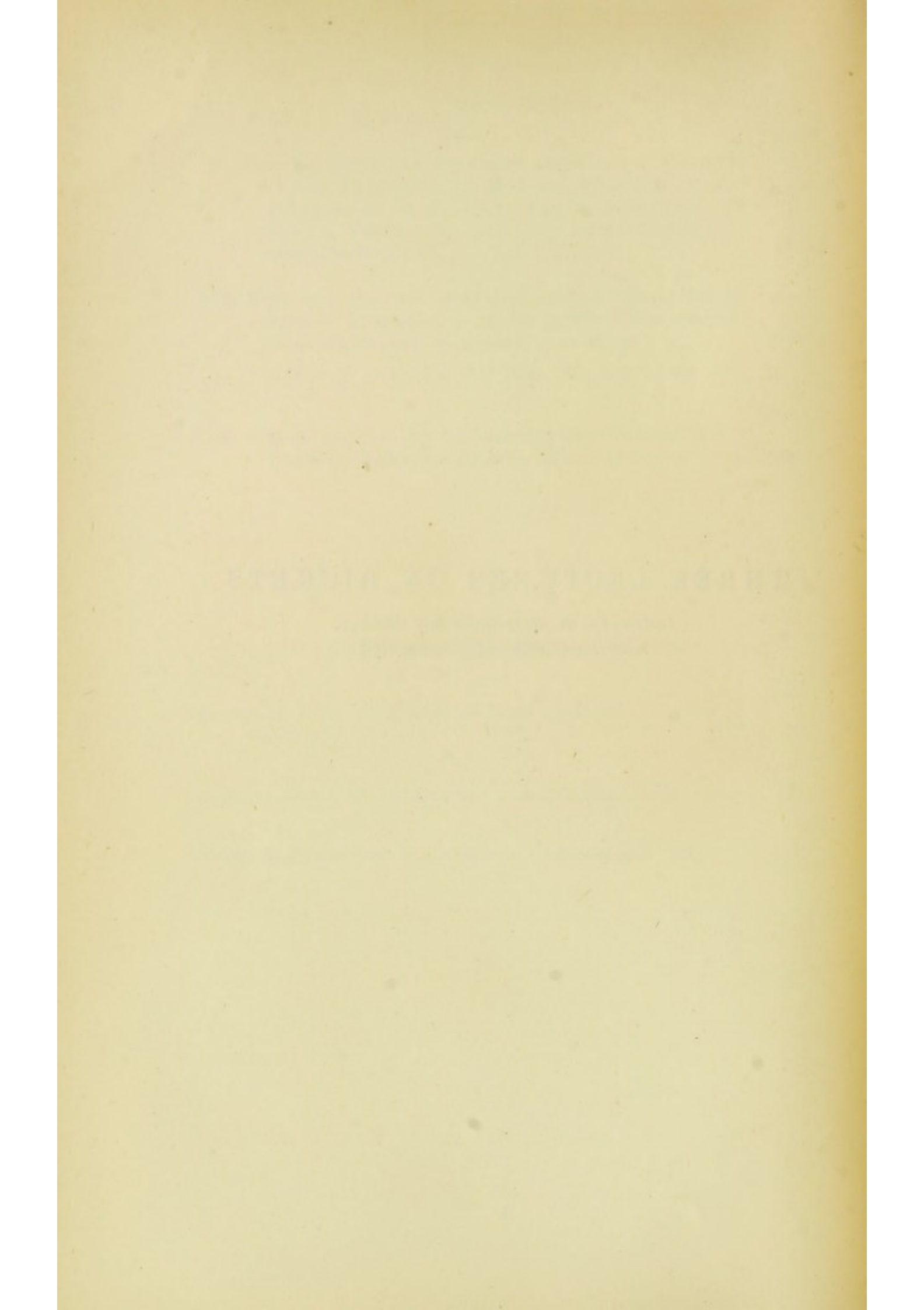
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THREE LECTURES ON RICKETS

*Delivered at the Hospital for Sick Children,
in December 1859 and January 1860*



ON RICKETS.¹

LECTURE I

Three Striking peculiarities of Infant Life, viz., the Sensitiveness of the Nervous System; the Unity of Organic Disease; the Frequency of Diathetic Disease—The four great Diathetic Diseases of early Childhood, viz., Rickets, Tuberculosis, Scrofulosis, Syphilis—The Grounds for their Separation—Rickets the most common and the most fatal of the Diseases which exclusively or chiefly affect children—Rickets a General Disease—Enumeration of the most common and striking Anatomical Lesions in Rickets—The Anatomical Lesions of the Bones and the Deformities that result considered at length—1. Enlargement of the Ends of the Long Bones: Its Anatomical Causes—2. Softening of the Bones—Chemical Constitution of the Softened Bones—3. Thickening of the Flat Bones: Its Anatomical Cause—4. Deformities following on Softening of the Bones—Curvature of Spine—Differences before and after Walking—Liable to be confounded with Angular Curvature—Deformities of Femur, Tibia, Ulna, and Radius, Humerus, and Clavicles, the Result of Pressure and not of Muscular Action—Deformity of the Thorax, the Result of Atmospheric Pressure—Mechanism of its Production.

GENTLEMEN,—If you have ever conversed with several persons who have visited together a spot celebrated for its beauty, you must have been astonished at the different impression each conveys to you of the most striking character of the scene. One dwells on the general beauty of the landscape, one on the extent of the prospect, one on the height of the mountains, one on the richness of the valleys. Those only agree in their description who have seen everything through the guide-

¹ *Medical Times and Gazette*—March to May 1860.

book. So is it with those who look at disease. Each may see the whole of what is before him, but the impression made by individual parts of that whole on each who looks for himself is different. To one, this peculiarity, to another, that, gives its feature to the disease. Nearly 60,000 sick children have been patients at this Hospital since its opening. A fair proportion of these have come under my observation; and I have endeavoured to look at them with my own eyes. Some of the general impressions I have received I shall, I hope, convey to you; and some of the facts that I have seen I shall describe to you. Much of what I have to say may be found here or there on record; but, having looked for myself, I shall describe only from my own observations; and trust that what is thus lost in general completeness will be gained in freshness, and in fulness of details in reference to special parts of the subject; those points in the view which have made most impression on my mind will doubtless stand out most prominently before you. Before occupying my present office I had read many excellent guide-books; I had been connected for some time with a large hospital; I had seen among my out-patients, there and elsewhere, many sick children; I thought I knew most of the paths and passes of that great section of pathology, diseases of children; and, had I been required, I should probably have undertaken to guide others through them. But a very brief experience taught me that I was mistaken—that I had much to learn before I ought to offer to lead others.

The first thing, then, I may say, which impressed me when I came to see diseases of children on a large scale, was the smallness of my knowledge in regard to them. I tell you this because I am anxious to impress on you my conviction that diseases of children do require special study, and time, and attention, to master them. At the same time I readily admit that the diseases of children are more easy to diagnose, and more easy to treat successfully than are the diseases of adults. Coming as I did to the study of the diseases of children after I had been engaged in teaching the diseases of adults, my mind was strongly impressed by three of the peculiarities of infant life—

1. By the comparative sensitiveness in the child of the

nervous system generally to impressions, but more especially of those parts of the nervous system which are concerned in the production of reflex movements, and in the development of the so-called sympathetic derangements. In reference to these points the child seems at one end of the scale, the aged at the other.

A striking illustration of the sensitiveness of the child to reflex stimulation is afforded by the following experiment:—

Pass the finger gently over the inner aspect of the upper two-thirds of the thigh of a young boy, and notice at the same time the movement of the testicle. You will see that it is instantly drawn up close to the external abdominal ring by the cremaster, and that with a rapidity which will surprise those who witness it for the first time. The scrotum remains flaccid; the testicle slowly descends. Repeat the stimulation on the opposite side, and the testicle on that side is now drawn up. Pass the finger over the skin of the outer part of the thigh, and the testicle is motionless. Touch the skin above the right pubis, and the right cremaster draws up the right testicle. Touch the skin above the left pubis, and the left cremaster is thrown into action. Stimulate the skin higher on the abdomen, and no movement of the testicle follows. Repeat the experiment many times, or use powerful stimulation, and the contraction of the cremaster grows gradually weaker, till at last you fail in your effort to excite it to action. Rest restores the excitability of the part. You will remember that branches of the ilio-inguinal and genito-crural nerves supply the skin at the situations I have mentioned; and you may, I believe, in every case map out by the experiment I have referred to the exact portions of skin supplied by those nerves.

With reference to the derangements commonly called sympathetic. The old man may, nay often does, die from acute inflammation of the lungs with mind entire, a pulse scarcely more frequent than in health, respiratory movements only a little quicker than natural, and a skin of normal temperature. The child eats some indigestible food, and forthwith its pulse is rapid, its respirations are doubled in frequency, its skin is burning hot, its mind rambles, it is convulsed in every limb.

2. By the frequency with which, at examinations after death, structural disease of one organ only is found. Now, the pathological anatomist who examines all the organs and structures of the subject, and not merely that which during life was supposed to be mortally affected, cannot fail to have noted that death from uncomplicated acute disease is of comparatively rare occurrence in the adult. This seems to be the consequence partly of degenerations commencing in some structures long before what we call old age, and partly of organs or tissues once structurally diseased, rarely if ever recovering their absolutely healthy condition.

3. By the far larger proportion of children than of adults that are the subjects of profound diathetic diseases before they are the subjects of local lesions of structure; and when the subjects of diathetic disease, by the vast number of organs found after death to be seats of structural change.

The great diathetic diseases of childhood are four, viz., rickets, tuberculosis, scrofulosis, and syphilis. All manifest themselves primarily by deviations from the standard of health, which deviations, *per se*, we do not call disease. For example, the delicate skin in tuberculosis, the thick complexion in scrofulosis, the muddy tint of the skin in syphilis, and the low muscular power in rickets.

All are distinguished by their more decidedly pathological tendencies, that is, by the frequency with which they produce or are accompanied by striking and peculiar deviations from the healthy structure of particular organs and tissues; *e.g.* by softening of the bones in rickets, by the deposit or formation of tubercle in tuberculosis, by a peculiar kind of ophthalmia in scrofulosis, by characteristic diseases of the skin and mucous membrane in syphilis.

I shall enumerate briefly the leading features of a typical case of each of these four general conditions.

TUBERCULOSIS.¹

Nervous system highly developed; mind and body active; figure slim; adipose tissue small in quantity; organisation

¹ I use this term to signify the condition of the system which precedes and accompanies the deposit or formation of tubercle, and which may or may not be accompanied by the deposit or formation of tubercle.

generally delicate; skin thin; complexion clear; superficial veins distinct; blush ready; eyes bright; pupils long; eye-lashes long; hair silken; face oval, good-looking; ends of long bones small, shafts thin and rigid; limbs straight. Children the subjects of tuberculosis usually cut their teeth, run alone, and talk early.

Leading Pathological Tendencies.—Fatty degeneration of liver and kidneys; deposits or formations of tubercle,¹ and their consequences; inflammation of the serous membranes.

SCROFULOSIS.

Temperament phlegmatic; mind and body lethargic; figure heavy; skin thick and opaque; complexion dull, pasty-looking; upper lip and alæ of nose thick; nostrils expanded; face plain; lymphatic glands perceptible to touch; abdomen full; ends of the long bones rather large; shafts thick.

Leading Pathological Tendencies.—Inflammation of the mucous membranes of a peculiar kind; so-called strumous ophthalmia; inflammation of the tarsi; catarrhal inflammations of the mucous membrane of the nose, pharynx, bronchi, stomach, and intestines; inflammation and suppuration of the lymphatic glands on trifling irritation; obstinate diseases of the skin; caries of bone.

RICKETS.

Mental capacity and power small; muscular force deficient; mind and body inactive; figure short; closure of the fontanelles retarded; face small, but broad; skin opaque, often set with downy hairs. Children the subjects of rickets are late in cutting their teeth, in running alone, and in talking, and their teeth drop early from their sockets.

Leading Pathological Tendencies.—Softening of the bones; enlargement of the ends of the long bones; thickening of the flat bones, and deformities consequent on these conditions of the bones; so-called hypertrophy of the white matter of the brain; chronic hydrocephalus; pulmonary

¹ I use both terms, because I am unwilling here to express an opinion on the question whether tubercle be a deposit, as is generally believed, or a formation, as some pathologists are now disposed to regard it.

collapse; laryngismus stridulus; convulsions; albuminoid infiltrations of the liver, spleen, lymphatic glands, etc.

SYPHILIS.

Adipose tissue small in quantity; muscles flabby; cutis rough—deficient in contractility; complexion muddy.

Leading Pathological Tendencies.—Suppurative inflammation of the mucous membranes of the nose; ulceration of the mucous membranes of the nose and of the lips, mouth, throat, and anus; falling of the hair; eruptions on the skin of peculiar character; induration of the liver; suppuration of the thymus, lungs, etc.

There are pathologists of high repute who regard rickets, scrofulosis, and tuberculosis to be mere modifications of the same disease. I and others hold them to be distinct affections.¹ The whole difficulty of the question lies in the difficulty of determining what is necessary to constitute identical diseases.² With reference to some diseases the grounds of separation are broad and unequivocal. Scarlet fever and measles are distinct diseases, because their specific cause is different. Syphilis and tuberculosis are undoubtedly not identical, because syphilis owes its origin to a specific cause, and tuberculosis does not. Rickets, tuberculosis, and scrofulosis are due, it is said, to mal-nutrition, and therefore it is urged they are essentially identical; but it is manifest that the term mal-nutrition is at once very vague and very comprehensive. All diseased action by which unhealthy structure is formed in the place of healthy may be called mal-nutrition. Pus is the result of mal-nutrition, cancer is the

¹ It has even been suggested that rickets is a variety of congenital syphilis, that it is not so seems to be proved by a consideration of the following facts:—The parent who infects his offspring has usually contracted syphilis before marriage, and the children first begotten after infection are those who suffer; while, as a rule, it is only the younger children of a family that suffer from rickets. The first-born of their parents being commonly healthy, though the later born are highly rickety.

² Many of the disputes which have arisen on the subject of identity of diseases have been the consequence of a want of definition of terms. Two diseases may agree in very many essential points, and yet not be identical, *e.g.* measles and scarlet fever. A and B are both letters of the alphabet, but A and B have very different significations. 1 and 2 are both numerals, but 1 and 2 are anything but identical.

result of mal-nutrition, tubercle is the result of mal-nutrition,—and yet the purulent, cancerous, and tuberculous diatheses cannot be held to be identical.

I hold rickets, tuberculosis, and scrofulosis to be distinct diseases in the sense in which tuberculosis and cancer are distinct diseases, and for the following reasons:—

1. Because the general condition in each is perfectly different from that in the other.

2. Because the pathological tendencies of those who manifest these different general conditions are different.

3.¹ Because we so rarely see the pathological tendencies of the one manifested by those, the subject of the others, *e.g.* rickets is absolutely unfavourable to tuberculisation²—tuberculisation to strumous ophthalmia.

4. Because, tuberculosis being unquestionably hereditary, we do not find the children of phthisical parents specially prone to rickets or to scrofulosis.

5. Because, although we often find several members of the same family the subjects of rickets, of tuberculosis, or of scrofulosis, it is comparatively rare for members of the same family to be the subjects of more than one of these diatheses.

6. Because rickets is not hereditary in the sense in which tuberculosis is hereditary.

7. Because the course, prognosis, and treatment of each of these diatheses are different.

Among the children of the poor in London, the most widely-spread of these diatheses is rickets. It is, however, by no means limited to the poor, or to London, or even to large towns. I have very often seen it in the children of the wealthy, and even in the children of the wealthy living in the country. It was a consideration of the enormous number of those that suffer from rickets, the very large number that die annually from its effects, its remarkable anatomical characters, its limitation to children, its permanent effects,

¹ I propose to analyse the facts on which 3, 4, 5, and 6 are founded in a separate paper. It is sufficient now to say the facts I have collected point to the conclusion that while more than 40 per cent. of tubercular children are born of phthisical parents, about 9 per cent. only of rickety children come of phthisical parents.

² Rickets does not by any means exclude tubercle. Rickety children may be tubercular, just as syphilitic children may be.

the very small space which it occupies in English books devoted to the diseases of children, and the contradictory statements on several most important points made by the best pathologists who have bestowed attention on the disease, that determined me to examine minutely the cases of rickets which came under my care in this Hospital.

I shall in the remainder of this, and in my two succeeding lectures, detail to you what I know, from my own observation of that which seems to me to be without question the most common, the most important, and in its effects the most fatal of the diseases which exclusively affect children.

Rickets is a general, or diathetic disease, manifested after it has existed a longer or shorter time, by certain lesions of the structure of the bones,—and, I say, manifested after a time, because in some cases the general disorder unequivocally precedes the local changes, and before these latter occur, we can predicate that they will occur.

In some books, rickets is classed among diseases of the bones. This is a mistake; rickets is no more a disease of the bones, than is typhoid fever a disease of the intestines. Rickets leads to disease of the bones in the same way that typhoid fever leads to disease of Peyer's patches; but there is a general disease preceding and accompanying the disease of the bones in the one case, as there is preceding and accompanying the disease of the intestines in the other. The change in the bones is the anatomical character of rickets.

The most constant and striking anatomical lesions in rickets are:—

1. Enlargement of the ends of the long bones,—of the parts where the bone and cartilage are in contact, *i.e.* where the cartilage is preparing for ossification, and where ossification is advancing in the cartilage.
2. Softening of all the bones.
3. Thickening of the flat bones, *e.g.* the bones of the skull—the scapula.
4. Deformities which follow from mechanical causes acting on the softened bones, *e.g.* the deformities of the thorax, pelvis, spine, long bones.
5. Arrest of growth, not only of the bones, but of all the

parts directly related anatomically and physiologically to the bones, *i.e.* of the muscles, vessels, nerves, and teeth.

6. Certain lesions of the pericardium, lungs, and capsule of the spleen, the direct consequence of the thoracic deformity.

7. Less constant, but highly important changes, most commonly affecting the nutrition of the brain, spleen, liver, lymphatic glands, and muscles, and now and then of every organ.

I shall now consider the anatomical lesions of the bones, and the deformities that are their consequences, at greater length.

1. *Enlargement of the Growing Ends of the Long Bones.*—When we look at a child suffering from rickets, we are at once struck by the large size of the wrists. It has been supposed that the enlargement is apparent, not real; that the wrist looks large because the arm has wasted; this, however, is incorrect. I measured the circumference of several wrists in the rickety and the non-rickety, and found that whether reference was had to the age or the height of the child, or to the length of the forearm, the circumference of the wrist was greater in the rickety than in the non-rickety. I recently measured the height, the length of the forearm, and the circumference of the wrist of three children, two of whom are now in the hospital, and obtained these results:—

Disease.	Age.	Height.	Length of Forearm.	Circumference of Wrist.
Rickets, . . .	4 yrs. 0 m.	30 in.	4½ in.	4¾ in.
Rickets, . . .	3 „ 2 „	30 „	4½ „	4½ „
Tuberculosis, . .	3 „ 0 „	35 „	5 „	3¾ „

We find similar enlargement of the costal end of the ribs, of the ankles, of the olecranon process of the ulna; in fact, of the extremities of all the long bones. Some pathologists have stated that the ends of bones which are the least covered by soft parts, are the most affected; my experience is opposed to this assertion. The head of the thigh-bone and of the humerus suffer as much as the more exposed extremities of the long bones. As to the intimate anatomical structure of the large ends of the bones: there is excessive formation of the structures which precede or form the nidus for ossifica-

tion, there is retardation and incomplete performance of the process of ossification.¹ In the healthy child the ends of the long bones measure more in circumference than the shafts, as the process of ossification is completed, the bone diminishes in girth.

In rickets there is an exaggeration of the condition we find in the first stages of ossification in the healthy subject, the completion of the process only is stayed. There is great development of the spongy tissue of the head of the bone, and of the epiphyses, and also of that layer of cartilage in which the primary deposit of calcareous matter takes place.

The layer of cartilage in which the cells are arranged in linear series, instead of being half-a-line, is from a quarter to half-an-inch in breadth.

Again, the calcareous granular deposit is wanting at the boundary of ossification, and there the cartilage-cells calcify before the matrix; the consequence of this is that you see the cartilage-cells, being apparently converted into lacunæ and imperfect canaliculi.

You may easily trace all stages in the deposit of calcareous matter on the inside of the cells, from that in which it forms a mere ring to that in which it is so thick as to leave only a vacant space resembling an almost perfect lacuna.² Kölliker has generalised from his observations on rickety bone, thinking that in rickets the normal process by which the lacunæ are formed is visible. My observations lead me to quite another conclusion—the calcification of the cartilage-cells in the growing cartilage in rickets seems to me identical with the calcification of the same parts³ occasionally seen in enchondromata. It is a pathological process, a petrification.

¹ Die Rhachitis eine Krankheit des Kindersalters, besteht in folgenden die beiden Momente im Wachstume des Knochens betreffenden Anomalien:—1. In übermässiger die Ossification vorbereitender Zellen-Wucherung im Epiphysen Knorpel mit Verbreiterung desselben, Markraumbildung in demselben, während der Verkalkungsprocess zurückbleibt. (Virchow.) 2. In wuchernder sog. periostaler Auflagerung an der Diaphyse, welche nur sehr spät in Folge der Aufnahme von Kalksalzen verknöchert, während im Inneren die Markraumbildung vorschreitet.—Rokitansky, *Lehrb. der Path. Anat.*, vol. ii. p. 135, third edition.

² Kölliker has figured this at page 241 of his *Handbook*.

³ See Quekett's *Lectures on Histology*; West's translation of Müller; and Gamgee 'On Enchondroma of the Testicle.'

The spongy tissue is much more spongy in appearance than natural, and from the interstices of its meshes a deep red pulp is expressible. This pulp is composed of colourless nucleated cells usually containing only one nucleus, now and then two, and occasionally several. blood-globules, and in some cases a very large quantity of free fluid fat. If, as Sharpey, Tomes, and De Morgan have supposed, these cells play an important part in the completion of the process of ossification, and I am—from my own observations on rickety bones, etc.—inclined to think they do, we see in their abundance in rickety bones only a further evidence that in rickety bone there is excessive preparation for the process of ossification and arrest of the completion of the process. The periosteum is thickened over the head of the bone as over the bone generally. It attains its maximum degree of thickening just at the point of junction of the bone with the cartilage.

The periosteum of the whole bone is often more vascular and thicker than natural, but in the many post-mortem examinations of extreme rickets which I have made, I have never seen any bloody fluid as described by Guerin beneath that membrane. A crimson pulp fills the canal, and all the interstices of the tissues of the long bones. It is composed of elements identical in appearance with those expressible from the spongy tissue of the head of the bones. In this pulp, however, I have never observed any such quantity of free fluid fat, as I have noted in the heads of the bones near the line of progressing ossification.

2. The *softening* of the bones is sometimes so great that the bones which in their healthy condition are the strongest, may be bent by the most trifling force, and those which naturally are the thickest may be cut with a knife with facility.

This softening of the bones is chiefly the consequence of the diminution of their earthy salts. It seems, however, from the experiments of Lehmann and Marchand, that the animal matter of rickety bones differs, in some cases, from that of healthy bone: for, in some of their experiments, the bones yielded no gelatine on boiling.

Putting together the results of the analyses of several

observers, we gather that the bones of healthy children yield about 37 parts of organic and 63 of inorganic matters; and that those of rickety children yield about 79 parts of organic and 21 of inorganic matters.

3. The *thickening* of the flat bones is the consequence of thickening and increased vascularity of the periosteum, and of the abundance of the nucleated cells, which, with blood, form the pulp, which occupies all the meshes of the bone. The thickening is usually greatest just within or at the growing margin of the bone, so that in the cranial bones it is greatest near to or at the sutures, and least at the centres of ossification.

4. *Deformities which follow from Pressure on the Softened Bones.*—The spine is bent; the cervical anterior curve is increased; the face is directed upwards; and the head falls backwards. This curvature is only strongly marked when the muscular debility of rickets (of which I shall speak hereafter) is very decided. The vertebræ being softened and the muscles weakened, the head is no longer supported, and it falls forwards or backwards as circumstances may determine. Usually the child favours the falling of the head backwards, in order that it may see what is going on around, just as when suffering from paralysis of the third nerves, the child sits or walks with the head thrown backwards, in order that it may see under the fallen upper eye-lids. There is a posterior curvature of the spine, if the child is unable to walk, commencing at the first dorsal and extending to the last lumbar vertebra. If the child walks then the posterior curvature is limited to the dorsal region, and there is an anterior curvature in the lumbar region. This posterior curvature in the child yet in arms is sometimes so extreme that it may be easily mistaken (and I have known it mistaken) for angular curvature. They are distinguished thus:—

If the child be held by the upper part of its trunk, the weight of the lower limbs will usually remove the rickety curve, and it may certainly be straightened if the nurse hold the child by the upper part of the trunk and the physician raises the lower limb with one hand and at the same time places the other on the curved spine.

This curvature of the dorsal and lumbar spine in rickets is the consequence of the muscular weakness, and softening of the bodies of the vertebra; its direction is determined by the weight of the head, etc. It is an exaggeration merely of the curvature always existing when the child of three or four months old is sitting unsupported on the nurse's arm. Lateral curvatures in the young child are less common than the antero-posterior. Their direction is determined by the position accidentally assumed by the child. For example, if the child be carried on the left arm constantly, there is a disposition to lateral curvature, and the convexity of the curve will be towards the left.

The femur is curved forwards and outwards. This curvature is produced before the child walks, by the weight of the legs and feet. The child sits on its mother's lap, or on a chair, and the lower extremities hang pendant. The bone, being flexible, yields. After the child walks, the weight of the trunk is the chief agent in determining the curvature of the femur. The curve which existed before walking will be exaggerated.

When the tibia curves before the child walks or has been placed on its feet, the curvature is almost always outwards—an exaggeration only of the normal curve in the young child, and is produced by the child sitting somewhat cross-legged, and bearing on the floor or bed with the outer malleolus. After walking, the weight of the trunk is the chief agent in determining the bending of the tibia, and the direction of the curve will depend on the circumstances which determine the point on which the chief amount of pressure is brought to bear.

The curvature of the ulna and radius has been attributed to muscular action—to the child placing its arm around the breast of the mother, etc.; these causes are altogether inefficient for its production. The curvature of the bones of the forearm is produced by the child, owing to want of muscular power to support itself in the sitting posture, throwing part of its weight on to its arms. The child places its open hands on the chair, bed, or floor, and throws a large share of the weight of its trunk on to the bones of the upper extremities; the bones of the forearm are twisted as well as curved outward.

The humerus is sometimes bent at an angle just where the deltoid is inserted. This curve is produced by the weight of the arm when the limb is raised by the action of the deltoid, and is increased by the cause which determines curvature of the forearm.

The clavicles are often the subject of extreme angular curvature. The chief bend is always at the same spot, viz., just outside the part to which the sterno-cleido-mastoideus, and the pectoral muscles are attached. The second bend is about half-an-inch from its scapular articulation. The first curve is forwards, and somewhat upwards; the second backwards. The curvature of the clavicle is produced partly by the weight of the arm on the humeral end of the clavicle—the sternal end being supported by the muscles just mentioned, and by its ligaments,—but chiefly by the force brought to bear on it, when the weight of the trunk is thrown on to the upper extremities, the child being in the sitting posture, with the hands on the floor, or crawling.

The deformity of the greatest interest to the physician is that of the thorax. The back is flattened. The ribs are bent at an acute angle where the dorsal and lateral regions unite. At that part the lateral diameter of the thorax is the greatest. From it, the ribs pass forwards and inwards to the point where they unite with their cartilages; on that line the lateral diameter of the thorax is the least, the cartilages curving outwards before turning in to unite themselves to the sternum. The sternum is thrown forwards, and the antero-posterior diameter of the thorax is abnormally great. The consequence of the direction of the ribs being inwards, and of the cartilages outwards, is, that the thorax is grooved from above downwards on its antero-lateral face from the 1st to the 9th or 10th rib; the deepest part of the furrow being just outside the nodes formed where the ribs and cartilages unite. This groove extends lower on the left than on the right side, but it is deeper on the 5th and 6th ribs on the right than on the left side; the heart and the liver respectively supporting, to some extent, their corresponding ribs. The points of maximum recession correspond to the 5th, 6th, and 7th ribs. A little below the level of the nipple the chest expands considerably, the chest-walls being borne out-

wards by the liver, stomach, and spleen. If we examine the thoracic walls from the inside, the appearance is most remarkable, where the ribs join with the cartilages there are much greater projections than on the outside; but the 11th and 12th ribs, which are not inflexed, have the same enlargement on the inside as on the outside.

The great determining cause of the thoracic deformity is atmospheric pressure; this is aided by the elasticity of the lungs. How is the pressure of the atmosphere brought to bear on the thoracic parietes? Suppose the external thoracic parietes were made of cast-iron or other unyielding material, then the diaphragm could descend only so fast as the air could enter at the orifice of the larynx, and overcome the elasticity of the lungs. The thoracic parietes, however, in their normal condition, are not absolutely unyielding; but then there is a due relation between their strength, the power of the diaphragm and the rapidity of its contractions, the size of the orifice of the larynx, and the elasticity of the lungs. The chest walls being healthy, and the orifice of the larynx of normal size, if the young child sobs violently—*i.e.* contracts the diaphragm with abnormal rapidity and force—the most flexible parts of the thoracic parietes will fall in during inspiration.

If the orifice of the larynx be narrowed, and if the diaphragm contract with only normal rapidity and force, there will be recession of the softer parts of the chest walls at each inspiration.

Again, if the orifice of the larynx remain normal, the diaphragm act as energetically as in a healthy child, and the chest walls be softened, then, at each inspiration, there will be recession of the most yielding part of the thoracic walls.

It is this last condition which exists in the rickety child. The part of the rib where ossification is imperfect and incomplete is so soft that, at each descent of the diaphragm it recedes, and the furrow of which I have spoken is the consequence. Just in proportion as the ends of the ribs are forced inwards the sternum is carried forward, and the consequence is the remarkable form of thorax, of which a model is on the table, of which some examples are now in the hospital, and of which it is rare to pass a day without seeing

cases in the out-patients' room. Rokitansky has maintained that this deformity of the thorax is the consequence of want of power in the inspiratory muscles. I have repeatedly dissected subjects in which this deformity was strongly marked, and find that there is no correspondence between the points of insertion of the muscles of inspiration attached to the outer surface of the chest walls and the points of recession. The preparations on the table bear out my assertion. Weakening of these muscles would have as its consequence general want of expansion of the lungs, and the thorax would have the form that it obtains in senile atrophy—viz., long, with narrow antero-posterior and lateral diameters.

Again, the diaphragm is said to cause the circular recession by its direct action—by drawing in the receding parts at each contraction. On the table are some dissections, which prove, when compared with the cases and the model, that the line of recession does not correspond to the points of attachment of the diaphragm. But it does correspond to the upper margin of the liver, spleen, and stomach; and is produced—as the longitudinal furrow is—by atmospheric pressure: the parts of the parietes below being prevented receding by the organs I have just mentioned. The influence of the organs beneath in preventing the recession of the chest walls is illustrated by the *apparent* bulging of the precordial region in every case of well-marked rickety thorax. The chest walls covering the heart do not recede so much as on the opposite side, and the consequence is that the left side is much fuller than the right; and, at first sight, it might be supposed that there was abnormal fulness of the precordial region.

In excluding muscular action from all direct share in the production of curvature of the long bones in rickets, I am, so far as I know, unsupported by any authority.

LECTURE II

The Thoracic Deformity in Rickets due to atmospheric Pressure—Elasticity of the Lung merely retards the entrance of the Air—Oval Chest in Tubercle, etc.—Deformities of the Pelvis in Rickets—Circumstances which determine the Differences in the Form of the Pelvis—Deformities of the Head in Rickets, and their Causes—Mistake to suppose the Tibiæ are invariably bent in Rickets—Bones suffer as if one Organ—Arrest of Growth—Late Dentition—White Patches on the Heart—Evidence afforded by Rickety Subjects of the Truth of the Attrition Theory—Pulmonary Vesicular Emphysema and Collapse of the Lung both due in Rickets to a Common Cause, and not related to each other as Cause and Effect—Mechanism of their Production—Most Common Cause of Emaciation in Rickets, Albuminoid Infiltration of Lymphatic Glands, Spleen, etc.—Anatomical Characters of the Lymphatic Glands and Spleen when the Seat of Albuminoid Infiltration.

GENTLEMEN,—At the conclusion of my last Lecture I was endeavouring to prove to you that the deformity of the chest in rickets, of which we have so many specimens on the table, is produced immediately by atmospheric pressure—the seat of the circular groove being determined, not by the contraction of the diaphragm, but by the position of the upper margin of the liver, stomach, and spleen; the seat of the vertical groove being determined, not by the loss of power of the respiratory muscles attached to the outside of the ribs, but by the softness and want of resilience of the ribs themselves.

That atmospheric pressure is the immediate cause of the

thoracic deformity seems to me to be further shown by the following experiment:—Apply to the abdomen of a child having a rickety thorax pressure so directed as to retard very decidedly the rapidity of the descent of the diaphragm during inspiration: the result is, even though the child inspires deeply, that the recession of the chest walls is very considerably diminished; remove the pressure and cause the diaphragm to act suddenly and rapidly, and then the recession of the chest walls is not only greater than it was when you retarded the descent of the diaphragm, but greater than it is in ordinary inspiration. Again, if any cause impedes the free passage of air into the lungs,—as, for example, narrowing of the glottis,—the recession of the chest walls during inspiration is enormously increased. Under these latter circumstances, you will note that the inspiratory muscles attached to the ribs are called powerfully into play: they put forth all their force, and yet the vertical groove is deeper than it is when the orifice of the glottis is of proper size and the inspiratory muscles are acting normally. As to the part played by the elasticity of the lung in producing the thoracic deformity:—In ordinary respiration the elasticity of the lung has to be overcome before the air can enter its substance. The elasticity of the lung is the same in the rickety as in the healthy child. The elasticity of the lung aids then in the production of the thoracic deformity only by the normal impediment which it offers to the ingress of air, and consequently to the rapid dilatation of the thorax. Lateral curvature of the spine will considerably modify the shape of the posterior part of the thorax, causing the side to which the convexity is directed to bulge; but it has little effect in modifying the special deformity of which I have been speaking.

It is interesting to compare the shape of the thorax in extreme rickets, with that which it presents in a non-rickety child, when the free entrance of air into the lungs is impeded for a considerable time, as from tubercular bronchial glands compressing the trachea or the largest bronchial tubes, or from chronic laryngitis, or chronic spasm or paralysis of the larynx. There is a boy in the hospital at the present time, suffering from long-continued laryngeal spasm, depending, I believe, on tuberculisation of the bronchial glands, who

illustrates the point in question. In the rickety child, the ribs are softer than their cartilages; in the healthy, and still more so in the tubercular child, the cartilages are softer than the ribs. The consequence is, that when any chronic impediment to the entrance of the air into the lungs exists in the healthy or tubercular child, the thorax obtains a very oval form; the antero-posterior diameter being less than in health, the lateral diameter greater. If the impediment be very considerable, or the cartilages softer than natural, the sternum may be forced by the atmospheric pressure backwards, so far as to be placed on a level lower than the point of junction of the ribs with their cartilages.

In the majority of cases where this deformity of the thorax has been produced during childhood, it is said in after life to have been congenital. I doubt altogether the existence of such congenital deformity of the chest.

The rickety thorax is constant in shape, not so the rickety pelvis. It has been said, that while the pelvis of mollities ossium is triangular, that of rickets is oval; but in fact, the pelvis in rickety children is much more frequently triangular than oval. Its form will vary, first, according to the direction in which it is compressed by the spine, and its superincumbent parts, on the one side, and the heads of the thigh bones on the other side,—and the direction will vary as the child is the greater part of its time lying, sitting, crawling on all-fours, walking, or shuffling along on the floor; and secondly, according to the age at which the compressing forces are brought to bear on the walls of the pelvis, and the consequent differences in the degree of ossification of the pelvic bones,—the cartilages being in the pelvis of the rickety, as we have seen they are in the thorax, less yielding than the bones.

The rickety head is distinguished:—

1st. By the length of time the anterior fontanelle remains open. In the healthy child it closes completely before the expiration of the second year. In the rickety child it is often widely open at that period.

2ndly. By thickening of the bones. This is usually most perceptible just outside the sutures, the situation of the sutures being indicated by deep furrows.

3rdly. By the relative length of the antero-posterior diameter.

4thly. By the height, squareness, and projection of the forehead.

The two first of these peculiarities of the rickety head are the result of the affection of the bones; the two last are due chiefly to disease of the cerebrum.

In consequence of the arrest of growth of the bones of the face and the sinuses, the forehead, as mentioned by Mr. Shaw in his most able papers on rickets, seems to project more than it really does. Guerin supposed that the rickety deformities were developed from below upwards, *e.g.* that the inferior extremities always suffered before the trunk. But this is not the case. If the child is the subject of rickets before it walks, the ribs, clavicles, and upper extremities, certainly become deformed; while the tibiæ, unless the child sits so as to press on them, escape bowing. It must be borne in mind, that if a very heavy child be placed on its legs at too early an age the tibiæ may bend a little, be a little more bowed outwards than natural, though there be no reason to suppose the child to be the subject of rickets. A child then, may be, nay, very often is, rickety in the highest degree,—its ribs softened so as to endanger life, its clavicles bent at an acute angle, its wrist swollen so as to measure as much in circumference as the length of the forearm, and yet its tibiæ be as straight as in health; and again, as I have just observed, the legs may be slightly bowed outwards, and the child not be rickety.

The enlargement of the ends of the long bones, and the softening of the bones, do not always proceed in an equal degree—the softening being very often out of proportion to the enlargement; the enlargement being sometimes out of proportion to the softening.

It is not uncommon to see the thoracic deformity lessen at the time the legs are bending. I think this is due to the disease having greatly diminished, and the muscular power increased so as to permit of the child walking before the bones of the leg are strong enough to bear the weight of the body.

Rickets being a general disease, the bones are affected as

one organ, just as the arterial system is in the degeneration of age; the consequence of this is, that no one bone is ever affected without all suffering, and that whether the disease manifest itself chiefly by enlargement of the ends of the bones, or by softening of the bones, or by both in a proportionate degree.

I have described the deformities that result from the severest form of rickets, a form which is very common among the poor, and not so uncommon as has been supposed among the rich. We see, however, all degrees of softening, from that in which the ribs only yield to extraordinary pressure, as during bronchitis, and then only sufficiently to flatten the antero-lateral surfaces, to that in which they yield at every inspiration, as was the case in the child from whom the model was taken. We see all degrees of enlargement of the ends of the ribs, and of the other long bones, from that where one might maintain the enlargement was only that proper to the child, to that in which the projections on the anterior wall of the thorax and the enlargement of the wrist would strike the most careless observer.

Arrest of growth of the bones and of the parts in relation with them, is a very important consequence of rickets. This arrest of growth commences during the progress of rickets, but it continues after the general disease has terminated. Hence, not only are children stunted in growth while the subject of the disease, but they never grow into ordinary-sized adults.

All the bones in the adult whose skeleton shows the effects of rickets are diminished in length; but the lower limbs, including the pelvis, are, according to Mr. Shaw's researches, disproportionately diminished in size, and the face is small in proportion to the skull.

I must refer you to Mr. Shaw's most able papers, for further details on the arrest of growth of the bones.¹

When speaking of the deformity of the head in rickets, I mentioned one important consequence of the arrest of growth—viz., the late closure of the interior fontanelle. There is another most important consequence of the arrest of growth in rickets, which, though it is well known, does not appear to

¹ *Medico-Chirurgical Transactions*, vols. xvii. and xxvi.

me to exert sufficient influence on practice—I mean the late period at which rickety children cut their teeth.

Healthy children commence teething pretty constantly between the seventh and eighth month, and cut the last of the first set of teeth between the twenty-fourth and twenty-fifth month. As a rule, children brought up by hand, supposing them not to be rickety, and children the subjects of tuberculosis, cut their teeth early.

If a child pass over the ninth month without teeth, you should carefully inquire for the cause. It may be, that an acute illness has retarded dentition. It may be, but this is very rare, that there is some condition of the gum which interferes with the advance of the teeth. It may be, and this is infinitely the most common cause of late dentition, that the child is rickety: fail not then, when called to a child in whom the teeth are late in appearing, to look if it be rickety, for if you do fail to look for rickets, you will most likely attribute to the irritation of teething symptoms which are the consequence of the rickety diathesis; the late dentition in rickets being in itself merely a symptom of the general disorder.

✓ The rickety deformities may be very trifling, and yet the teeth be considerably retarded in their development.

You are familiar with the 'white patches' so common on the visceral pericardium in the adult. These 'white patches' are not common in children. The more advanced the age of the subject, the more frequently are they found.¹

I use this term 'white patch' to signify circumscribed opacity of the pericardium itself, and patches, limited in size, of organised lymph, seated on the surface of the pericardium. In some cases the patch is composed of a thin, smooth layer, of more or less perfect fibrous tissue, in others it is highly villous—the interior of the villi being perfectly-formed fibrous tissue, containing vessels; the exterior a layer of epithelium.

¹ Bizot examined sixteen male subjects under seventeen years of age without finding in a single case a white patch on the heart; while one-third of twenty-four male subjects, between the ages of eighteen and thirty, had white patches on the heart, and three-fourths of thirty-two subjects between the ages of forty and seventy-nine.

We find every stage between the opalescent spot due to a little thickening of the pericardium, and the most shaggy growth from its surface.

Two theories have been advanced to explain the origin of the 'white patch.'

According to the pathological anatomists whose opinions have the greatest weight in this country, white patches are produced, in the vast majority of cases at least, by inflammation of the pericardium.

Dr. Hodgkin is said to have advanced the second theory. He attributes the origin of white patches to 'pressure, aided by the movements of the heart.'¹ Dr. Wilks, in his recently-published valuable Lectures on Pathological Anatomy, has termed this the 'Attrition Theory.' Now I told you a little while since that 'white patches' are not very common in children, speaking generally. They are, however, very common in children whose chests are deformed from rickets. But, while in those advanced in years by far the most common seat of the white patch is about the centre of the anterior surface of the right ventricle, in children with rickety chests the chosen seat of the 'white patch' is on the left ventricle, a little above its apex; in fact, just at the spot which impinges against the fifth rib where it projects or knuckles inwards. ✓

The white patch is here evidently produced by attrition; and these cases form important links in the chain of facts which unite the white patches and friction to each other as cause and effect,—and they afford the strongest grounds for believing that circumscribed opacity of the pericardium, the smooth, thin layer of fibrous tissue on the pericardium and the villous-looking tuft, are mere varieties of one pathological

¹ *Lectures on the Morbid Anatomy of the Serous Membranes*, 1836, p. 98. Five-and-twenty years before the *Lectures on the Serous Membranes* were published, Corvisart wrote:—'On a attribué la formation de ces plaques blanches à l'impression des parois de la poitrine sur le cœur, quand sa contraction le porte vers les côtes.'—*Essai sur les Maladies du Cœur*. Par J. N. Corvisart. Second Edition, 1811, p. 42. Corvisart recognises the pathological identity of the white patches on the heart with the white patches found on the liver, lungs, arachnoid, etc. He doubts their origin being inflammatory:—'Le mot inflammation doit-il même être prononcé pour donner un air de vérité à l'explication de ce phénomène, dont la cause, je avoue, me paraît absolument inconnue?'—P. 44.

state, and due to one and the same cause—that cause being, I believe, friction of the spot where the white patch is found against some hard, resisting substance.

You may say, ‘But how comes the apex of the heart so far to the left in the young child as the junction of the fifth rib with the cartilage, seeing that in the healthy child, as in the adult, the apex of the heart impinges inside the nipple?’ The answer is, that the sternum of the rickety thorax being forced forward, the relative positions of the chest walls and of the heart are no longer those of health; and the apex usually strikes outside the nipple.

Unless you bear this in mind you may suppose in such a case, as I once did, from the apex impinging outside the nipple, that the left ventricle of the heart is dilated; and this mistake is favoured by the chest walls in the rickety thorax being pressed so closely in contact with the *apex* of the heart that the force of the impulse seems to be greater than natural.

In children whose chests are oval, the antero-posterior diameter being small, you may often, by moderate pressure with the stethoscope, force the sternum far enough backwards to compress the pulmonary artery; and thus you may produce a systolic basic murmur. In consequence of the length of the antero-posterior diameter of the chest of the rickety child, you cannot by any amount of pressure on its sternum produce a basic systolic murmur.

In children whose chests are greatly deformed by rickets, we sometimes find a white patch on the spleen, identical in structure with those which are found on the heart. In such cases the white patch on the spleen owes its origin to the same cause as the white patch on the heart, viz., friction against one or more ribs projecting inwards—the spleen rising and falling, you know, with the inspiration and expiration.

There are two lesions of the lungs constantly present when the thorax is deformed from rickets, and which are, in fact, direct consequences of the softened state of the ribs, and the deformity which accompanies it. These two lesions are emphysema and collapse.

I need scarcely remind you that pulmonary vesicular emphysema has been considered by some to be always secondary

to collapse of lung-tissue; and the frequent conjunction of the two lesions in the rickety thorax, at first sight, seems to lend support to the theory; for when found conjoined it has been pretty generally admitted that the emphysema is related to the collapse as effect to cause.

A careful study, however, of the lungs and thorax of a child who has died while the subject of extreme rickety deformity of the thorax, proves that the two lesions may co-exist in the same lung without having any such relation to each other.

With the abnormality of the thorax to which I have referred, there is, I say, always conjoined, as consequences, pulmonary vesicular emphysema and collapse of lung-tissue. The emphysema is that variety which has been termed insufflation. It is mere over-distention with air of the vesicular tissue of the lung. It invariably occupies the same situation in the lungs of the rickety child, viz., the whole length of the anterior border of both lungs, extending backwards for about three-quarters of an inch from the free margins. The emphysematous portion is separated from the healthy part of the lung by a groove formed by collapsed lung. The groove of collapsed tissue corresponds to these projections of the ribs inwards, which are situated at the points where they unite with their cartilages.

On the table, illustrating these facts, is a wax model of the inside of a thorax; a wax model of one of the lungs from the same thorax; and the thorax and lungs from which the models were made. The mechanism of the production of the lesion in question is as follows:—The softened ribs, instead of being drawn outwards at each inspiration, are forced inwards by atmospheric pressure; the consequence is that not only are the lobules of lung beneath not expanded, but they are compressed. The compression of the lung, aided by its elasticity, causes the collapse.

The common cause of collapse of lung-tissue is undoubtedly obstruction of the bronchial tubes leading to the collapsed portions: it has been confidently affirmed that it is the sole cause. The facts I have brought before you refute the assertion.

The emphysema of the anterior border is produced thus:—

The lateral diameter of the thorax is diminished at the part corresponding to the line formed by the junction of the ribs and cartilages. Here, as we have seen, at each inspiration the ribs recede; but in proportion as the ribs at this part are forced inwards, the sternum must be thrust forward; and just as less air, or no air, enters into the tissue under the receding ends of the ribs, so an excess of air is drawn, as we commonly call it, into the lung-tissue subjacent to the abnormally-advancing sternum and cartilages of the ribs.

The collapse is directly consequent on the recession of the ends of the ribs during inspiration; the emphysema is directly consequent on the thrusting forward during inspiration of the sternum.

The groove of collapsed lung-substance, and the border of emphysematous tissue, are all the lesions of the lungs *invariably* present in the rickety thorax. But we so often find a very large portion of one or of both lungs collapsed, and that collapse is so directly connected with the defective mechanism of the inspiratory power of the rickety thorax, and is so *very, very* often the cause of death in rickets that I must here especially direct your attention to it.

The collapse of the lung, of which I am now about to speak, occupies especially those parts of the lungs which are the seats of bronchitis,—*i.e.* the posterior and inferior portions, now and then the greater portion of the inferior lobes of one or of both lungs, and some part of the superior lobes.

If we call to mind the mechanism of the process by which we expel mucus from the bronchial tubes, we readily comprehend why a child with a rickety thorax, when the subject of bronchitis, is sure to suffer, and must often die, from collapse of the lung. When we desire to expel mucus from our bronchial tubes, we inspire deeply, and so fill the pulmonary tissue with air to the utmost; we then close the glottis, compress, by the aid of the muscles of expiration, the air contained in the lung: when the compression has reached a certain point, we open the glottis. The air is, of course, driven from the vesicular tissue of the lung with a force proportionate to the compression it was experiencing at the instant the glottis was opened. The greater the force with

which the air is driven along the bronchial tubes, the more certainly, other things being equal, are they cleared from their secretions.

Mucus in the bronchial tubes, when in any quantity, impedes the entrance of the air into the vesicular structure of the lung. But the free entrance of the air into the vesicular structure of the lung is essential for the expulsion of the mucus from the bronchial tubes; hence the violent inspiratory efforts made when an adult or a child is the subject of bronchitis. Watch an adult or child, past infancy, the subject of severe bronchitis; note how every muscle of inspiration is brought into play. He sits upright, he catches hold of the arms of his chair; he takes instinctively the position that enables his muscles of inspiration to act with the greatest results. Now one reason why the healthily-formed young child so frequently dies from bronchitis, is the flexibility of the lower part of its thoracic parietes, and the consequent mechanical difficulty under which it labours in overcoming the resistance to the entrance of the air into the pulmonary tissues of the lower lobes, when mucus is secreted in quantity into its bronchial tubes.

If the flexibility of the chest walls in the young child, normally built, has this influence on the termination of bronchitis, what must be the influence of the extraordinarily flexible chest walls of the rickety? Reduce the human chest walls to a membranous state, and the diaphragm would be as useless as a muscle of inspiration to a man as to a frog. In extreme rickets, the chest walls have more resisting power than have those of the frog, but they have infinitely less than those of the healthy child. Observe a child the subject of extreme rickets, but otherwise healthy. See the business it is to him to breathe. The chief occupation of his life is to get by effort the air into enough of the air-cells of his lungs to enable him to live. You will see at once that the least obstruction in the bronchi must suffice to render all his efforts useless.

He has bronchitis; you strip him, and see that all the recessions of the chest walls which accompanied inspiration before the attack of bronchitis are doubled in degree. The air cannot be drawn into the vesicular tissue beyond the

obstructing mucus, and, as a necessary consequence, the mucus cannot be expelled. By the elasticity of the lung and the action of the abdominal muscles the air can be expelled. It cannot from the defect in the inspiratory apparatus, *i.e.* the softening of the ribs, be drawn into the lung. Correctly speaking, we should not say, in these cases, that the child dies from the collapse; the collapse being the result merely of that which causes death—*viz.*, the want of power in the inspiratory apparatus to overcome the mechanical impediment to the entrance of the air offered by the mucus in the bronchial tubes. The loss of power which the muscles of inspiration experience, in common with those of the body generally, in rickets, increases in some degree the difficulty of overcoming the resistance to the entrance of the air. The want of inspiratory power, and the consequent accumulation of mucus in the bronchial tubes, affords an explanation of the extraordinary mortality of measles, whooping-cough, and bronchitis, in rickety subjects.

The lesions of structure hitherto considered stand in direct relation to the affection of the bones; those to which I am about to advert are dependent on the constitutional disease. Whoever sees rickets on the large scale, must be struck with the fact, that while some highly rickety children are extremely emaciated, others are so well covered with adipose tissue that they might have awarded to them, if weight decided the question, a prize at a baby-show. At first I thought that the emaciated children were probably the subjects of a deposit of tubercle; but a few post-mortem examinations showed me that such was not the case. I found emaciation carried to the highest degree, as it was in the boy, J. H., whose spleen, liver, etc., is on the table, and whom some of you probably saw in the wards; and in J. F., lately out-patient of this Hospital, and part of whose spleen is exhibited: and yet not a vestige of tubercle could be found in the body.

The emaciation in rickets is almost always due to albuminoid infiltration of one or several organs. Commonly the lymphatic glands and spleen are the chief seats of the disease; but I have not unfrequently seen the liver, kidney, brain, heart, and thymus suffer in a high degree.

I strongly incline to the opinion that in rickets this exudation is never limited to one or two organs, but that in all cases every organ, and maybe every tissue, is more or less its seat. The lymphatic glands are, in regard of albuminoid infiltration, to be regarded as one organ—just as I told you the bones in rickets are to be regarded as one organ. And as in the osseous system we never find one bone only affected with softening, and the other lesions characteristic of rickets, so in the lymphatic system we never find one gland only, or one set of glands only, the seat of albuminoid infiltration. The knowledge of the oneness, so to say, of the glandular system in reference to albuminoid disease, is of much practical importance; inasmuch as we can readily ascertain the state of the lymphatic glands in the groin, axillæ, and neck, while it is difficult to determine by the eye or touch during life the condition of the mesenteric and other internal glands.

During life we feel the lymphatic glands, if they be infiltrated with albuminoid lymph, in the groins, axillæ, and neck, varying in size from that of a large pin-head to that of a sweet-pea, hard, and very movable. The skin over them is natural in colour. There is no sign of inflammation. On examining the body after death in such a case, we find the deep glands of the neck, the mesenteric, the lumbar, the bronchial, etc., resembling those seated in the superficial regions. The cut surface of the glands is singularly pale and transparent, compact, smooth, tolerably moist, and, to the unaided eye, uniform in appearance. The substance is tough, and the gland heavy in proportion to its size. In rare cases, instead of being pale, the glands may be purplish in colour.

I have never examined a rickety subject after death in which the lymphatic glands were the seat of this albuminoid disease without finding the spleen more or less extensively infiltrated with the same substance.¹

As to the size the spleen may attain when the subject of albuminoid infiltration, it may be that the organ is only just perceptible to touch when the child inspires,—*i.e.* when the

¹ It is a point of interest that the spleen, the lymphatic glands, and Peyer's patches experience decided atrophy about the same period of life, and that diseases of the spleen, of the lymphatic glands, and of Peyer's patches are infinitely more common in the child than in the adult. ✓

diaphragm descends and detrudes somewhat the spleen; it may be that it reaches nearly to the middle line, just below the umbilicus, and downwards to the crest of the ilium.

I have never seen this extreme enlargement of the spleen in the child as the consequence of a deposit of tubercle in its substance; it has always, in my experience, depended on albuminoid infiltration. And albuminoid infiltration of such a degree as to cause very great enlargement of the spleen in children is rarely seen except in those of the rickety diathesis. Sometimes the bone disease is extreme, in others, as in the two cases to which I have before referred, the bone disease is moderate, or even trifling in degree. One of those children certainly, and the other probably, had the bone disease developed after their spleen, lymphatic glands, etc., were the seats of albuminoid infiltration.¹

The albuminoid spleen of rickety children presents after death the following characters:—It is increased in size: the increase may be trifling or extreme. Thus I have seen it little larger than in health, and I have seen it measure as much as eight inches from above downwards, over its convex surface, and four inches from side to side. It is never adherent to the parts adjacent, as a spleen containing tubercles often is, and its capsule generally, is scarcely, if at all, thickened. Its anterior border is pretty sharp; it is firm to the touch, and smooth on the surface; its weight, regard being had to its size, strikes one as considerable.

The substance is tough but elastic, and the thinnest sections can be cut with facility. The cut surface is remarkably smooth and transparent. It is not unlike what one might suppose would be its appearance if the whole organ were infiltrated with glue. Only a little pale blood can be expressed from the cut surface.

Usually the organ is pale red, but occasionally it is dark purple. The more transparent any given part is, the paler it is; the most transparent parts are almost colourless.

The splenic corpuscles are sometimes more readily seen than in a healthy spleen; they may be mistaken for grey

¹ February 2, 1860.—I have now under my care a child, aged three months only, whose spleen reaches below the level of the umbilicus. The child is suffering from catarrh. There are no signs of rickets or tubercle.

tubercles. I have never seen in the spleen of rickety children the sago-like little masses so often present in the spleens of those who die of phthisis.

The parts of the spleens of the boys H. and F., now on the table, afforded when recent good specimens of the disease. They have now lost much of their transparency, still they preserve many of their characters, *e.g.* hardness, elasticity, smoothness. I have never seen this disease conjoined with ascites.

LECTURE III

Anatomical Characters of Albuminoid Infiltration of Spleen, Lymphatic Glands, Liver, Kidney, Thymus, and Brain—State of the Voluntary Muscles—Symptoms—Age, Constitutional Symptoms which precede the Bone Disease—Derangement of Digestion—Perspiration of Head—Desire to lie cool at Night—General Tenderness—Commencement Abrupt or Gradual—Softening of the Bones most marked when the Constitutional Disturbance is Severe and the Child very young—Symptoms consequent on the Softening of the Bones—Loss of Muscular Power—Large Abdomen of the Rickety Child, and its Causes—Intellect deficient in Power and Capacity—Teeth—Skin—Fontanelle—General Aspect—The chief Causes of Death in Rickets—Influence of Softening of the Ribs on the Fatal Termination of Bronchitis—Symptoms of Albuminoid Infiltration of Lymphatic Glands, Spleen, etc.—Laryngismus Stridulus—Pathology of Rickets—Not mere want of Lime in the Bones—Causes—Special Influence of Mother—Hygienic Conditions—Treatment.

GENTLEMEN,—At the conclusion of my last lecture I described the appearances presented by the spleen and lymphatic glands when the seat of that disease from which rickety children so often suffer—viz., infiltration with a homogeneous, firm, tough, transparent, glue-like substance. And I told you that the disease was rarely, perhaps never, limited to those organs. In both the children to whose cases I referred when last addressing you, the liver and kidneys were infiltrated with the same substance as the spleen and lymphatic glands. The liver when the seat of the albuminoid infiltration, as I have observed it in rickety subjects, is larger than natural, heavy in proportion to its size, very tough, its cut surface

smooth, its substance semitransparent; sometimes the exudation infiltrates the portal canals and the interlobular spaces, in others it invades the circumference of the lobules. I have never seen the whole of the structures of the liver infiltrated.

In the boy F., the cut surface of the liver had the appearance of stiff not very well-clarified size, tinted red and thickly studded with small opaque yellowish spots. These latter were lobules, the cells of which were in a state of fatty degeneration. In some cases the infiltration of the organ is concealed by its congestion, but a brief soaking in water removes the blood and makes the lesion of structure visible. The kidney of the rickety child when the seat of this same disease is somewhat enlarged, heavy for its size, tough, more transparent than natural, and, as a rule, very pale. When the disease attains a high degree, all appearance of structure is lost to the naked eye. Fatty degeneration of the cells may accompany it.

The thymus in rickety children is often larger than natural; its increase in size being due, in some cases at least, to its infiltration with the same substance as that found in the spleen, etc. The so-called hypertrophy of the white matter of the brain seems really to be albuminoid infiltration of that structure. The transparent substance which I have described as infiltrating the organs of certain rickety children, presents neither blue, violet, nor crimson reaction with iodine and sulphuric acid, such as are said by Virchow to be characteristic of lardaceous infiltration. I am therefore inclined to believe that it differs in nature from what that pathologist considers to be lardaceous, but for which, probably, the name he has himself proposed, viz., amyloid, is preferable.

The voluntary muscles that have lost their power in rickety children are small, very pale, flabby, and soft. Examined with the microscope, their fibres are found to be singularly colourless, transparent, and soft, the transverse striæ very delicate, sometimes scarcely to be made out. I have never been able to detect in these fibres a particle of olein. The disease from which they suffer seems to be the very opposite of fatty degeneration.

Symptoms.—I have never seen congenital rickets. I have

often heard the mother say that the rickety deformities of her child had existed from its birth; but no value can be attached to such assertions if unsupported by strong confirmatory evidence. The general cachexia very rarely manifests itself before the fourth month; usually between the fourth and twelfth months. I have now a boy under my care in whom the symptoms of the constitutional disease did not manifest themselves till he was a little more than three years old, and I saw some years since, a girl, aged nine years, who was then only beginning to suffer. It is rare, however, for the general cachexia to first manifest itself after the child has passed its second year.

At the outset of the disease there is no deformity of the bones, no enlargement of the wrists, of the ends of the ribs, etc.; no thickening of the flat bones, no bending of the long bones. The child is dull and languid; its skin is hot; it is drowsy, or sleeps little; its appetite is lost; it is thirsty; if it has begun to walk, it is 'taken off its legs.' It lies about, is unwilling to play or to be amused. The bowels are irregular,—confined, or more commonly relaxed—the stools being usually of a dirty brown or leaden colour, and most offensive. The offensive odour is peculiar, resembling that of rotten, half-decayed meat. In all these symptoms there is nothing diagnostic. They might arise from deranged digestion, from improper food, or from tuberculosis. By many they are referred to that over-ridden hobby, the irritation of teething; or to that cloak for ignorance—infantile remittent fever.

When conjoined with that infiltration of the spleen and lymphatic glands which I previously described, as it was in the boy H., whose spleen, etc., were on the table at my last lecture, it is extremely difficult to distinguish from tuberculation; and in some cases it is only from the state of the lymphatic glands, or after the anatomical changes proper to rickets occur, that the diagnosis is possible.

Commonly, however, there are certain symptoms present which at once mark the nature of the disease, render the diagnosis easy, and enable us to predicate that the bone affection will show itself.

One of the most remarkable of these symptoms is profuse perspiration of the head, or of the head, neck, and upper part

of the chest. Not uncommonly, it is because this symptom has arrested the mother's attention that she seeks medical aid. She uses the strongest terms to express the amount of the perspiration: 'It stands in large drops on his forehead'—'it runs in streams down his face'—'his head is all of a reek'—'the pillow is soaked.' It is especially when the child sleeps that these copious perspirations of the head occur, but they are not infrequent at other times, as when the child is at the breast, or even resting its head on the mother's arm. A little increased exertion, a little increased temperature, may induce them at any time. When these profuse head-perspirations occur, the superficial veins of the scalp are generally large and full, and sometimes the carotid arteries may be felt strongly pulsating. At the same time that the head, face, and neck, are bathed in perspiration, the abdomen and inferior extremities are usually dry and hot.

The second symptom which especially indicates that the general derangement of which I spoke is the precursor of the rickety deformity of the bones, is the desire of the child to be cool particularly at night. As a consequence of this desire, the child kicks the bedclothes off, or throws its naked legs on to the counterpane. 'He is always catching cold, because he will lie without any clothes at night,' is what one is repeatedly told by the mother in these cases. I have frequently gone into our wards, after the children have been some time asleep, and seen the rickety children lying exposed, and have been assured by the nurses that they had put the bedclothes over them again and again, but to little purpose,—and this even in cold weather, when the other children were well covered.

A third highly characteristic symptom is general tenderness. The child cannot be moved without its uttering a cry; pressure on any part is followed by like evidence of suffering. 'He is tender all over,' says the mother, or, 'I can't think what has come to the child, if I do but touch him he cries.'

A child in health delights in movements of every kind. It joys to exercise every muscle. Strip a child, of a few months old, and see how it throws its limbs in every direction, it will raise its head from the place on which it lies, coil itself round, and grasping a foot with both hands thrust it into its mouth

as far as possible, as though the great object of its existence at that moment was to turn itself inside out. The child suffering severely from the general cachexia which precedes and accompanies the progressive stages of the bone disease, ceases its gambols, it lies with outstretched limbs as quietly as possible, for voluntary movements produce pain. Its unwillingness to be moved is so great, that, as Stiebel has observed, it will cry at the approach of those who have been accustomed to dance it—of those at the sight of whom it previously manifested extreme pleasure.¹ As the disease progresses, the child gets a peculiar staid and steady appearance; its natural lively expression is replaced by a pensive, aged, languid aspect; its face grows broad and square, and when placed upright on its mother's arm, it sits, as she says, 'all of a heap.' Its spine bends and its muscles are too weak to keep it erect. Its head seems to sink between its shoulders, its face is turned a little upwards.² The

¹ See Stiebel's admirable article on Rickets, in Virchow's *Path. und Therap.* Band 1.

² I subjoin a case illustrating some of the points mentioned in the lecture, and I do so because I am confident that the symptoms present in these cases are very rarely correctly interpreted. A. V., aged 3½, male. His present ailment commenced about four months since, shortly after 'a severe cold on the chest,' with the following symptoms:—Heat of skin, especially at night; thirst; loss of appetite; profuse sweating about the head; extreme tenderness of the whole body, so that he could not be touched without crying from the pain it caused him; relaxed bowels, the stools being, to use the mother's own words, 'stinking,' a 'rotteny smell'; desire to lay exposed at night; again to use the mother's words, 'even in that bitter cold weather he would never lay covered over,' 'in the previous winter he liked to lay warm.' Although he had long run alone, he was soon 'taken off his legs.'

Present state:—Rather thin; muscles very flabby; evident tenderness of head, trunk, and extremities. The muscles seem to partake of the tenderness; and the abdominal muscles are as tender as those of the thighs. Sits in his chair unwilling to move from morning to night. Cries if his brothers or sisters approach him. Feverish at night; throws the clothes off; sweats over the head profusely; the perspiration is limited to the head; appetite very small; bowels act once a day, but stools very offensive. Intellect decidedly less acute than that of his brothers and sisters was at the same age. Head large, square. He cut all his teeth long before his illness commenced. Spine curved backwards from about the first dorsal vertebra to the sacrum, and forward from first to last cervical vertebræ. Ribs very soft, so that there is a great recession of each rib where it joins the costal cartilage at each inspiration. Physical signs of trifling catarrh. Very little enlargement of the ends of the long bones. No enlargement of glands, liver, or spleen.

general cachexia is sometimes very severe, at others extremely trifling. And one or other of the more characteristic symptoms may be scarcely observable or wanting, while one or other may be so strongly marked, as to give a general feature to the case. Instead of commencing more or less abruptly, the disease may begin and progress most insidiously, so that the mother cannot say when the child began to suffer. Often the changes in the shape of the bones are the first abnormalities she notices.

Ere the general disease, if that be severe enough to attract attention, has lasted long, the bone deformities commence. If the attack be attended with severe general symptoms, the softening of the bones usually precedes, and is out of proportion, for some time, at least, to the enlargement of the ends of the bones. The younger the child also, the softer are usually the bones.

And now the consequences of the bone disease are super-added to the general derangement. It is strange to see a little child sitting placidly on the bed without moving for hours together,—its legs placed so as to escape pressure, its spine bowed, its head thrown backwards, the chief weight of its body cast on to its anus; and to know that notwithstanding the apparent calm, the tiny thing is indeed fighting the battle of life; for it is striving with all the energy it has to keep in constant action every one of its muscles of inspiration, endeavouring so to supply the mechanical defects of its respiratory apparatus due to the softening of the ribs. It wants no toys. It is the best of children if you only leave it alone; move it, and you inflict pain on its tender frame; show it the horse or the doll that was once its delight, and it turns away its head or stares vacantly; to notice would divert its attention too much from the performance of those respiratory movements which are essential to its existence.

At this time the appetite is often good, but the bowels are deranged; the stools being either fetid or white, or the food is passed as it is eaten. As the disease progresses the muscles lose power and waste; but the loss of power is infinitely greater than can be accounted for by their diminished size.

A girl, aged six years, was some time since brought to the

hospital, in whom the loss of muscular power was so extreme that she was not only unable to stand, but even to support herself in the least possible degree. She lay across the arms of the person who carried her like a large half-stuffed rag doll. When placed in bed, she was incapable of changing her position without assistance; nay, she could not raise her arm an inch from the bed. Long after, when greatly improved, she could not feed herself, and had to be tied in a chair and her head placed on a pillow at its back. If her head fell forward, the nurse had to raise it, for, unaided, she could not lift her chin from her breast.¹ And yet I have often seen tubercular children of the same age, with muscles much more atrophied, walking about, and performing for themselves all necessary acts, as cutting their food and dressing.

Although it is rare to see the loss of power in the muscles so complete as in the case referred to, it is very common to see children of two, three, or even four years of age, who are quite unable to support themselves in an erect position; and if a child has commenced to walk before it becomes the subject of extreme rickets it loses the power.

The abdomen of all young children is large in proportion to the size of the chest; hence the physician often has a child brought to him because its mother fancies its abdomen is larger than it should be, when, in fact, it is only of normal size.

The causes that conspire to produce the large abdomen proper to the child are,—

- 1st. The flatness of the diaphragm.
- 2ndly. The size of the liver.
- 3rdly. The shallowness and small size of the pelvis.
- 4thly. And especially the weakness of the muscles of the abdominal and intestinal parietes, which afford facilities for the accumulation of flatus.

The abdomen of the highly rickety child *is* larger than natural, and usually very much larger, for all the causes which make a large abdomen proper to a young child are greatly more potent in the rickety.

¹ This child recovered so much as to walk about without assistance. After her return home, she fell downstairs and was killed by the fall.

1st. The chest is smaller and the diaphragm more depressed than in health.

2ndly. The liver and spleen are often larger than natural.

3rdly. The capacity of the pelvis is diminished.

4thly. The muscles of the abdomen and intestines are less powerful even than they are in their normal condition ; and, moreover, derangement of the digestion is always present to favour the excessive formation of flatus.

It is curious to note the frequency with which writers state that the intellect of the rickety child is precocious, nor is it difficult to account for the origin of the error—for error it unquestionably is. In regard of the intellect of a child, speaking generally, the mother's opinion must be weighed before it is received as correct. If a child be not suffering from chorea, and the mother states that it is mentally deficient, her statement is, I believe, invariably correct ; but the mother constantly tells us that her child is very clever, quite a prodigy, when it is only a few degrees removed from an idiot. ✓

The little rickety child separated in consequence of its physical defects from other children, and thrown necessarily much into the society of adults, catches their tricks of expression, their phrases, and even some perhaps of their ideas, and hence is thought, by the mother especially, to have a larger intellect than other children.

Children, the subjects of extreme rickets, are almost always deficient in intellectual capacity and power. They are not idiots, they offer no signs of idiocy, they resemble rather children of low intellectual capacity and power much younger than themselves. Their mental, like their muscular power, is not merely lowly developed, but it retrogrades as the rickety diathesis progresses. When the disease ceases, the mind like the body regains all its powers. The muscles of those who were once rickety, in after-life are often marvelously powerful, their bones singularly strong, and their intellect certainly not below the average.

The teeth are always retarded in their development in rickety children. I dwelt on this fact in my first lecture. Not only, however, are the teeth cut late, but they fall from their sockets very early ; thus I have seen the incisor fall from the jaws before the second molars of the first set had

made their way through the gums. Occasionally, instead of falling from their sockets, the teeth decay quickly.

The back, arms, and sides of the face are very often covered with downy hair. The anterior fontanelle is frequently open till the child is three or more years of age.

On the deformities of the rickety child I dwelt so long when speaking of the morbid anatomy of the disease, that I must now pass them by.

The general aspect of the rickety child is so peculiar, that when the crooked limbs, the large joints, and the deformed thorax are concealed, you may even detect its ailment at a glance. Its square face, its prominent forehead, its want of colour, its large, staring, and yet mild eyes, its placid expression, and its want of power to support itself, like other children of its age, on its mother's arm, all conspire to form a picture which has no like in the gallery of sick children.

I told you in my first lecture how often rickets terminates in death,—that it is a most fatal disease. The great causes of death in rickets are :—

1. Intensity of the general cachexia.
2. Catarrh and bronchitis.
3. Albuminoid infiltration of organs, especially of the lymphatic glands and spleen.
4. Laryngismus stridulus.
5. Chronic hydrocephalus.
6. Convulsions.
7. Diarrhœa.

It is in rare cases only that the cachexia of rickets proves directly fatal. Death is commonly the immediate effect of some one of the other diseases which I have just enumerated. In this particular it agrees with those other general cachexiæ of children—tuberculosis, scrofulosis, and syphilis.

Catarrh and bronchitis are unquestionably the most common cause of death in rickets. The softening of the ribs renders the mechanical power by which inspiration is performed so defective, that the impediment offered to the entrance of the air by the mucus in the bronchial tubes cannot be overcome, and collapse of large portions of the lungs follows. Of this cause of death, and of the state of the lungs in such cases, I spoke at length in my last lecture.

From what I then said you will have seen that the danger of catarrh and bronchitis in rickets is in proportion, not only to the intensity of the inflammation of the air-tubes, but also to the degree of softening of the ribs; so that, in estimating the danger of bronchitis in the rickety child, it is by no means sufficient to listen to the chest, or to note the lividity of the lips, or the action of the nares, or the frequency or severity of the cough, or the heat of skin and other evidences of febrile disturbance; but you must strip the child, and note to what degree the ribs are softened, how much they recede during inspiration, and to what extent they are forced outwards during expiration.

Albuminoid infiltration of the lymphatic glands, spleen, and other organs is by no means an uncommon cause of death in rickets. The two great features, during life, of albuminoid infiltration of these organs in a young child are emaciation and pallor. The anæmia is often most remarkable; and if, as is sometimes the case, there is a little serosity effused into the cellular tissue, the child has that peculiar transparent, waxy, greenish yellow tint, which is sometimes seen in the anæmia of young women. Now and then there is decided anasarca; the face as well as the extremities, the hands as well as the feet being œdematous. The emaciation may be very great. I showed you a child suffering from this complication of rickets at my first lecture, who was very thin, and in the boy Howie, then in the ward, emaciation was carried to its utmost limit. The rickety deformities, in such cases, may be moderate or extreme; they may precede or they may follow the infiltration of the organs. The glands thus diseased are never very large. Usually they vary in size from a large pin-head to a sweet-pea. We feel them in the groins, the axilla, and the neck; they are not tender, and rarely, if ever, inflame; they roll under the finger, proving their freedom from undue adhesion to each other, to the cellular tissue in which they lie, and to the skin. When the child is very thin they are visible to the eye. They are hard to the touch, and rounded in form. The spleen is usually, at the same time with the glands, the seat of extensive albuminoid infiltration. It is strange how often enlargement of the spleen is overlooked in the child,

seeing the ease with which it may be detected by touch. In every obscure disease of early childhood the absence of enlargement of the spleen should be established. If we place the fingers of the right hand directly under the left twelfth rib, just outside the mass of the lumbar muscles, and the fingers of the left hand a little to the left of the middle line, in front and half-way between the umbilicus and the ensiform cartilage, and then press the parts forward with the right hand, and backwards and to the left with the left hand, the enlarged spleen may always be readily felt in the left hypochondriac region. We know the hard mass we feel to be the spleen, by the sharpness of its anterior margin, by the anterior margin passing from under the cartilage of the 8th, 9th, or 10th ribs obliquely downwards and inwards, towards the median line. The obliquity of this line is such that usually if continued downwards it would cross the median line about half-way between the umbilicus and the symphysis pubis. The anterior edge is usually nearer the middle line in front in the child than it is in the adult, because in the child there is a fold of peritoneum, not usually, if at all, described in English books on anatomy, extending from the left side of the arch of the colon to the left twelfth rib, and over the anterior edge of this the spleen must pass before it can extend low enough to be detected by the hand. This fold of peritoneum causes the enlarged spleen to lie more forward as well as to have a more oblique position in the child than in the adult.

The enlarged spleen is distinguished not only by its position, and by the character and the direction of its anterior margin, but also by its movability. If the spleen be greatly enlarged, and the parietes of the abdomen be thin, the notch in its anterior margin can often be felt. The liver in the rickety child is comparatively rarely so much affected with this disease as to be greatly enlarged. Its edge is usually somewhat lower than natural, allowance being made for the depression of the organ from the flattening of the diaphragm. Although the liver be not much enlarged, the edge feels harder and sharper to the touch than natural. Notwithstanding the enlargement of the lymphatic glands and spleen, there is no increase in the number of white

corpuscles in the blood. This fact I have verified by repeated observations, and on many cases.

The connection between rickets and laryngismus stridulus is very close. I think it is about four years ago that I was struck by the connection between them, and since that time I have seen a vast number of cases of laryngismus, and in every case, saving two, the child was the subject of rickets; and I believe the reason of laryngismus stridulus being so constantly referred to the irritation of teething, is that the rickety condition retards the development of the teeth, and the practitioner refers the laryngismus to that which like itself is the consequence of the constitutional disease. Carpopædal contractions, and even general convulsions, are not, as is well known, unfrequent in these cases, and are like the laryngismus to be referred primarily to the irritability of the nervous system and muscular debility. The convulsions in such cases may prove fatal, and nothing be found within the cranium to account for death.

Pathology.—In propounding his theory of Inflammation, Mr. Paget dwells in his most excellent manner on the fact, that, concerned in the process of nutrition, are four agents—viz., the nerves, the cells, the blood, and the blood-vessels—any one of which being deranged at a particular spot, derangement of the others necessarily follows; and he points out that, when inflammatory action is established in a part, all four are in an abnormal condition,—that inflammation is a disease of nutrition. In cancer, and in rickets also, without doubt, all four agents of nutrition are in an abnormal condition. Cancer and rickets, then, are both diseases of nutrition. In rickets, moreover, there is necessarily no pathological exudation or new formation; there is, so far as we know, merely a change in quantity and arrangement of normal structures and secretions. This is true not only of the bones and muscles, but of the secretions of the skin and kidney. Rickets, then, is essentially and purely a disease of nutrition, not of one part only, but of the whole body. But, if we admit this as proved we have advanced a very little way on the road to the discovery of its intimate nature. But little as we have progressed, we certainly are in advance of those who still regard rickets to be merely a chemical abnormality of the bones

—viz., a deficiency in their earthy salts. That this latter view of the pathology of rickets is altogether erroneous seems to me to be proved by the fact, that not only is there an insufficient deposition of the lime-salts in the growing extremities of the long bones, but there is an error in position of the small amount deposited there. The earthy matter is found in the cartilage-cells instead of the matrix. And yet, further, not only is there an insufficient quantity of the lime-salts and error in position of those present, but there is absorption of those deposited ere the disease began; for bones previously hard, soften. The lime is taken up from the well-constituted shafts of the long bones and from the flat bones, enters the blood, and is thrown out of the system in the urine. It has been said, Deprive a hen of lime, and she lays eggs with soft shells; deprive a child of lime and its bones will be soft. But there is no pathological relationship between the soft shell of the hen deprived of lime and the softened bones of the rickety child. In the former, the lime has never been deposited; in other respects the growth is normal. In rickets, the lime has been deposited; it is reabsorbed, and then excreted in another place from the blood; and the growth of the bone is abnormal, irrespective of the absence of lime. The agents concerned in the nutrition of the bones not only do not take the lime from the blood, but they take the lime from the bones.

It is not probable that there is any lack of lime in the blood, seeing that one secretion from the blood, viz., the urine, was found, in Marchand's experiments, to contain six times its normal quantity of lime-salts.

I adverted in my first lecture to that singular change in the chemical constitution of the bones in rickets pointed out by Lehmann and Marchand, viz., that they no longer yield gelatine on boiling,—a fact, if fact it be, which shows some far deeper change in the nutrition of the bones than a mere want of lime. I should have thought it unnecessary to dwell on so superficial a theory, had I not so often seen it adopted as a basis of treatment. And even the frequency of rickets in London has been supposed to depend on adulterations of the bread whereby its lime-salts are deprived of their solubility.

Of Meyer's opinion that rickets is an inflammatory

affection of the periosteum and endosteum, I shall only say that my many examinations of rickety children after death have enabled me to lend no support to such a notion; that I have seen no sign of pre-existing inflammation of the bone or its covering, although I have looked carefully for such. It is, therefore, so far as I can judge, not only an hypothesis without foundation in fact, but an hypothesis to which all known facts are opposed.

It has been said that there is an excessive formation of lactic acid in the stomach of the child; that this acid enters the blood, and that to its presence in the blood all the phenomena which I have described as the symptoms and lesion of structure of rickets are directly secondary. I can only say of this theory that I know of no facts which remove it from the category of pure hypothesis; while the fact that lime is deposited in abnormal situations is opposed to it.

Causes.—It is of much greater interest to the patient and to the practitioner to determine what are the circumstances which cause a child to become rickety, than it is to learn the nature of rickets. I know of no facts to prove that rickets is hereditary. The health of the mother, however, has a decided influence on the development of rickets in the child. Whatever renders her delicate, whatever depresses her powers of forming good blood, *that* tends to induce rickets in the offspring. Of the influence of the father I am very sceptical. Of this much I am sure, that where the mother is in delicate health, in a state of which anæmia and general want of power form the prominent features without being the subject of disease usually so called; there the children are often in a very decided degree rickety, and that although the father is in robust health, and the hygienic conditions in which the children are placed are most favourable. On the other hand, I know no case (though I do not deny that there may be such) in which the mother being robust, the hygienic conditions favourable, and the father delicate, the children have proved rickety.

Phthisical parents are no more likely to have rickety children than are non-phthisical parents. Nay, the facts contained in a table made for me by my friend Dr. Edwards, some years ago resident at this Hospital, and now Physician

to the Consumption Hospital at the East of London, renders it probable that they are even less likely.

It is very common for the first, or the two or three first, born children to be free from any signs of rickets, and yet for every subsequent child to be rickety. Again, if a woman have one rickety child, in the large majority of cases all her subsequent offspring will be rickety. The explanation of this fact is that among the poor the parents are generally worse fed, worse clothed, and worse lodged, the larger the number of their children—the man's wages remain stationary, the calls on his means are increased. And among the rich and poor the larger the number of children the more has the mother's constitutional strength been taxed, and the more likely is she to have lost in general power.

Whatever external conditions are favourable to the formation of hydræmic blood in a child seem to be favourable to the development of rickets. Impure air constantly breathed—food insufficient in quantity or defective in quality taken daily—deficient light—want of cleanliness.

Whatever ailments interfere with nutrition, and so with the formation of good blood: deranged conditions of the digestive organs—diarrhœa—attacks of local inflammation, especially if neglected or if treated by excess in blood-letting, mercury, or antimony. Active treatment is sometimes necessary to save a child's life; but be careful, I pray you, how you employ active depleting remedies in children—you may cure the disease for which you administer your agents, but you may at the same time kill the child by the injuries inflicted on its general powers. And with reference to mercury, I would advise you to have your grey-powder bottles marked *Dangerous, especially in alterative doses*. I do not mean that such are never given with advantage; but I do mean that where they are once given wisely they are many times given to the injury of the child's health.

The frequency of rickets among the poor is no doubt partly the result of the improper food with which the children are so often dosed even from their birth. This is the common mode of rearing the children of the poor in London.

For the first two or three days after birth their tender stomachs are deranged by brown sugar and butter, castor-oil

and dill-water, gruel, and starch-water; as soon as the mother's milk flows they are, when awake, kept constantly at the breast. And well for them if they are not again and again castor-oiled and dill-watered, and treated with a few doses of mercurials,—for the poor have learned the omnipotent virtues of grey-powder.

After the first month bread and water sweetened with brown sugar is given several times a day, and during the night the child is, when not too soundly asleep, constantly at the breast. As soon as the little ill-used creature can sit erect on its mother's arm it has at the parents' meal-times, 'a little of what we have'—meat, potatoes, red-herring, fried liver, bacon, pork, and even cheese and beer daily, and cakes, raw fruits, and trash of the most unwholesome quality as special treats, or as provocatives to eat when its stomach rejects its ordinary diet. Then instead of being weaned when from ten to twelve months old, the child is kept at the breast when the milk is worse than useless, to the injury of its mother's health, and to the damage of its after brothers and sisters, in the hope that it may retard the next pregnancy. The children are sacrificed that the passions of the parents may not be restrained. Can we wonder that rickets is prevalent among the poor of London? Can we fail to wonder that geography, history, and crochet-work form so large items in the instruction imparted at our national schools, and the doctrines of life so small. Let the girls there educated be taught that Constantinople is the capital of Turkey if it be any advantage for them to know it, but let them also learn how to dress, nurse, feed, and lodge an infant, so that it may run a fair chance of not swelling the amount of that truly awful column in the Registrar-General's returns—'Deaths under one year.'

I have told you that rickets causes, primarily or secondarily, more deaths than any other disease of childhood; from what I have said of its causes, you will have also learned that it stands very high on the list of preventable diseases.

Dr. Merei¹ collected some, not all very trustworthy, facts bearing on the comparative prevalence of rickets in different parts of England, Scotland, and Wales. The subject is one of very great practical interest. If you, who hereafter will

¹ *On the Disorders of Infantile Development and Rickets.* London, 1855.

practise in many parts of the country, would each pay attention to the prevalence of this disease in your own sphere of observation, and the causes that induce it, much valuable information would soon be collected, and practical conclusions readily deduced.

Treatment.—There is no specific for the cure of rickets. Whatever agents are calculated to improve the general health are the most efficient for curing the rachitic diathesis, and where that is not possible for preventing its worst effects. In the stage preceding the bone affection, if the child be feverish, the diet, the ventilation of the rooms in which the child lives, and the state of the digestive organs, are the points which ought chiefly to engage the physician's attention. If, as is usually the case, the child be under eight months old, and brought up by hand wholly or in part, milk diluted with about a fourth part of lime-water, and with a teaspoonful or two of cream added to the half-pint, will generally be found the best food. It is better not to add sugar to the milk—if sugar is used, it is said by some that sugar of milk is preferable to cane-sugar, and I have fancied that it is. If farinaceous food is required, a little gruel or plain biscuit, as Robb's, or baked flour, may be added to the milk. The children of the poor especially should be fed with a spoon rather than a bottle, as the cleanliness of the feeding-apparatus, so essential for preventing acid fermentation of the food, is rarely secured. A little beef-tea and bread, eggs and farinaceous pudding, may be added when the child is older. Should the child be still at the breast, it will be necessary, if the mother's milk is deficient in quantity, or defective in quality, to partially or completely wean it, or to obtain another nurse. There is no objection to giving the child two or three meals of milk and lime-water in addition to the breast-milk. Be careful to see that the child is not always at the breast, but gets its meals at stated intervals. It should be well washed all over at least once in the twenty-four hours with warm water and soap. Daily tepid or cold sponging, according to the weather and strength of the child, is useful. The room in which it sleeps should be well ventilated. It should lie alone. It is well to place a good-sized lamp in the chimney, for the purpose of aiding

ventilation. In the day-nursery light is as essential for health as fresh air.

As to medicines, it is well if the stools are very offensive, even though the bowels are rather relaxed, to give a single dose of aperient, such as a teaspoonful of castor-oil, or grey-powder and jalap, and then about once a week a dose of rhubarb and soda. These and a little prepared chalk and soda once or twice a day, to correct acidity, will generally be all that is required at this stage of the disease. When the febrile disturbance has subsided, the child must be frequently taken out of doors. In fact, it should, as far as may be, live in the open air, care being taken that it is warmly clad and not exposed to cold and damp winds. If practicable, it should be removed into the country. Dry, bracing sea air is the best. The east coast, as Scarborough and Lowestoft in the hot months; Brighton when London is enveloped in fogs. Tunbridge Wells, though inland, has a special advantage; for not only is the air of the place good for such cases, but as iron is an invaluable medicine in rickets the water from its springs is a powerful curative agent; and many young children will drink the waters readily. Steel wine, though it contains very little iron, is extremely useful. I think it one of the very best forms for administering iron to rickety children. A teaspoonful or two of steel wine, with half a grain of quinine, and a drop or two of dilute sulphuric acid, constitutes a capital mixture for such cases. It should be taken just before meals.

Cod-liver oil is considered by some French writers of repute a specific in rickets. One advises us to be careful how we administer it to children much deformed, or in the course of a week or ten days we may consolidate the bones, and then recovery from the deformity becomes hopeless. But although my experience of cod-liver oil does not confirm the statements of Bouchut, it enables me to say that it is a very valuable remedy. It is best to give it immediately after meals. Orange-juice and orange-wine are the vehicles for its administration most agreeable to children.

The condition of the intestinal discharges requires, at this stage of the disease, also to be attended to. Occasional aperients, castor-oil, or rhubarb and soda, or a little essence of

senna or magnesia, are usually all that are required. When any of the food is passed from the bowel as it is taken by the mouth, it will generally be found that it is improper in quality or imperfectly masticated.

The teeth of rickety children are so often absent or defective, that great attention must be paid to this point. It is right to pound their meat in a mortar—it is not enough to cut it small. Potatoes should be carefully mashed; and you cannot too strongly impress on the mother the importance of seeing that no little lumps escape, for such little lumps will assuredly be swallowed whole; and if you examine a dish of mashed potatoes, you will frequently find that the majority of the pieces that have escaped crushing are either half cooked only, or diseased.

Rickety children of twenty months or two years of age require a small quantity of meat every day, in addition to good beef-tea. Milk should form, for them as well as for older children, the night and morning meals.

If the stools are reported at any time to be white, they should be examined, as it may be that the white colour is due to the quantity of undigested curd contained in them. Under such circumstances, it is no use to stimulate the liver or to give alteratives; antacids and a little lime-water with the milk, or the substitution of beef-tea for part of the milk, is indicated.

With reference to the bone deformities and their consequences, you will find, when the ribs are much softened, a well-adjusted bandage round the abdomen useful, by retarding the rapid descent of the diaphragm during inspiration. It is in exceptional cases only that benefit is derived from mechanical supports to the spine and extremities.

I agree with Dr. Merei, whose work on infantile development and rickets contains very much that is most excellent, when he says, 'Both the morbid conditions of the bones, and the constitutional state of rachitic children do not admit of any notable degree of mechanical compression, or embarrassment of movement by steel apparatus, of which frequent instances are observed to the detriment of the patient.'

I have sometimes directed splints to be applied in such a

way as to project below the feet, for the purpose of preventing walking.

Time does not permit me to dwell on the treatment of the complications which so often cause death in rickets, I shall therefore conclude by two or three general remarks, having reference to certain points respecting their treatment.

Active depletion in any form, especially blood-letting, whether by a single leech or otherwise, is not to be employed in any of the inflammatory complications of rickets.

Large doses of antimony, so useful in the pneumonia of children of healthy constitution, and even in the subjects of tuberculosis or scrofulosis, are to be held as poison to the subjects of progressive rickets.

Mercury, unless as an aperient in conjunction with some other drug, is equally objectionable.

Ammonia, with or without ipecacuanha and citrate of potash, is the great remedy for the inflammatory, bronchial, and lung affections of progressive rickets.

Iron, cod-liver oil, good diet, and fresh air are the great agents for the cure of laryngismus stridulus and general convulsions.

Iodine, iron, and cod-liver oil for the hydrocephalus.

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CASES ILLUSTRATING THE SYMPTOMS
AND PATHOLOGICAL APPEARANCES
OF ALBUMINOID INFILTRATION
OF THE
SPLEEN, LYMPHATIC GLANDS, ETC.,
IN CHILDREN.

1860

THE
HISTORICAL
AND
GEOGRAPHICAL
DESCRIPTION
OF
THE
CITY
OF
NEW-YORK
AND
THE
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1790
BY
J. C. CALVERT
OF
THE
COUNTY
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CASES ILLUSTRATING THE SYMPTOMS AND PATHOLOGICAL APPEARANCES OF ALBUMINOID INFILTRATION OF THE SPLEEN, LYMPHATIC GLANDS, ETC., IN CHILDREN.¹

IN the Lectures on Rickets delivered by me during the past winter (1859-60) at the Hospital for Sick Children, I dwelt on the relation which exists between Rickets and Albuminoid Infiltration of various organs, and described generally the anatomical characters of the latter.

The object of the present article is to endeavour to fix the attention of the profession on albuminoid disease, and especially albuminoid disease of the spleen, as observed in children, by detailing cases of that rather common, though in this country rarely diagnosed, affection.

The uniformity of the symptoms, and of the lesions of structure found after death, in all the fatal cases which have come under my observation, is remarkable; and when the symptoms in the not-fatal cases are compared with those present in the fatal cases, no reasonable doubt can be entertained that the same lesions of structure were present in both.

CASE 1.—William F. B., aged 1 year and 1 month, a fair-skinned child, with light hair and eyes. Parents in decent circumstances, formerly well off; reside in an open situation viz., the Caledonian Road.

Mother and her Family.—Mother aged 35, delicate, has never suffered from hæmoptysis or other sign of phthisis; has been married twelve years, and during that time has had seven children; had syphilis when pregnant with her first child; the catamenia have always appeared, at regular intervals, during the

¹ *Medical Times and Gazette*, December 8, 1860.

whole time of suckling. Her own family, in all its branches, very healthy.

Father and his Family.—Father aged 33, healthy. He is said to have lost a brother and a sister from consumption.

Other Children.—The fourth child died, at the age of five months, from inflammation of the lungs and diarrhoea. Six children living, of which William is the youngest; the ages of the five are respectively—11 years, 9 years, 7 years, 4 years, and 3 years. Of these, the three eldest never showed signs of rickets—the two youngest are rickety. None of the children have suffered from ‘bad eyes’ or from eruptions on the skin. William had for some months no other food than his mother’s milk; then bread and cow-milk were added. When he came under observation he was still at the breast.

The following were the first notes, made September 1859. Extreme emaciation; never walked; cannot sit up on the floor; cries when moved; is evidently very tender. Has now been undressed and shrieked much, the mother says, ‘It is that (*i.e.* the tenderness) which makes him cry so.’ Perspires freely about the head and face, especially at night, the mother says ‘the perspiration is dreadful,’ cries much at night, and kicks off the bed-clothes. Four incisor teeth.

Head hot. Two months since it was, according to the mother, ‘dreadfully hot.’ Forehead projects; antero-posterior diameter of the head very great. Anterior fontanelle widely open, neither depressed nor elevated. Exceedingly irritable in temper.

Abdomen large, globular, tympanitic generally.

Bowels now relaxed; stools very offensive, watery; at times the stools are formed, and then are not offensive.

Spleen very large, movable, reaches nearly to the crest of the ilium, but not so far inward as the umbilicus. Anterior border, oblique, hard, and sharp; posterior border, perceptible to touch.

Liver reaches nearly to the umbilicus; inferior border too easily perceptible by touch.

Lymphatic Glands in groins, axillæ, and neck vary in size from small shots to small peas; very hard, round, movable.

Chest.—The deformity of the thorax characteristic of rickets, very great; the lateral groove, very deep; the antero-posterior diameter during inspiration, $5\frac{3}{4}$ inches; the lateral diameter at the point of greatest depression, $3\frac{1}{2}$ inches during inspiration; $4\frac{1}{2}$ inches during expiration.

The softening of all the long bones is very decided, the

enlargement of the ends of the bones is comparatively trifling. The bones of the forearm, upper arms and thighs, are bent, those of the legs are straight.

This boy died February 16, 1860; at that time he was 1 year and 6 months old. I saw him repeatedly after the date of the foregoing notes. He was one of the cases brought down to the Hospital for the purpose of illustrating rickets and albuminoid infiltration of the spleen, etc., at the time I was lecturing on those subjects.

He continued to emaciate and lose colour; the spleen and glands increased somewhat in size; the chest deformity attained a most extraordinary degree; the tenderness became so great that he could not bear his mother to wash, dress, or even touch him; the weight of the bed-clothes seemed to cause pain.

He continued as long as he lived to sweat profusely over the head. During the latter part of his life the stools were solid, but offensive.

The immediate cause of his death was the mechanical impediment to inspiration offered by the softened ribs. His breathing grew 'worse and worse,' 'everything he drank took away his breath,' His mother did not consider him worse than usual for more than a quarter of an hour before his death; then she noticed his breathing was more difficult, still she had no idea he was dying till five minutes before he expired, he then seemed quite unable to 'get his breath,' and died with a slight convulsive struggle.

The blood was examined microscopically during life, there was no excess of white globules.

The examination of the body was made at the house of the parents.

The head could not be opened.

Emaciation was carried to its utmost limits; the muscles were very pale, flabby, and small.

The chest deformity was extreme.

The anterior margins of the lungs were emphysematous. There was some collapse of the left lung, and extensive collapse of the right lung, quite enough to account for death.

The heart was healthy.

There was no fluid in either pleura.

The peritoneum was healthy, it did not contain any fluid.

The spleen was very large, about 4 inches in length, and three in breadth. It was free from adhesions. It was firm and tough, its cut surface was smooth, rather pale, mottled red, and almost colourless; thin sections could be made with facility, the edges remaining quite sharp; it was remarkably transparent—the more colourless the part the more transparent—no blood oozed from the surface, and only a little red watery fluid was expressible. There was no increase in the size of the splenic corpuscles, and no sago-like masses.

The liver was slightly enlarged, very tough and dark from congestion; when a piece was soaked in water the blood soon escaped, and then a little transparent substance was seen to separate the lobules.

The spleen, liver, and lymphatic glands were tested with iodine and sulphuric acid; none of the reactions characteristic of 'amyloid degeneration' could be obtained.

The lymphatic glands were pale and hard, their cut surface pale, smooth, and homogeneous.

The mesenteric glands had the same characters. These latter varied in size from a small shot to a small split-bean.

In the case of William F. B. the extreme tenderness of the trunk and extremities, the profuse perspiration of the head, the desire to lie cool at night, and the deranged state of the intestinal secretions and functions, were all well marked. These symptoms are proper to rickets, and nowise connected with the albuminoid disease. Death resulted, as it so often does in rickets, from extreme softening of the ribs. At last the softness was such that dilatation of the thorax became impossible. Five months before William F. B. died, it will be noted, that at each contraction of his diaphragm the softened ribs were pressed so far inwards by the weight of the atmosphere, that the lateral diameter of the thorax was diminished to $3\frac{1}{2}$ inches; when the lungs were compressed, preparatory to the expiratory act, the diameter at the same part increased to $4\frac{1}{4}$ inches, thus affording a good illustration of the power of the expiratory efforts, by forcing air from one part of the lung to another, to distend the soft parts of the thorax. After death the extent of the pulmonary collapse indicated clearly the mechanical difficulty that had existed

during life in the apparatus for increasing the capacity of the thorax. The least obstruction to the entrance of air afforded by the presence of a little mucus in the bronchial tubes, sufficed to cause collapse of lung-tissue. No air could be drawn beyond, from defect of inspiratory power and the mucus consequently could not be coughed out.

CASE 2.—Joseph L. B., aged 15 months,—dark eyes,—light hair,—lives at Camden Town.

Mother and her Family.—The mother, aged 28; very delicate in appearance; anæmic; vomited blood on one occasion; has been married six and a half years, and during that time has had two miscarriages and three living children. Her mother died, aged 51, of dropsy; her father is still living and healthy, aged 57. She is the only surviving child of five: two of her brothers died when young, and two of her sisters died of consumption.

Father and his Family.—Father, aged 27, did not run alone till he was 3 years of age; some time since spat up blood daily for a week or two; is now reported to be healthy. His mother and father are living, and are said to be healthy.

Other Children.—Of the three children born alive, one died, aged 4 months, of convulsions; one, aged $4\frac{1}{2}$ years, is now the subject of rickets; Joseph is the third.

Joseph was, at the time of birth, a fine healthy child; but, when six weeks old, he had hooping-cough, and since that has never been well. He was weaned when eight months old because of the delicacy of the mother. He was fed from a very early period on bread and milk, and 'a little of anything we had.' Some months before the subjoined notes were taken, he was a patient of my colleague, Dr. Harris, and was then rickety and suffering from the perspirations of the head, general tenderness, and other symptoms characteristic of progressive rickets.

Present State.—Extreme emaciation and pallor; at places on the skin are many large, hæmorrhagic spots—the mother says that they are flea-bites, but adds, 'the marks are so long going away.' There is a bruise on his right cheek, the result of a blow given at least three weeks since; there is also a bruise (how produced is not known) on the right leg. Considerable rickety deformity of the thorax from softening of the ribs; clavicles bent; spine curved backwards; very little enlargement of the wrists or ends of other long bones. Tibiæ and fibulæ straight. Tolerably intelligent; anterior fontanelle very widely open—the mother thinks it is larger than it was. No teeth; appetite very good; no thirst; abdomen large. Has lately passed from his bowels, on 'several

occasions, a little blood and corruption.' For a long time the bowels have been now confined and then relaxed, the stools having a very 'nasty smell,' 'deathly.'

Spleen.—Anterior and posterior margins readily felt. The anterior margin passes from under the cartilage of the eighth rib on the left side obliquely downwards and inwards, crossing the middle line about $\frac{3}{4}$ inch above the umbilicus; it extends $1\frac{1}{2}$ inch to the right of the umbilicus; from that point the inferior margin passes in a curved direction downwards to the pubis, and then upward to the anterior superior spinous process of the ilium. The convex surface of the spleen is smooth, its anterior border hard and sharp, the notch in its edge being perceptible just above the umbilicus. The whole organ is very movable.

Extreme length of the spleen from above downward, $6\frac{1}{2}$ inches. Extreme breadth of the spleen from before backward, 5 inches.

The liver is harder, but scarcely, if at all, larger than natural.

The lymphatic glands in the neck, axillæ, and groin are about the size of large shots, hard, and very movable.

The child evidently has no idea of walking.

Blood.—A drop from the tip of the finger examined. No excess of white corpuscles.

This boy was admitted into the Children's Hospital the day after the foregoing notes were made, and died the following day from the same cause as William F. B., *i.e.* from the softened state of the ribs diminishing the inspiratory power and so necessitating extensive pulmonary collapse when a little mucus in the bronchial tubes afforded a trifling impediment to the free entrance of the air into the lung-substance. In this case, as in the last, death was sudden and not expected at the precise moment it occurred.

The body was examined twenty-five hours after death. Emaciation was extreme. The upper and lower extremities were slightly œdematous. Scattered over the trunk and extremities were numerous small purple spots; at first these were supposed to be flea-bites, but they were probably true purpuric spots, seeing that similar spots were subsequently found in the mucous coats of the stomach, the pleuræ, peritoneum, etc. On the right side of the face was a large purple patch. The muscles were extremely pale. The rickety deformities of the chest-walls, etc., were as noted during life.

Head.—On detaching the scalp there were found at the frontal eminences and at a point on either side just anterior to the parietal protuberances, red patches of about an inch and a half in length. At first sight the colour seemed the result of extravasation of blood, but on cutting into the bone they were clearly seen to be due to increased vascularity of the bone itself at those parts. On removing the calvaria several small extravasations of blood into the substance of the dura mater were noted. The brain itself was singularly pale, and everywhere of good consistence. About one drachm of fluid was found in the ventricles, and rather more fluid than usual at the base of the brain. At the junction of the frontal and parietal bones the thickness of the calvaria was three-eighths of an inch; at the parietal eminence one-twelfth of an inch only; at the frontal eminence one-eighth of an inch.

Chest.—The pericardium contained a normal quantity of fluid. The heart was healthy. There was only a trace of fluid in the pleura, numerous spots of extravasated blood were seen beneath the serous membrane near to the spine. There was a furrow of collapsed lung-tissue corresponding to the projections inwards of the large extremities of the ribs; anterior to this furrow the lungs were emphysematous. The middle lobe of the right lung, as well as the anterior inferior angle of the upper lobe, and the inferior border of the lower lobe of the left lung were also collapsed. There was only a little mucus in the bronchial tubes. The bronchial glands were large and red. In the peritoneal cavity was about half an ounce of yellowish serosity. The subperitoneal cellular tissue in the iliac fossæ and in the mesentery was infiltrated with blood. The mesenteric glands varied in size, some being not larger than a pin's head, others an inch in length. They were smooth and red. The liver weighed $10\frac{1}{4}$ ounces; its capsule was transparent; its cut surface was smooth and pale; its substance tough. Viewed with a lens the lobules were generally tolerably distinct, though here and there more fused together than is natural.

The *spleen* was much enlarged; its length was, over the convex surface, $7\frac{1}{4}$ inches, its breadth $3\frac{3}{4}$ inches.¹ Its weight was $9\frac{1}{2}$ ounces. Its capsule was transparent and easily separable from the substance. The substance of the organ was very firm and tough, and its cut surface much more like that of the liver than the spleen. All its parts seemed matted one to another. Some foreign substance had evidently infiltrated its whole texture, and bound it compactly together. The section being examined by

¹ The oblique position of the spleen during life accounts for the difference in measurements before and after death. The measurements after death were taken when the organ lay on the table.

a lens, this substance was noted to be colourless, transparent, and spread generally and pretty equally through the whole organ. The blood which escaped from the spleen was pale and thin—watery-looking.

The *left kidney* weighed 1 ounce 5 drachms. The capsule separated readily, leaving the surface even. The substance of the organ was pale and firm, its cut surface smooth. The pyramids were remarkably anæmic. The cellular tissue about the pelvis of the kidney was infiltrated with blood.

The *right kidney* resembled the left in all particulars.

The *stomach* was distended with flatus and food. Its mucous membrane generally softer than natural, and at the cardiac half studded with purpuric spots.

The mucous membrane of the whole extent of the *intestines* was very soft; the solitary glands of the small intestines and Peyer's patches were unduly prominent, the mucous membrane over the latter being redder than natural and extremely soft.

Examined microscopically, the cells of the liver were found to contain scarcely a trace of oleine,—a few proteine granules were seen in their interior and also in the cells of the kidney. The substance which infiltrated the spleen so extensively was homogeneous. Iodine and sulphuric acid gave with it none of the reactions characteristic of amyloid degeneration.

In addition to the points previously mentioned, these cases illustrate the influence of delicacy of health in the mother on the development of rickets in the child; the fact that when a woman has borne one rickety child, all her subsequent children may be expected to suffer from the same cachexia; the want of relation observed in many cases of rickets between the enlargement of the ends of the bones and softening of the bones; the late period at which rickety children cut their teeth.

In Case 2, from an early age the child had eaten, or rather, as it had no teeth, had swallowed, daily, meat, including pork and bacon, cheese, potatoes, and whatever beside the parents had for their food. It will be observed that no trace of tubercle existed in the body of either child.

The emaciation of the children and the extreme pallor of the skin and mucous membranes, were due to the albuminoid disease, and no doubt this complication tended greatly to favour the progress of the rickets. The œdema was due to the state of the blood, and to the impediment to the

circulation offered by the state of the thorax. Although loss of power in the muscles is a symptom of rickets, still it is probable that the size and power of the muscles were further reduced by the albuminoid disease. The large size the spleen attains when the seat of albuminoid infiltration ($9\frac{1}{2}$ ounces, the liver being $10\frac{1}{4}$ ounces in Case 2), the condition of the lymphatic glands when so diseased, and the appearances which both ordinarily present after death, are all well illustrated by these cases. The liver in both cases was found to be the seat of albuminoid disease, but the exudation was noted in the first case to be almost limited to the periphery of the lobules. During life, the hardness of the edge of the liver had led me to the conclusion that it was, like the spleen and lymphatic glands, the seat of albuminoid disease. As is so constantly the case in this disease, there was neither jaundice nor ascites. The kidneys were very slightly affected with the disease in both cases. The tendency to hæmorrhage into various tissues in the second case is worthy of note.

ON TUBERCULOSIS AND THE EVIDENCES
OF THE DEPOSIT OR FORMATION
OF TUBERCLE IN THE CHILD

Lectures delivered at the Hospital for Sick Children

1861

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ON TUBERCULOSIS AND THE EVIDENCES OF THE DEPOSIT OR FORMATION OF TUBERCLE IN THE CHILD.¹

LECTURE I

GENTLEMEN,—Whoever desires to comprehend the diseases of infant life must view them in conjunction not only with the diseases of adult life, but also in their relation to the diseases of declining life. The pathological processes of the one period throw light on those of the other. Now, it is the consideration of some easily-understood physiological or pathological condition in the child which enables us to understand the less patent disease in the aged, and then it is some easily-understood pathological process in the aged which throws light on an obscure point in the diseases of the child. He, then, who would study successfully the diseases of childhood must study them as part of the great field of pathology, and not merely as a speciality. To devote time to the study of this or of that special disease in its relation to other diseases is most useful to him who studies and to those who are to profit by his studies. To study this or that disease, or set of diseases, as a mere specialist, to view them by themselves only, is to cramp your own minds, to obtain a narrow view even of the special subject,—to injure yourselves mentally, and to retard the progress of scientific and even of mere practical pathology.

Before passing to the special diseased states to which I propose in this and the succeeding lecture to direct your attention, allow me briefly to review some of those peculiarities in the structures, the physiology, and the habits of the child which dispose it to diseases never seen in the adult, modify

¹ *Medical Times and Gazette*, July 6 and Oct. 26, 1861.

those common to both, make rare those frequent in after life, and exclude those we daily witness in the aged.

The body of the child is growing. The organs and functions by which the building up of the frame is performed are in full activity; they are more perfect than in after life; the power of repair in the child is marvellous. There is no degeneration of structures essential to life or to the progress of growth. Only parts which have finished their work, have contributed probably to the perfecting of other parts essential to the perfecting of the body, decay, or degenerate, *e.g.* the thymus.

I must for a moment break the thread of my discussion, to tell you what I mean by this word 'degenerate,' so much abused in its use. I mean by it changes of the intimate structure of tissues or organs, independent of life force. The chief degenerations of the structures of the body observed by the pathologist may be grouped under three heads:—1. Disintegration of structures, *e.g.* granular degeneration of muscular fibres. 2. Precipitations on to or into the elementary constituents of tissues and organs, *e.g.* calcifications of various parts. 3. Re-arrangement of chemical elements, *e.g.* fatty degeneration.

The body of the child is growing. Hence, not only is there activity of all the building-up functions, but there is imperfect formation, and consequent imperfect performance of function of many of the parts which are being built up, *e.g.* the bones.

Contrast this with the state in old age; not only has the body then ceased to grow, but all its parts have passed by the date of their perfection. The structures especially concerned in building up the frame are no longer needed, they have long been inactive, and inactivity of a function involves the degeneration of the structures performing it. They have then either disappeared or are degenerating prior to disappearing. The power of repair is almost *nil*, and death by old age is death by the degeneration of structures, and consequent imperfect performance of functions essential to life. It is no longer parts which have finished their building-up work which degenerate, it is those concerned in the preservation of life itself which decay. The more gradual and the

more general the degeneration, the more correctly do we speak of such death as death from old age. It is rare, as you well know, for all the parts to degenerate equally and to that degree that the functions of all cease together. Usually, some one organ passes to decay more rapidly than the others, and death is said to occur from the lesions of this or of that one organ—as the old watch that has long ceased to keep time at last stops, because some one part necessary to the movements of its hands has worn a little faster than the others. All its structures are more or less decayed, but it finally ceases its work because the balance-wheel breaks, the chain gives way, the main-spring snaps.

If, now, we consider the tendency of special parts to degenerate, we find the lymphatic glands, the spleen, Peyer's patches, the solitary glands of the small intestines, all actively at work in the child. The lymphatic glands are large; the Malpighian bodies of the spleen are swollen; Peyer's patches and the solitary glands are prominent. In middle life they all begin to waste, and they go, like the thymus, when the function of building up is done; when perfection has been reached; when life begins only to flag, and long ere we like to say old age is on us.

There is one tissue which plays so large a part in the performance of some of the functions essential to the continuance of life, and the importance of the decay of which has hitherto, it seems to me, been imperfectly appreciated, that I must for an instant direct your especial attention to it—I mean the elastic tissue.

A loss of elasticity gives its most characteristic aspect to age. Think of the smooth cheek of the child, and compare it in your mind's eye with the wrinkled skin of the aged; and remember how large a part in this difference is played by the difference in the elasticity of the skin at the two extremes of life. You know the influence which the elasticity of the coats of the arteries plays in the circulation. In the child the elasticity of the arteries is at its maximum. Bend the child's arm, and see how perfectly the brachial artery follows the changing length of the limb between the elbow and the axilla; then bend the arm of the old man, and note how the same artery is thrown into an S-shape coil—it has

lost the power of retracting, and therefore of adapting itself to the diminution of the length between the elbow and the axilla when the forearm is flexed on the upper arm.¹ Granular disintegration of its structure, precipitation of albumen and fibrin, and of calcareous granules into them, and rearrangement of their chemical elements have taken place. The one or other of these degenerations predominating in different cases, the elasticity as well as the contractility of the vessel is lost, and thus the whole of the arteries are practically lengthened;² friction is therefore increased, and undue impediment to the flow of blood through them is the consequence, and the enlargement of the heart, said to be proper to the aged, necessarily follows.

It is, in reference to the subjects we are reviewing, of interest to note the fact that it is common for special parts to experience the decay proper to age, not only before the period of life at which such decay is the natural course of events, but also long before other parts of the body decay. Thus, one man is old as regards his scalp—his hair is grey, or his hair-follicles have decayed and wasted so as no longer to perform any function, and the man is bald; in another it is the alveoli which waste before their time; in a third the heart and arteries; in a fourth the nervous system, etc.

This tendency of particular parts to grow old before due time, is sometimes hereditary. Early baldness, early falling of the teeth, and an early aged-look, we all know, occur in many members of the same family at about the same period of life. So, also, we see member after member of the same family cut off about the same age by apoplexy, by heart-

¹ If an equal length of the artery be removed from a young and an old subject after death, and the elasticity of the two arteries tested, the difference is very remarkable, and that even when to the eye the artery of the old person appears healthy. The whole arterial system degenerates at the same time, and, consequently, the state of the brachial artery is an index to those of heart and head. I have collected many facts indicating the importance of this subject and its bearing on diseases of various organs.

² As in the healthy person, the length of the artery varies with the positions of the limb, and as the artery scarcely shortens when its coats are degenerated, it is, of course, practically speaking, lengthened. If the disease be extreme, then it is actually lengthened, as shown by its tortuous condition. When the limb is extended dilatation of the artery necessarily accompanies the lengthening.

disease, by asthmatic complaints as they are called. These diseases being the result, in fact, of degenerations occurring at or about the same period of life,—the arteries, or the heart, or the lungs having been so constructed originally, as to begin to decay after so many years of ordinary wear,—though care may retard, abuse hasten the decay, and so anticipate or delay the term natural to the individual. Men are old, then, physiologically considered, not when they have lived so many years, but when the structures essential to life have degenerated to a degree almost incompatible with the continuance of life.

In my Lectures given in this place last year, I contrasted the state of the nervous system, and especially the reflex nervous system, in childhood and age, I need not repeat what I then impressed on you.

Applying what I have said to the special diseases of childhood, we understand that as malformations and rickets are diseases of development and of rapid growth, they must be limited to childhood; that as the mechanical inspiratory apparatus is imperfect in the child (from the softness of ribs and cartilages), bronchitis must be very fatal to it; that from the irritability of the reflex nervous system convulsive affections of excentric origin will be common in early life; that as the lymphatic system, the glands of Peyer, the spleen, etc., are in a state of activity in childhood and youth, they will be prone at that time to certain diseases; that as syphilis, tuberculosis, and scrofulosis are the most hereditary diatheses, they will manifest themselves early; that as diseases of the arteries, veins, and heart,—excluding acute inflammations and malformation and their effects,—are the consequences of degenerations, they will be unknown in infancy; that hæmorrhage into the brain and softening of that organ (excluding softening from acute œdema) being due in the vast majority of cases to degenerative disease of the arteries, will be infinitely rare in childhood; that as cirrhosis of the liver and kidneys and certain stomach diseases result, in the vast majority of cases, from intemperance, they will be very rare before puberty. And so we might pass in review the whole of those diseases which are proper to, unknown in, rare in, or especially mortal in early life, and trace these

peculiarities of each to established physiological or pathological laws.

In the Lectures which I gave in this room last year, I directed attention to one of the most widely spread of the diathetic diseases of childhood, viz., rickets. To-day, and in my succeeding Lecture I propose to consider another great diathetic disease so common in, though not like rickets proper to, childhood, viz., tuberculosis.

As when describing rickets I drew altogether from my own experience, so in describing tuberculosis and its consequences, I shall draw my descriptions as little as possible from the observations of others, and shall limit myself to what I have seen. I shall have to leave out little of importance, because the large field which this Hospital affords for the study of this common disease affords ample opportunities for observing all its chief general varieties and local developments. There are three words I shall so constantly use, to which such different meanings have been attached, that it will be necessary for me to explain the meaning I attribute to them.

When I say a child is tubercular, I mean that it has tubercles in one or more of the tissues or organs of its body. When I say a child is tubercularising, I mean that tubercles are being deposited or formed in one or more of the tissues or organs of its body. And when I say a child is the subject of tuberculosis, I mean that it is from some general state of all its parts disposed to tubercularise.

In the adult, and yet more so in the aged, occupation, exposure, mental wear, the passions, and accidental diseases, have so modified the general aspect of the man, that although much may still be learned by the practised eye, without a question to the patient, still we are often far from correct in our surmises. It is very different with the child; in reference to its diseases the eye is the great inlet of knowledge. It is so in regard of tuberculosis. The ear and the touch, the test-tube and the microscope, give us no aid in the diagnosis. We judge almost solely from inspection of the patient.

It is when tuberculosis is inherited from the parents that its characteristic features are the most strongly marked. The germ anterior to the formation of the blood, and even before

it has divided into parts, is the subject of tuberculosis, and, as a consequence, the parts formed from and by that germ partake of its constitution; and we see the result, not in this or in that part, not in the blood or bones only, but in every fibre and every cell of the frame, at least in every part made up of such cells. Let me sketch for you the appearance of a child who is the subject of well-marked tuberculosis,—one concerning whose predisposition to become consumptive we should have no doubt. I did so briefly in the Lectures I gave here last year, when contrasting the leading features of tuberculosis, scrofulosis, rickets, and syphilis.

The child's loveliness is often the pride of the parents; they are charmed with its tall and thin figure, its straight and thin limbs, the delicacy and transparency of its skin, the clearness of its complexion, the beauty of its eyes, so bright, with so large pupils, and so long lashes, the oval contour of its face, and the silkiness of its hair; it is so forward, so intelligent, so clever. It lives rapidly, it cuts its teeth early, it talks before others, it quickly runs alone.

In the mother's and the poet's eye—and every mother is a poet when describing her child—the child is a pretty little fairy; in the physician's eye it is an interesting pathological specimen. If we examine such a child a little more closely, we find that its bones are all small in circumference, long, and singularly firm; that its cartilages are comparatively soft. We press our stethoscope on its sternum, and we are surprised to find how easily it is depressed—*i.e.* how flexible are the cartilages. The ends of its long bones are very small, and on cutting them through vertically, we are struck by the thinness of the layer of cartilage which, in the state of preparation for ossification; instead of being, as in rickets, greatly increased, is in tuberculosis diminished. Health is intermediate. This difference is most striking in the ribs—*i.e.* at the points where the ribs unite with the costo-sternal cartilages: in rickets, a row of nodules; in health, ditto; in tuberculosis, *nil*.

But there is another important difference between the state of the ribs in the rickety child, the healthy child, and that the subject of tuberculosis—I mean the firmness of the ribs. In rickets, the softness of the ribs modifies all its chest

disease in regard of symptoms and danger, and give to the chest that remarkable form with which we are familiar. In tuberculosis, the ribs, in common with all the bones, are even firmer than in health; and the consequence is, that any diminution in the size of the lungs must be followed by a very different alteration in the shape of the thorax from that which occurs in rickets. In rickets, supposing an impediment exists to the entrance of the air, the soft ribs are thrust in by the weight of the atmosphere, and of course recede or are driven in farthest at the part where they are the softest.

Now we may lay down this law in regard of the diseases of the lung—that all diminution in the size of one or both lungs, if not accompanied by the presence of air or fluid in the pleura, necessitates the falling in of the chest parietes; the walls of the chest follow the receding lung. In tuberculosis the lungs are small; they do not grow with the increasing length of the trunk; they are not only absolutely, but relatively to the height, small; and bear in mind that this want of size in the lungs is anterior to the deposit of tubercle. It is not the upper lobe which is especially affected—every part of the lung seems equally diminished in size; but while diminished in size absolutely and relatively, we often find its air-vesicles dilated—the lung rarified. There is no damage in texture—no loss of elasticity—only a little dilatation of air-vesicles, compensating a little, and but a little, for the smallness of the organs.

I told you that the chest walls must follow (no air or fluid being in the pleura) the receding lungs; and that little lungs necessitated a small thorax. In rickets the softness of the ribs permits of their being driven in by the weight of the atmosphere. In tuberculosis the firmness of the ribs forbids such a change. Nature here uses other methods for diminishing the capacity of the chest.

We find three forms of thorax in those who are the subjects of tuberculosis, all three consequent on the small lungs of the child. 1. The long, almost circular, thorax. 2. The long thorax with narrow antero-posterior diameter. 3. The long pigeon-breasted thorax. The key to the understanding of the two first is afforded by the thorax of the aged, who are the subjects of atrophous emphysema, or

senile atrophy of the lung. As the lung diminishes in size the ribs become more oblique, and as a consequence the four upper intercostal spaces next the sternum are remarkably widened, the four lowest almost obliterated. The sixth, seventh, eighth, and ninth ribs are united at an acute angle with their cartilages—the ribs passing downwards, the cartilages upwards,—to be affixed to the sternum or to cartilages above. The result of this alteration in the obliquity of the ribs is that the antero-posterior and the lateral diameters of the thorax are greatly diminished, and the diaphragm, remaining as regards its highest point at its proper level, is necessarily, by the lowering of its attached margin, considerably more arched than natural; while a certain number of the ribs, in some cases even the eighth, ninth, tenth, eleventh, and twelfth ribs—lie in contact over the greater part of their extent with the diaphragm—have, that is to say, no lung under them. In this way, although the thorax is much lengthened, its capacity is greatly diminished.

The cause of this form of thorax in the old person whose lungs atrophy is the almost absolute inflexibility of their outer chest walls, the result of their costal cartilages being to a great extent calcified. Then, whenever the cartilage and the ribs in the child, adult, or aged person are particularly firm and the lungs atrophy generally, the thorax obtains the form I have described; we get the long, circular thorax, with narrow, but pretty equal antero-posterior and lateral diameters, oblique ribs, wide upper intercostal space next sternum, and obliterated lowest intercostal spaces.

Now, if you have followed my line of argument, you will have already understood the cause of the difference between the flat and the circular long thorax. The difference is due to the relative softness of the cartilages in the two. If the cartilages be soft you will have the chest flattened from before backwards, as well as lengthened; and if they be very soft you will have the sternum below the level of the cartilages.¹

The third form, viz., the long thorax with pigeon-breast,

¹ Various functional derangements of the heart, basic murmurs, etc., result from the caging up of the heart when the antero-posterior diameter of the thorax is thus much shortened.

is the consequence of repeated trifling catarrhs affecting the lower lobes of the lungs of a child whose lungs generally are small. The air during the catarrh being unable to find ready access into the lower lobes of the lungs, the lower ribs are driven inwards at each descent of the diaphragm, bearing the sternum forward. The degree to which the sternum is advanced will depend on the height to which the impediment to the entrance of the air extends in the lungs, or, rather, in the bronchi. The sixth and seventh ribs have, I think, the greatest effect in advancing the sternum. If a child be constitutionally healthy and suffer repeatedly from bronchitis, we ultimately obtain the same form of thorax; for a certain amount of collapse becomes permanent, and the lower lobes of the lungs are permanently diminished in size.

How is this last form of thorax to be distinguished from the pigeon-breasted thorax of rickets? In the early stage there is no danger of confounding the two; but when the ribs have consolidated, I have often seen the one mistaken for the other. There is, however, little difficulty in distinguishing the deformity consequent on rickets from that I am now describing. In the pigeon-breast from rickets, all the ribs being softened, the deformity extends certainly as high as the second rib. In the pigeon-breast from catarrh or bronchitis, the upper part of the thorax is flattened from before backwards; it is only the lower end of the sternum which is thrust forward. The child is pigeon-breasted, but it is only pigeon-breasted at the lower part of its chest.

We often find, in conjunction with this form of thorax, a knuckling forward of the cartilages just next to the sternum, the yielding cartilage being compressed between the solid rib and sternum.

I desire, then, fully to impress on you that small lungs are common in the child who is the subject of tuberculosis, and that the form of thorax which in the child indicates small lungs is one of the three I have described.

By inspection we detect these forms of thorax; the eye, then, is the great inlet of knowledge here as in reference to the general aspect of the child when seeking to determine its disposition to become tubercular. When I come to speak of the physical signs which indicate that the child is tubercular-

ising or even tubercular, we shall see more fully the importance of the knowledge the eye conveys to us. How often is a child and even a young adult tubercular in the most extensive degree without our being able to prove by auscultation, percussion, or palpation, that any organ or tissue is the seat of tubercle? The build of body characteristic of tuberculosis is not, however, always notable when the disposition to the deposit of tubercle is considerable; and this is to be expected, seeing that tuberculosis is admitted to be sometimes acquired after the building up of the body is nearly or quite complete. And again, when tuberculosis is inherited from one parent, the transmitted influence of the non-tubercular parent may modify the aspect of the child.

The deposit or formation of tubercle may take place slowly or rapidly. Tuberculation may therefore be acute or chronic; but I need scarcely remind you that there is no sharp line of demarcation between the acute and chronic forms of any given disease. There is no disease that occurs as an acute affection and also as a chronic affection but offers all intermediate terms of duration. Tuberculation in the child is distinguished from tuberculation in the adult by the large number of organs in which the deposit occurs at or about the same time and by the small amount of the deposit at one spot. It is this latter which makes palpation, percussion, and auscultation so often small aids in the diagnosis of tubercle, and which compels us to trust especially to a study of the general symptoms, of the conditions appreciable by the eye, and of the etiology of the disease.

Symptoms of Acute Deposit of Tubercle.—Children rarely tubercularise acutely without they inherit from one or both parents a disposition to become tubercular, or have recently been the subjects of measles or whooping-cough, or have been placed in singularly unfavourable hygienic conditions as regards air and light. With regard to these latter, they are, like errors in diet, more often merely exciting causes of tuberculation in the child already the subject of tuberculosis, than the originators of tuberculosis itself. The cases of acute tuberculation that I have seen may be grouped so as to form three varieties, which we may call the insidious, the active febrile, and the adynamic.

In the first variety the child is observed to be languid, to be unwilling to make exertion, sits or lies about, leaves its playmates, is dull and heavy or irritable in temper; the skin is hot, but the degree of heat varies much. At some parts of the day it is scarcely greater than natural; at others it is, as the mother says, burning. The thermometer shows that there is an appreciable difference in the temperature at different parts of the day and on different days. At times it is almost or quite natural; at times it rises to 102° , and even higher. On the whole, the elevation of temperature is most marked at night. Usually when the temperature is high the cheeks, one or both, are flushed. It is the irregular feverish heat which makes the greatest impression on the mother. The pulse is always frequent, but very rapid when the temperature is high. The tongue is furred more or less, and the lips and nares dry, and often bleeding from picking. The appetite is lost or variable, and only to be excited by tasty and indigestible substances; the bowels confined or irregular; the stools more or less clay-like, or putty-like, or pale, or parti-coloured and offensive. The abdomen is free from tenderness and normal in form. There is usually a trifling cough, rapid breathing, and may be the physical signs indicative of slight catarrh, or the respiratory murmur is rough, respiration more blowing than natural. From first to last the child steadily, and if the febrile disturbance be considerable, rapidly loses flesh.

All the special symptoms I have mentioned may vary in severity, and sometimes one, sometimes another, gives a peculiar feature to the case. Now, the fretfulness, or headache, or drowsiness, or sleeplessness leads the practitioner to consider all the other symptoms secondary to some special intra-cranial disease. Now, it is the furred tongue and the deranged intestinal secretions which lead him to the erroneous conclusion that the febrile disturbance, etc., are secondary to the state of the stomach, bowels, or liver. Again, it may be the catarrhal symptoms that predominate, and the disease is supposed to be a bad cold on the chest merely. The child may be ill enough only to keep the house or its room. After three, four, five, or six weeks even, the disease terminates in one of two ways, either some one organ becomes so gravely

affected as to cause death with symptoms unequivocally referrible to that, or the child recovers.

If the child dies we find after death, in addition to the lesion which caused death, grey granulations scattered through the lungs, the liver, the spleen, the kidneys, under the pleuræ and peritoneum, and grey and yellow tubercles in the lymphatic glands, especially the bronchial and mesenteric. Note that I tell you that grey as well as yellow are found in the lymphatic glands. You will find it stated generally in books that grey tubercles are not found in the lymphatic glands. This is indisputably an error. They are common enough in the interior of all the lymphatic glands.

Suppose the child to recover, what was the condition of its organs?—undoubtedly they too were the seat of tubercle. An acute deposit of tubercle is constantly recovered from. It is by no means a necessarily fatal disease; nay, I feel satisfied that recovery from it is very common. The foundation for a belief in the possibility of recovery from an attack of acute tuberculisaton rests mainly on anatomico-pathological ground, or on these superadded to the history of the patient.

After death we every now and then find scattered through the lungs solid nodules, varying in size from a swan-shot to a large pea, and may be even larger. On section we find the centre formed of a hard, tough, almost black airless substance; about the margin of this we may find a number of small grey granulations, each surrounded by black pigment-loaded tissue; and then again, perhaps, outside these transparent grey granulations free from pigment, and evidently deposited during the last illness of the patient. The centre of such a nodule is formed of obsolescent tubercle, the layer around of tubercles becoming obsolescent. The patient has manifestly had three attacks of acute tuberculisaton, from two of which he had recovered, for obsolescence is evidence of recovery, and in the third of which he has died. The bronchial glands, etc., in many cases give equally valuable and trustworthy evidence of recovery from acute tuberculisaton.

With reference to diagnosis in these cases, it rests mainly on the irregular febrile disturbance, *i.e.* the high but variable temperature lasting for some time, and the steady loss of flesh, with the absence of any evidence of local lesion of a

grave kind. The gravity of the case is constantly, even by the experienced practitioner, unappreciated till some one organ is suddenly so severely affected as to place life in the greatest danger.

I will sketch for you a case that occurred long ago in my own practice, and made an impression on me not to be forgotten. The patient was five years of age, and the child of a medical man. The child had recently suffered from an attack of measles, from which it had recovered quickly, but had soon seemed ill again. She lost her appetite, and sat about listlessly, neglecting her toys, not all day, but only playing with them occasionally, and then with little spirit. Her brothers and sisters worried her. She was taken out of doors; was thought by her mother and myself, even at that time, to want routing. Routing did not answer, and she was noticed to flush in the face and be feverish at night. Now I observed her tongue was loaded, and learned that she had been coaxed with tasty, indigestible food; so I said, 'It is all error of diet.' The diet was regulated, but the child did not improve. On going in one day, and seeing the child look brighter, more lively, and its skin cool, I said, 'Jenny is better to-day.' 'Oh no, I assure you,' was the reply; 'she seemed as well yesterday afternoon, and then all last night she was in a burning fever.' I examined the stools; they were fetid, dark, and too solid. 'Yes, it is no wonder that with such a state of secretion the child is feverish at night,' I said: 'A dose of grey powder and jalap will put all right.' But it did not put all right. The excreta were improved; the diet was all that could be desired; and then I began to think, is not the mother making more of the ailment than it deserves? And so one, two, three weeks wore on, when a sudden attack of convulsions occurred, and in three or four days the child was dead from tubercular meningitis. Every organ in the body was found, after death, to be the seat of a recent deposit of grey granulations.

In the active febrile form of acute tuberculisations the febrile symptoms are more decided from the outset, and the patient at an early period is confined to bed. In the third or adynamic form of acute tuberculisations, the illness begins somewhat suddenly, after at least a trifling sense of malaise

of a few days' duration. The symptoms are chilliness, hot skin, frequent pulse, moist furred tongue, headache, loss of appetite, confined bowels, vomiting, considerable sense of weakness, great unwillingness to be disturbed, with irritability of temper. After a week or ten days, the mind wanders occasionally. The bowels are usually confined, and the abdomen flat or concave. Now and then, however, the stools are relaxed, and the belly swollen. The skin continues hot, dry, and harsh; the tongue becomes dry and brown; sordes collect about the teeth; prostration is extreme; and the patient sinks about three or four weeks after the outset of the disease.

LECTURE II

GENTLEMEN,—In my last Lecture I described the symptoms of acute tuberculisation. I have to-day to speak of chronic tuberculisation.

With reference to the general symptoms of the chronic forms of tuberculisation I need now only remark that no line of demarcation can be drawn between them and those of the insidious acute form I described in my previous Lecture; but the symptoms are less severe, sometimes even so trifling as to escape notice, and they are diffused over a longer time. Before considering the evidences which indicate the tuberculisation of special parts, I desire to express what seem to me to be two laws in regard of the etiology of local tuberculisation, viz., that activity of organic life is favourable to tuberculisation, and that afflux of blood, active congestion of an organ or tissue in one who is the subject of tuberculosis, may determine the tuberculisation of that organ or tissue. The truth of these rules is daily manifested, and the practical consequences in reference to prophylaxis and treatment which flow from them are most important.

There is a point of much interest which must here suggest itself to the pathologist who limits his attention to no one period of life, viz., the striking general contrast which exists in these respects between cancer and tubercle. Organs which are yet imperfectly developed, but which are on the way to perfection, and organs which are performing their functions with full vigour, are prone to tubercularise. The periods of growth and of active life are the periods most favourable to the formation of tubercle. Organs which have passed perfection, or which have just ceased to perform their functions, are in the state most prone to cancer. The period of languid organic life, or of incipient decay, local and general, is the period favourable to the development of cancer.

Cutaneous and Subcutaneous Tubercle.—The majority of writers have either failed to notice, or have passed by as of little importance, the subject of cutaneous and subcutaneous deposit of tubercle. But it is of importance to detect tubercle in these situations, less for itself than because tubercle formed here is evidence of a profound constitutional taint. Tubercles in the skin and subcutaneous tissue are often associated with, or followed by, tuberculisation of bone and its consequences.

Cutaneous and subcutaneous tubercles have often been mistaken for syphilitic, and now and then for glandular, swellings. In the skin and cellular tissue beneath I have seen grey and yellow tubercles—crude and softened tubercle.

The first thing observed is a hard, indolent, painless mass, usually in the subcutaneous cellular tissue about the size of a pea; it may be as big as a small nut. The skin over it is natural in colour and temperature. The little tumour is free from adhesions—it can be moved about in the cellular tissue and the skin can be moved over it. After a time the mass adheres to the skin—the skin becomes discoloured, assumes a purplish red tint, and gives way, and the softened tubercle mixed with pus is evacuated. Less commonly the disease begins in the substance of the skin itself. If all the tubercle have softened before the skin gives way, the whole may be at once evacuated and the part healed, but if it have not all softened, or if fresh tubercle be deposited around, then a small aperture remains, from which every now and then a discharge occurs. The margin and floor of such an ulcer is hard in proportion to the amount of tubercle in the skin and subcutaneous tissue. Deep cicatrices commonly follow on the healing of these tubercular ulcers. They are often called scrofulous. Usually there are not more than four or five collections of cutaneous and subcutaneous tubercle present at the same time, now and then there is one only, and now and then there are a dozen or more. In several of these cases I have seen the bones of the hand become diseased (a model taken from a case of this kind is on the table), and in one case some of the cervical vertebræ suffered. It is in early childhood especially that tubercles are formed in the skin and subcutaneous tissue. I have seen them in a child of eight months old, and in a girl of fifteen; they are most common,

I think, between the ages of ten and thirty months. From the evidence they afford of profound diathetic disease, the prognosis in cutaneous and subcutaneous tubercles is always grave.

Tubercles in the Lymphatic Glands.—As activity of functions of a part *disposes to* the formation of tubercle in the same part, and as undue afflux of blood to a part *determines* the formation of tubercle, the lymphatic glands in the child are more often than any other organ¹ the seat of tubercle; for in the child the lymphatic glands are in a state of functional activity, and become, from the least irritation, the seat of active congestion.

For the purpose of facilitating description, we may divide the lymphatic glands into two sets, viz., those which are seated more or less directly under the skin, and those which are seated in the chest and abdomen. But it must not be forgotten that, in reference to the local state of the glands, to the predisposing cause, and to the essential points of the determining cause of the deposit or formation of tubercle, the two sets of glands are identical.

In order to give a clear idea of the characters of the subcutaneous glands when the seat of tubercle, I will briefly recapitulate the pathological causes of enlarged glands, and the mode of distinguishing glands enlarged from different pathological conditions from each other.

1st. The most healthy child who has impetigo of the hairy scalp—*i.e.* suppurative inflammation of the hair-follicles—will be certain to have enlargement of the glands to which the lymphatics from the inflamed follicles lead. The enlargement of the glands under these circumstances is probably the consequence of the passage into the gland of some irritating substance brought by the lymphatics from the inflamed follicle to the gland. The enlargement of the gland is the result of an excess of blood in it, the effusion of serosity, and the exudation of more or less lymph into it. If the source of the irritant, viz., the inflammation of the hair-follicle, be quickly cured, the lymphatic glands quickly resume their normal size; if the cure be long and delayed,

¹ The lymphatic glands are one organ when viewed with reference to their susceptibility to diseased actions.

then the lymphatic gland remains permanently enlarged; if the primary mischief, the scalp disease, be severe, and the disease neglected, then suppuration of the lymphatic gland may be the consequence. If several glands be secondarily affected, then the glands nearest to the source of the irritant are first and most severely inflamed. I have illustrated what I desire to impress on you by reference to what takes place in suppurative inflammation of the hair-follicles; but the same consequence follows in the lymphatic glands whenever the *substance* of the true skin is inflamed. Primary inflammation of a lymphatic gland is in a child a rare disease; the cases I have seen might be counted by units. Inflammation of lymphatic glands secondary to distant mischief is one of the most common of diseases; the cases I have seen might be counted by thousands. vv

As to the diagnosis of glands enlarged from this cause:—

(a) The enlargement is limited to one set of glands; for example, in the case I have supposed, impetigo of the scalp, to the glands of the neck, and often to those of one part of the neck, as to those behind the sterno-cleido-mastoideus when the occiput alone is the seat of the impetigo, or to those under the symphysis menti, when the impetigo is limited to the chin.

(b) In the earlier stage and in the severer form, the glands are soft and tender, and their outline not very sharply defined.

(c) The source of irritation may be found at the spot from whence the lymphatics leading to the gland arise.

(d) In the chronic stage, the nature of the enlargement is to be inferred, and only inferred, from the small number affected, their small size, their hardness, their moderate movability, their indolence, and duration without alteration, and now and then by the history. Lymphatic glands chronically enlarged, from the pressure of unabsorbed lymph in their substance, rarely, if ever, inflame again, and consequently manifest no tendency to suppuration.

2nd. I have spoken of what takes place when a healthy child is the subject of inflammation, and especially suppurative inflammation, of the true cutis; but if the child, instead of being healthy, be strumous, then an amount of inflammation of the skin which would cause only the most trifling

active congestion of the lymphatic glands to which the lymphatics of the inflamed part lead, would cause very great enlargement of the glands, and even suppurative inflammation.

But in the strumous child the lymphatic glands are generally more perceptible to touch, somewhat larger than are those of the healthy child. At the same time they are not hard, nor very sharply defined, nor particularly movable, as when the seat of albuminoid infiltration. The whole of the glands are pretty equally affected. The enlargement is not limited to one set, but those of the neck, axillæ, groin, etc., are all in the condition described.

3rd. When the glands are the seat of albuminoid disease, they are very hard, sharply defined, and movable. At the same time, though larger than natural, they never attain a really large size, are singularly indisposed to inflammation, and especially indisposed to suppurative inflammation. All the lymphatic glands are affected, and, as a rule, pretty equally. In the neck, axillæ, and groins we feel under the skin deep and superficial glands, like shots and peas.

4th. For the so-called hypertrophy of the lymphatic glands in leucocythemia the blood affords the diagnostic sign.

5th. The lymphatic glands of a child the subject of tuberculosis are not more perceptible to the touch (unless they themselves be the seat of some localised disease), than are the glands of a healthy child. But children the subject of tuberculosis are more disposed than are healthy, though less so than rickety children, to suffer from albuminoid infiltration of the lymphatic glands. So that a child being the subject of tuberculosis, we may find its lymphatic glands hard, movable, and presenting the characters I just now described as evidencing the existence of this lesion.

Uninflamed lymphatic glands in which crude tubercles are present are large, hard, irregular in form, oval or round, but usually more or less uneven or nodular on the surface. It is rare for a single gland only to be diseased, usually several of the same set of glands suffer, and very commonly the corresponding set of glands on the two sides of the body; thus the chain of glands down the neck on both sides, or the

glands in both groins. The lymphatic glands of the neck most commonly suffer, then those of the axillæ, and then those of the groins. While the glands on the two sides so commonly suffer at the same time, it is rare for them to suffer in an equal degree. There is no symmetry in the lesions, and in the same set or chain of glands we find some much more extensively affected than others, and supposing it to be the glands of the neck which are so diseased, there is no order in the degree of disease that is to say, one or more glands in the uppermost part of the chain may be greatly diseased, then several scarcely at all, and then near the lower part of the chain one or more may be greatly diseased.

Sometimes there is a large mass, evidently made up of several enlarged glands, intimately united by condensed and thickened cellular tissue. We can only make out an imperfect division between the separate glands. The skin over the glands, while the tubercle is crude and the glands uninfamed, *i.e.* in the state in which I have been describing them, is colourless, and not adherent to the mass. The part is free from pain and tenderness. In this stage of tuberculisation of the exterior lymphatic glands, the diagnosis rests practically on their large size, and on the length of time they remain unchanged. I was about also to say, 'and on their chronic development,' but in the lymphatic glands, as elsewhere, the formation of tubercle is favoured, not only by activity of the functions of the part, but also by active congestion; and, as a consequence of this, when a child who is the subject of tuberculosis has local disease of the skin or mucous membrane of the mouth or throat, of a nature to cause enlargement of the lymphatic glands to which the lymphatics from the inflamed part lead, that child is liable to have tubercle form in the glands which, in a healthy child under the conditions specified, would merely have serum and lymph exuded into them. So that whenever the lymphatic glands are greatly enlarged, even though it be acutely, and the primary mischief being cured, the lymphatic glands still continue unchanged in size, we may feel very sure, after some time has elapsed, that the glands have tubercularised.

Again, whenever a lymphatic gland has been enlarged for some time, and then spontaneously inflames, the skin over it becoming red and adherent, we may be confident that the cause of the inflammation is the presence of tubercle in the gland.

Wherever the characters I have mentioned are present, there can be no doubt about the nature of the disease; but now and then cases are met with in which the diagnosis is impossible. In these cases the amount of tubercle is very small, so that the gland is scarcely larger than in health; then the gland will be smooth on its surface, movable, indolent, hard,—in fact, it will resemble a gland the seat of albuminoid infiltration, or a gland that was once in a state of active congestion, and the seat of exudation of lymph from distant local irritation.

In a doubtful case you attach much importance to the hereditary predisposition of the patient, and to the general characters of tuberculosis which may be present. The swellings in the neck of the girl who was here at my last lecture presented most unmistakable characters of being lymphatic glands the seat of tubercle; but had they been of more doubtful nature, the aspect of the girl, viz., that of strongly marked tuberculosis, would have given immense importance to the few characters of local deposit which might have been present.

Let me, before concluding this subject, recall to your minds the most common seats of primary inflammation leading to enlargement of superficial glands: Active congestion, etc., of the glands under the rami of the jaw and symphysis menti is the consequence of inflammation of the true skin of the chin, lips, gums, and mucous membrane of the gums and mouth generally; early lancing the gums would save many a strumous child from sub-ramal glandular abscesses. Active congestion, etc., of the glands in front of the ear is the consequence of palpebral abscess—so-called styne—or of infiltration of the cutis of forehead or anterior part of scalp; active congestion, etc., of the glands behind the angle of the jaw, to inflammation of the tonsils or parts in their vicinity; active congestion, etc., of the glands directly under the ear, to disease of the ear; active congestion, etc., of the glands down

the neck anterior to the sterno-mastoid, to disease of the pharynx and larynx and front of the scalp; active congestion, etc., of the glands posterior to the sterno-mastoid, to disease of the occipital region of the scalp. With reference to the glands of the groin, I need only remind you that they are not uncommonly in a state of active congestion in the child in consequence of inflammation of the prepuce and glans penis in the male, and of the orifice of the vagina in the female.

There are some great facts I desire by all this to impress on the mind of the youngest of my hearers, viz.:—

1. That primary inflammation of the lymphatic glands is a very rare disease, even in scrofulous children.

2. That secondary inflammation of the superficial lymphatic glands is a very common disease.

3. That a superficial lymphatic gland being in a state of active congestion, by seeking out and treating the primary lesion, we can in all but every case prevent the occurrence of suppuration of the gland.

4. That when a superficial lymphatic gland inflames and suppurates without there being inflammation of the tissue from which the lymphatics to the gland lead, in almost all, if not in all cases, the gland so inflaming is the seat of the deposit of tubercle.

5. That the lymphatic glands of scrofulous children, and glands which contain tubercle inflame and suppurate from an amount of irritation which would merely lead to moderate active congestion of the glands in a child of healthy constitution.

Let us now consider the symptoms which indicate that the internal lymphatic glands are the seat of tubercle.

There are only two sets of internal glands which need occupy us, viz., the bronchial and the mesenteric. The symptoms which indicate that these glands are the seat of tubercle may be grouped under three heads:—

- 1st. The general symptoms which indicate that the patient is the subject of tuberculosis. The symptoms are those I described to you in my first Lecture.

- 2nd. The symptoms which result from the direct interference with the special functions of the glands affected,

consequent on the damage of their texture resulting from the presence of the tubercle in them, or partly from that and partly from the damage inflicted on the glands by the active congestion, effusion of serosity, and exudation of lymph, which preceded and accompany the formation of the tubercle in them.

Now, as the lymphatic glands in the bronchial and mesenteric groups are numerous, and as, although when one gland of the set is the seat of tubercle, many others are commonly the seat of the same product, yet we find that many of each set so commonly escape, that enough remain undamaged to perform the necessary functions of these glands, and that the symptoms derivable from this source, in the present state of our knowledge, are, for the purpose of diagnosis, altogether insignificant.

3rd. The local signs and the local symptoms, connected directly with the presence of local lesion.

It is not many years since every child whose abdomen was larger than natural, or was supposed to be larger than natural, was commonly said to be the subject of mesenteric disease. A large abdomen at once called to mind disease of the mesenteric glands, and disease of the mesenteric glands signified tubercular deposit in the mesenteric glands. But, so far from tubercular disease of the mesenteric glands being the ordinary cause of a large abdomen in the child, it is very rarely the cause of it. It will be well for my younger hearers to recall to their minds the fact, that children under three years of age rarely have large deposits of tubercle in their mesenteric glands, while they very often have an exceedingly large abdomen.

In reference to the mesenteric glands, the only positive evidence that they are the seat of a deposit of tubercle is the detection by the hand of the large glands. When the mesenteric glands are the seat of a large deposit of tubercle they form a hard, nodular tumour about the umbilicus, which may be perceptible to touch. Sometimes the tumour is fixed, unable to be moved from side to side or from above downwards; sometimes a part of it, some nodules, are movable; sometimes the whole can be moved a little.

To what other conditions can such a tumour be due?

1. Fæcal accumulations.

These are to be distinguished from tubercular mesenteric glands,

(a) By their seat. They usually occupy the transverse and descending colon.

(b) By their shape. They are usually long, their long axis being in the direction of the long axis of the bowel.

(c) By their odour. Retained fæces always, I believe, obtain a peculiar odour, and are accompanied by the frequent expulsion of flatus having the same stale stench.

(d) By their colour and consistence. The fæces that are passed have a peculiar appearance. They are pale, somewhat lumpy, and brittle, with a granular fracture.

But a knowledge of these points only guides to the idea that the tumour is formed of retained fæces. The test of the correctness of the idea is the effect of enemata. Aperient after aperient by the mouth may be given without removing the mass, when a single enema will produce such an effect as to leave no doubt on the mind.

Suppose you desire to use an enema for the purpose of diagnosis in such a case. Strip the child, lie it on the bed, and examine the abdomen, carefully noting the size and position of the tumour or tumours. Then administer yourself, or see well administered, a large enema of soap and water with four ounces of olive oil and a tablespoonful of oil of turpentine. Let the oil and turpentine be mixed with eight ounces of soap water and thrown by a good syringe—not a bladder and pipe—into the bowels, and then as much soap and water be thrown up as the child will bear, the object being thoroughly to distend the bowel, stimulate it to contract and excite peristaltic action far above the point to which your injection reaches.

When an injection is thrown into the bowel a griping pain is soon produced. The child must be quieted. The pain subsides, and then a large quantity—a pint or more—may be injected into the bowel of a child of four years old. Then follows a sense of distention, and you feel that the abdomen is distended. Now the child must be restrained for a few minutes from expelling the injection, the orifice of the rectum being compressed pretty firmly by a cloth. If the enema has

been well given, the child will soon, however, have to be placed on the pan.

The contents of the pan, and especially the altered size, or loss of the tumour, renders the diagnosis certain. Remember, although not directly bearing on our special subject, that *faeces* having once accumulated in quantity sufficient to produce a tumour in the abdomen of a child, are, unless great care be paid to the case, sure to accumulate again; so that if you direct an enema to be given, and do not see the child for some time after, you may find the tumour little changed.

2. Tubercle occasionally forms in quantity in the great omentum, and a tumour so constituted may easily be mistaken for a mass of tubercular mesenteric glands; as a rule it is less nodular, its edge is sharper, and it lies more superficially. Sometimes the diagnosis between the two is impossible and the diagnosis is never of practical importance: a trial of skill, an interesting medical puzzle, and that is all.

As to other tumours that may be confounded with tubercular mesenteric glands, I am not practically acquainted with them. A case of cancer of the kidney was once mistaken for mesenteric disease; such could not have happened in any case of renal disease I have ever seen in a child or adult; and there is a case of disease of the pancreas on record in a child which *might* have been confounded with mesenteric disease, but was not. But tumour is not always perceptible. The tubercle in the glands may be small in quantity, or the glands affected widely separated, and so no tumour be there; or the state of the intestines may prevent the mass present being detected by touch. However, without there being a nodular mass in the umbilical region, we may sometimes feel by careful manipulation the tubercular glands, the parietes of the abdomen being thin and flaccid. The nodular *mass* is, as a rule, most easily and distinctly felt by direct compression from before backwards of the anterior abdominal wall; the *single* glands by grasping the abdominal wall between the fingers and thumb, or between the fingers of the two hands, and compressing it laterally; in this way I have repeatedly felt glands not larger than a small nut floating or moving with the movements of the mesentery.

As to the less constant signs and symptoms of tubercular mesenteric glands,—the abdomen may be of normal size; it may be, and very often is, retracted; now and then it is much distended. The direct cause of the distension is the accumulation of flatus in the intestines, due either to the generation of excess of flatus, or to the want of power to expel that generated.

Now, either the excessive formation or great accumulation may be, and frequently is, due to that condition of the mucous membrane of the intestine and especially of its glandular structures, which is the cause of the active congestion of the glands, that determines the deposit of the tubercle in the mesenteric glands. So that you will have gathered from what I have been telling you, that a large abdomen is by no means a necessary concomitant of mesenteric disease, and that when a large abdomen does accompany mesenteric disease, it is due not directly to the mesenteric disease, but commonly to the state of intestine which induces the mesenteric disease.

The nodular tumour from mesenteric disease is only to be detected by depressing the parietes, and when the nodular tumour is large, the abdomen is often small. There is another symptom frequently observed when tubercles are present in the mesenteric glands, and depending, like the large abdomen, on the determining cause of the deposit of tubercle; that symptom is diarrhoea. The diarrhoea, like the large abdomen, depends on the state of the mucous membrane of the intestines, loose stool and flatus going together. An uneasy sensation, or pain and tenderness of the abdomen, are both common concomitants of the disease of the abdominal lymphatic glands. These symptoms are due, usually, at any rate, if not always, to inflammation of the serous membrane over the glands—now and then to the state of the small intestines. It is certainly very rare for the glands to be so large as to compress the vena cava and induce œdema of the lower extremities.

A little serosity is not uncommonly present in the peritoneal cavity when the mesenteric glands are filled with tubercle; it is the consequence of the state which causes the tenderness, viz., the inflammation of the serous membrane

covering the glands. We find after death in such cases increased vascularity of the serous membrane at the parts indicated.

When ascites is present, it is due either to compression of the vena portæ, by tubercular glands in the portal fissure, or to peritonitis.

Emaciation is a very common, but by no means a constant result of tubercular disease of the mesenteric glands. When the local disease is very extensive, it is generally present; but then you are to bear in mind that extensive tubercular disease of these glands is never present without tubercles forming in other parts. It is difficult to tell the parts played in producing the emaciation by the various organs damaged. Still, I am disposed to think from what I have seen, that when the great majority of the mesenteric glands are damaged from tubercular or albuminoid infiltration, that emaciation, and even extreme emaciation, is the necessary consequence.

Conjoined with tuberculisation of the mesenteric glands we so often find tuberculisation of the peritoneum, and so many of the symptoms of the latter have been attributed to the former, that now, it seems to me, is the best place for its consideration.

In acute general tuberculisation the peritoneum often partakes of the process, and we find it studded more or less thickly with grey granulations remarkable for their small size and their transparency. No local symptoms indicate their existence. The child suffers from the symptoms which indicate general tuberculisation, but we cannot tell in any special case whether the peritoneum is or is not thus affected. Chronic tuberculisation of the peritoneum is generally accompanied by inflammation of the peritoneum; and tuberculisation of the peritoneum, when accompanied by symptoms, is accompanied by the symptoms of chronic peritonitis.

The abdomen is distended and blown, and at the same time the abdominal muscles are tense, so that the abdomen is large and resistant. As the distension is due to the presence of air in the intestines, the abdomen is resonant. As the air in the intestines is partly the result of accumulation, and that accumulation is due to inflammation of the peritoneum, pain (colicky in character) and tenderness are present.

Emaciation, derangement of the digestive functions, constipation, and diarrhoea, now one, now the other, are additional symptoms. As the disease progresses adhesions take place between the coils of intestines, and then a solid, massy sensation is conveyed to the hand on manipulating the abdomen; and as in these cases there is often here and there between the coils of intestines a little fluid, there is often more or less fluctuation.

There are, in many cases, a peculiar expression of face, a worn, anxious look, and a care in movement which are quite peculiar. Occasionally sudden shaking of the abdomen causes more pain than actual pressure. The consequence is that the child in moving briskly, and especially on descending stairs, etc., puts its hands on to its abdomen to stay its vibrations and to afford it some support.

The spleen is frequently at the same time as the peritoneum the seat of tubercles, and can be felt extending a little below the false ribs. The spleen enlarged from the presence of tubercles in its substance never attains the large size of the organ, when the seat of albuminoid infiltration; its edge is less sharp, and adhesions generally unite its peritoneal covering to the parietal peritoneum, and in consequence it is less movable than the albuminoid spleen.

The liver may be enlarged from fatty degeneration, as it is called, of its cells; and we often find scattered through it small grey tubercles, sometimes so transparent as to be easily overlooked.

The Bronchial Glands.—The general symptoms here also are, as in the other cases, those of tuberculosis.

If the tubercles be few they are unaccompanied by local signs. It is only when the glands, one or more, are stuffed with tubercle that we can ascertain their presence. The bronchial glands are situated chiefly about the bifurcation of the trachea, *i.e.* under the first bone of the sternum and a little below it. In the young healthy child there is, from the presence of the remains of the thymus, a little want of resonance at this spot; when the bronchial glands are distended by tubercle the dulness is considerable and passes below the first bone of the sternum and is much greater than natural, laterally. It is only rarely that appreciable dulness is to be

detected between the scapulæ, and after death we see why this should be so, for in the vast majority of cases the enlarged bronchial glands are covered posteriorly by a considerable thickness of lung.

As the bronchial glands accompany the bronchial tubes, it is usual to have on one or the other side some masses of glands extend under the anterior margin of the lungs, and therefore it is common to have a cracked-pot sound on percussing the cartilages of the upper three ribs on one or both sides. The air-containing lung is compressed during percussion between the solid mass of glands behind and the in-driven parietes in front; in this way the air is forced out suddenly from the healthy piece of lung, and the chinking sound is produced.

In the healthy child the breath-sounds have the bronchial character over the first bone of the sternum. When the bronchial glands are stuffed with tubercle, the breathing is more tubular than in health, and preserves that character almost to the base of the heart. It has sometimes the same character between the scapulæ. The large hard glands not unfrequently compress the descending vena cava and produce a permanent venous hum audible over the course of the vessel; they may also compress the pulmonary artery and produce a systolic murmur having its point of greatest intensity at the second left interspace.

The veins of the neck are often greatly distended, the distension being extreme during coughing. Slight œdema of the face and upper extremity is sometimes a consequence of this impediment to the return of blood from the superior vena cava; epistaxis, and even hæmorrhage into the arachnoid, have resulted from the same cause. I have seen hydrothorax from compression of the vena azygos. I never saw dysphagia from compression of the œsophagus by tubercular glands in the child, but I have read of it. The bronchial tubes may be narrowed by the pressure of the glands, and impediment to the entrance of air into the lungs be the consequence; hence will follow still greater atrophy of the lung than is proper to the patient from the tuberculosis. Cough is excited, and from this follows emphysema of the least supported and least compressed parts of the lungs. The

nerves are irritated, continuous spasm may be the result; whispering voice, cough in successions, as in whooping-cough, and even permanent, long, noisy inspirations, such as occur in whooping-cough and in laryngismus stridulus.

Time does not permit me more than very briefly to refer to the terminations of these affections, a subject scarcely within the scope of these lectures. In the superficial glands the grey tubercles, which are very common, either become obsolescent, or they pass into the stage of yellow tubercle, soften, and are evacuated by suppuration. Tubercles in this situation easily calcify.

When tubercles form in the mesenteric glands, death most commonly occurs while they are in the unsoftened yellow stage. Now and then the tubercles do not pass beyond the grey stage, the grey tubercles becoming obsolescent and the patient recovering. Not uncommonly, and that even when the quantity of tubercle in the mesenteric glands is large, it calcifies, and the patient recovers. Sometimes after death softened yellow tubercle is found in some of the glands, and crude yellow tubercle in others.

In the bronchial glands obsolescence and calcification are extremely common terminations of tubercle. In comparatively rare cases softened tubercle is eliminated through an opening into a bronchial tube, and a cavity formed. Calcified tubercle may be eliminated through a communication between the gland and one of the air-passages, usually (as Carswell has figured) near to the bifurcation of the trachea.

Tubercular peritonitis seldom ends favourably. When this does occur, adhesions form between the coils of intestines and the parietal peritoneum; the grey granulations become obsolescent, the masses of tubercle calcify.

ON THE DIFFERENTIAL DIAGNOSIS
OF ACUTE LARYNGITIS
AND THE
FEIGNED LARYNGITIS OF HYSTERICAL WOMEN

Abstract from a Clinical Lecture delivered at University College Hospital

1857

ON THE DIFFERENTIAL DIAGNOSIS OF ACUTE
LARYNGITIS, AND THE FEIGNED LARYNGITIS
OF HYSTERICAL WOMEN¹

No class of cases are more calculated to test our acumen and practical skill than the hysterical, and for this among other reasons, viz., that there is scarcely a disease manifested by symptoms appreciable only by the patient, or by abnormal movements of parts over which the will in health exercises control, that the hysterical female may not simulate. He who has seen much practice, and possesses a little tact, may often detect these cases at a glance; but even he is sometimes deceived for a time, and to the young practitioner they are generally very puzzling. A disease every now and then simulated by hysterical females is acute laryngitis. Now, acute laryngitis is a very dangerous affection, and to confound the hysterical simulacrum with the real disease may be to endanger even the life of your patient; for when acute laryngitis is very severe, we occasionally have to open the trachea, and to open the trachea of a healthy woman is certainly to place her life in some danger. It is with reference to the diagnosis of this hysterical affection that I desire to recall to your minds the case of the woman who lately lay in the last bed in ward No. 3. I directed your attention to her at the time she was in the hospital. Now I am about to explain to you, more fully than I could at the bedside, the grounds of my opinion that the disease under which she laboured was not what it at first sight appeared, and was even by some supposed to be, viz., severe laryngitis.

The woman in question was admitted into the hospital in

¹ *Medical Times and Gazette*, December 12, 1857.

the early part of the afternoon, because she was supposed to be suffering from laryngitis. About 4 P.M. so severe did the symptoms appear that I was sent for; and Mr. Quain being in the hospital, was requested by my excellent assistant, Mr. Morgan, to see the patient, it being a question whether the larynx ought to be opened.

On entering the ward I heard, long before reaching the patient's bed, around which some of you had assembled, the loudest tracheal breathing. The patient, a stout, anæmic-looking woman, was lying with her head thrown back over the pillows, against which she was leaning her back, and which had been placed on her entrance so as to support her in a sitting position. Her eyeballs were turned, the right upwards and outwards, the left upwards and inwards, just as they are directed by those who *desire* to appear *very* bad. There was no lividity of the face; no purple hue of the lips; no evidence of imperfect aëration of the blood. At the same time the pulse was firm, full, and only 84.

These symptoms alone decided Mr. Quain and myself to put aside all idea of opening the air passage for the present at any rate.

Had the woman been faint (as from her position and expression might at first sight have been supposed) the lips, etc., might not have been livid, even though very great impediment had existed to the entrance of the air into the lungs. But her heart was acting so steadily and so strongly as to exclude altogether the notion that the absence of lividity was due to fainting, and therefore, notwithstanding the noisy tracheal breathing and the orthopnœa, from which she had suffered ever since her admission, it was manifest that abundantly sufficient air for the due aëration of the blood entered the lungs; and of course if air entered in sufficient quantity by the glottis no good end could be served by making an opening into her trachea. After settling this point you may remember I laid bare the patient's thorax that I might watch its movements; and what did we see? At each inspiratory effort there was some recession of the supra-clavicular regions, a little deepening of the supra-sternal fossæ, and trifling narrowing of the costal angle, occasioned by the falling inwards of the lower ribs and their cartilages.

These facts indicated that the diaphragm descended more rapidly than the air entered by the larynx, and, consequently, as the inspiratory act was rather slowly performed, they indicated that there was some impediment to the free entrance of the air into the lungs. But as the recession was everywhere trifling, the same facts also indicated most conclusively that the impediment to the entrance of the air was not extreme. So now we had proof that the noise was out of proportion to the structural change in the larynx, supposing *any* such to exist.

When the woman answered my questions she did so in a faint whisper, but although very feeble the voice was not hoarse. Now, had the larynx been the seat of disease sufficient to produce the tracheal breathing, hoarseness would assuredly have been present. Here then was another discrepancy in the symptoms.

On placing the stethoscope over the larynx, the sound produced by the passage of the air through it was rougher and louder than natural, but it was only when the stethoscope was placed near to the angle of the jaw, that is to say, above the larynx, that the very loud tracheal breath-sound was well heard. Here was its point of greatest intensity. Thus then, it was demonstrated that the sound which gave to the case its chief alarming feature was generated in the pharynx, *i.e.* in a part, the muscles of which are in a great measure under the control of the will.

When the larynx is the seat of intense acute inflammation the epiglottis is commonly involved, and then, in many cases, by the aid of a firm tongue depressor, and a good light, you can see the red and erect epiglottis. I thought it right to determine, if I could, its condition in this case, as well as to inspect the pharynx. For the purpose of persuading the patient to exert herself enough to assume a posture convenient for my object, I sprinkled her pretty freely with cold water, and with very good effect. On placing the depressor on her tongue she closed her mouth, and resisted for some seconds with her mouth closed all my efforts to inspect the pharynx,—another proof that there was no great impediment in the larynx to the free passage of the air through it. For had there been such, the desire for air would have been

stronger than the will to resist my efforts. The will was now employed in directing the efforts requisite to resist me, and the consequence was cessation of the voluntary exertion of the muscles of the pharynx, and the free passage of the air through the nares and pharynx into the larynx, and thence into the lungs.

When I did succeed in my endeavours, only a little more redness than natural of the mucous membrane covering the arches of the palate was perceptible. I now learned that the present attack was of two or three days' duration,—that she had one such attack some months before,—that the catamenia had been suppressed for nine months. As no one came with the patient to the hospital, as she spoke in a faint whisper only, and with seeming difficulty; as the symptoms appeared so urgent, and the nature of the disease—viz., acute laryngitis—was supposed to be clear, no good history of the case was obtained on admission.

You will remember my mentioning that I had seen a case very like to this in the early part of the year, which was under the care of two highly educated and able, but rather young, practitioners. In that case, I told you it was supposed that the trachea must be opened; but a dose of opium placed the patient in a sound sleep, during which the breathing was so tranquil, that the idea of severe laryngitis was laid to rest. I determined to place this patient under the influence of opium. She did not fall asleep till she had taken some 60m. of laudanum. When asleep her respirations were in every way tranquil. The next day a mental tonic, so administered as to lead her to express her regret that she had ever entered the hospital, and to cause the nurse even and the patients around, who pitied her apparent sufferings very much, to think me most unkind, and a dose or two of calomel and colocynth completed the cure. You will remember that I learned, on the second day of her residence in the hospital, that this woman was the very same I saw with my two friends in the spring, and to whom I had likened her symptoms on admission. I told you at that time that she had, however, advanced a step in the art of simulating laryngitis since I last saw her; for then there was no recession of the chest walls during inspiration. Now, there was enough recession

to prove that she did really impede the full entrance of the air into her lungs, by voluntarily narrowing some part through which the air has to pass before entering the lungs.

One word more on the treatment. Leeches had been applied to the throat, and calomel and antimony prescribed every three hours, before I saw the woman. Had I known her to be my old friend, I should have tried the physical effects of a cold douche, and the mental influence of a very decided expression of opinion on the symptoms. But while I felt confident that the case was one mainly of hysteria, I did not feel so sure as I should have done had I recognised her, that it might not be an hysterical exaggeration of slight catarrhal inflammation of the mucous membrane of the larynx; so I allowed the medicine she was taking to be continued till I saw the effect of a full dose of opium and a large turpentine enema.

Now, in conclusion, let me sum up my reasons for having regarded the case as one of simulated, and not real laryngitis.

1st. If the sound heard at the bedside had been generated in the larynx, it would have indicated disease of that organ, interfering seriously with the free passage of air through it.

2nd. The expression was that of a person feeling faint, or desiring to look very bad, and not the anxious look of impending suffocation.

3rd. There was no lividity, at the same time that the heart proved by its action that the patient was not fainting.

4th. The small amount of the recession of the more flexible parts of the thoracic walls during inspiration demonstrated absolutely that no great impediment existed to the entrance of the air into the lungs.

5th. The voice, though reduced to a whisper, was not hoarse.

6th. The direct evidence afforded by the stethoscope was in favour of the sound being generated in the pharynx.

7th. The fact that the patient could breathe without difficulty for several seconds with her mouth voluntarily closed, pointed to the absence of any organic impediment to the free passage of the air through the larynx.

One word of warning. I once saw a physician, of deservedly high reputation, mistake a case of tubercular meningitis for hysteria, because some well-marked hysterical phenomena were present; and I shall have occasion at my next lecture to refer to a case of typhoid fever, in which the co-existence of hysteria gave peculiar features to the case.

ON THE INFLUENCE OF PRESSURE
IN THE PRODUCTION AND MODIFICATION OF
PALPABLE VIBRATIONS AND MURMURS
PERCEPTIBLE OVER THE HEART
AND GREAT VESSELS, LARYNX
AND LUNGS

A Clinical Lecture delivered at University College Hospital

1856

ON THE INFLUENCE OF PRESSURE IN THE PRODUCTION AND MODIFICATION OF PALPABLE VIBRATIONS AND MURMURS PERCEPTIBLE OVER THE HEART AND GREAT VESSELS, LARYNX AND LUNGS¹

THERE are three patients still in the hospital, and others have recently left it, in whom I pointed out to you some facts illustrating the influence of pressure in the production, intensification, and modification of certain morbid sounds generated in the region of the heart.

To the case of the lad Exley, suffering from pericarditis, I lately directed your attention at length; I will recall some of the points of the case to your memory: when the stethoscope was placed over any part of the præcordial region between the fourth and seventh costal cartilages, two abnormal sounds were heard with each beat of the heart—one systolic, the other diastolic—I say systolic and diastolic, though they were not completely synchronous in point of time with the heart's systole and diastole. Loud as were the murmurs, especially the systolic, over the heart, they were not audible when the stethoscope was placed at a very little distance from the præcordial region. In character the murmurs were rubbing. I pointed out to you at the bedside, that the case offered us a perfect example of the to-and-fro sound of Dr. Watson. Moreover, the sounds impressed on all who heard them the idea that they were generated very near to the stethoscope. As the parietes of the lad's thorax were tolerably flexible, a moderate force sufficed to bring the two layers of the pericardium more closely into contact, and so the friction resulting from the passage over each other of the roughened visceral and parietal layers of the pericardium was increased, and consequently the murmur generated by the

¹ *Medical Times and Gazette*, March 1, 1856.

friction was intensified. At one time it was supposed (and is so still by some) that murmurs generated in the pericardium could always be intensified by pressure in the young: and also, that when a murmur, audible over the heart, could be intensified by pressure, that absolute proof was obtained that the murmur was generated in the pericardium.

However, we now know that neither of these statements is true; for Dr. Walshe has observed one or more cases in which a pericardial murmur was diminished in loudness by pressure, the free play of the surfaces being impeded by the pressure. On the other hand, the girl Stone, and the boys Perrin and Whiting, afford us indisputable evidence that a murmur may not merely be intensified, but may even be generated at the base of the heart, by pressure. These children are aged respectively 7, 9, and 10; the girl was the subject of chorea, one boy is suffering from enlargement of the spleen, the other from a skin affection; all have well-formed chests, but in all the thoracic walls are very flexible. Moderate force causes recession of the sternum, and of the cartilages of the ribs, and consequently of the parts subjacent.

In reference to these children, and several other cases of a like kind, which I have noted, I wish to impress on you the following facts:—

1. That no murmur was audible when the stethoscope was applied without force to the sternum, or to the cartilages of the ribs.

2. That when a moderate amount of pressure was exerted, through the medium of the stethoscope, on the sternum, over the base of the heart, a systolic murmur was audible.

3. That the loudness of the murmur varied with the degree of the pressure.

4. That pressure on the base of the sternum did not elicit a murmur.

5. That pressure on the sternum, opposite and above the fourth cartilage, and below the first costal cartilage, elicited a murmur.

6. That the healthy sounds of the heart only were to be heard at the second right costal cartilage, whatever the amount of force to which it was subjected.

7. That a moderate degree of pressure on the second left

costal cartilages, or on the second left intercostal space, near the sternum, rendered a murmur audible.

Judging, then, from the situations in which the murmur was producible, viz., over the base of the heart and upwards to the first left interspace; the period in the heart's beats which it occupied, viz., the systole; and the manipulation required to elicit it, viz., pressure, I think there can be no doubt—1. That in these cases the murmur was generated in the pulmonary artery. 2. That the murmur was produced by the passage of the blood through a narrow part of the vessel into a wider part. 3. That the direct force exercised on the thoracic parietes by the stethoscope, was the immediate cause of the local diminution of the calibre of the vessel.

The only observer who has, so far as I know, recorded a case identical with those to which I have just referred, is Dr. Latham. You will find the case in his third Clinical Lecture; it is so admirably told that I must read it to you:—‘A little boy, aged $8\frac{1}{2}$ years, high-spirited and vivacious, but thin and out of health, was brought to me under a suspicion of disease of the heart. Its impulse was not felt beyond the apex, but there it was in excess; yet there was no larger space of dulness than natural in the præcordial region. Upon auscultation, however, this remarkable peculiarity was made out; when the ear or the stethoscope rested gently upon the præcordial region, no unnatural sound whatever was heard; but when either the ear or the stethoscope was applied with such force as to cause the ribs to sink a little below their natural level, then a loud bellows murmur sprang up. The space at which it was heard, and not beyond it, was just so far as the mouth of the stethoscope covered, when it was placed upon the cartilage of the third rib as a centre. This case, which occurred to me,’ continues Dr. Latham, ‘five years ago, has made me watchful ever since, lest haply I might create the murmur I was in search of; and it is no needless caution where the patient is young, and the framework of the chest is yielding. Never, indeed, the chest being not deformed, never but in this single instance have I produced a murmur simulating that of valvular disease. But very often, when over-earnest in what I was about, I have

pressed too heavily on the præcordial region, a sort of jarring sound has reached my ear, and brought with it the suspicion of disease; until setting the heart free from the weight and the restraint which I had inadvertently imposed on it, I have at once lost the sound and the apprehension too, which had arisen from my own awkward manœuvring.'

Since, however, my attention was directed to the subject by hearing the produced murmur in the girl Stone, I have found that I have been able to produce a basic systolic murmur in a large number of healthy children, and I am satisfied that Dr. Latham's caution is highly necessary, viz., that when examining a child you must be careful not to exercise much pressure at the base of the heart, lest you produce a murmur which may cause considerable anxiety in regard to the ultimate fate of your patient. For you will remember that the murmur produced by the weight of your own head on the stethoscope placed over the base of the heart, in the girl Stone, was infinitely louder than you or I ever heard in the same situation from mere anæmia, or spanæmia, as what we lately knew as anæmia is now often called. Had I not detected the cause of the murmur, I should have supposed the child to be the subject of congenital disease of the heart.

In the girl May, lately admitted into ward 3, because the subject of tapeworm, we found that pretty firm pressure on the thoracic walls over the pulmonary artery modified materially the first sound, and although we could not say that a murmur was produced by the pressure, we could not but admit that so long as the pressure was maintained the first sound was not healthy.

This again confirms the accuracy of Dr. Latham's observations, recorded in the quotations I have read to you from his lectures.

I have told you that the murmur produced by pressure over the base of the heart, in the cases in the hospital, originated in the pulmonary artery; I must add that I doubt much whether pressure can be exercised over the base of the heart, powerful enough to diminish the calibre of the aorta, and for these reasons:—1. Because of the relative situation of the aorta and pulmonary artery; 2. Because of

the comparative thickness of its walls. I am inclined, then, to think that systolic basic endocardial murmurs produced or intensified by pressure have their origin in the pulmonary artery. So when in doubt as to whether a systolic basic endocardial murmur is generated in the pulmonary artery or aorta, I have derived some assistance in arriving at a correct conclusion from a consideration of the effect produced on it by pressure.

The little boy Perrin now in ward 4, and whom you can see after the conclusion of the lecture, affords us an example of a very common condition, viz., one in which the patient can exercise pressure on his own pulmonary artery, sufficiently powerful to generate a murmur. So long as this child is inspiring there is no cardiac murmur, but at the termination of expiration there is a loud blowing, systolic murmur; this murmur has its point of greatest intensity at mid-sternum, opposite the third interspace; it is louder at the first left than at the first right intercostal spaces next to the sternum; it is not audible at the apex. These facts show the pulmonary artery to be the seat of the murmur. During forced expiration the anterior surface of the thorax is flattened, the antero-posterior diameter of the thorax is considerably shortened, and so the pulmonary artery is pressed on. This child, as you know, is remarkably anæmic, there is a very loud venous hum in his neck, and it is difficult to place the stethoscope over his carotid artery without compressing it enough to generate a systolic murmur.

Compression of the pulmonary artery by the patient in expiration, or by the physician with his stethoscope, is a common determining cause of the basic systolic murmur so often heard in anæmia. In anæmia, too, the arterial walls appear to resist pressure less strongly than in health, and this is one of the reasons why it is sometimes almost impossible to place the stethoscope on the carotid artery of an anæmic young woman without diminishing its calibre at the spot, and so generating a loud murmur.

But the pulmonary artery is sometimes compressed by the descent of the sternum, during expiration, sufficiently to generate a murmur when the patient is not anæmic; I saw a capital example of this a day or two since, in a

fine healthy-looking young man. My opinion was asked about him, because, among other ailments, he was supposed to have valvular disease of the heart, indicated by a bruit at the base. On first applying my ear to his chest, I heard no murmur; then I heard a loud blowing sound, and then again I lost it for a little while, my stethoscope remaining all the time at the same spot, viz., over the sternum, opposite the fourth costal cartilage. By examining the case more closely, I found that this gentleman was one of those whose thorax is considerably flattened during deep expiration, that the murmur was only heard at the termination of expiration, and that it was audible at the second left, but not at the second right, cartilage; I concluded, therefore, that the murmur was generated in the pulmonary artery when that vessel was compressed by the receding sternum. It may be supposed that in such cases there is really an organic aortic murmur, and that during inspiration it is inaudible, in consequence of the advance of the sternum, and the interposition of a little lung between the aorta and the sternum; but it is not so, for this reason, a murmur so loud as is heard in some of these cases would assuredly be heard at the top of the sternum, and at the aortic or second left cartilage, even during the deepest inspiration.

And now, as to the form of the chest in which basic and systolic murmurs are, or can be, generated by pressure. You may remember that I directed your attention at the early part of the session to two great varieties of the thorax in regard of form, viz., one in which the lateral diameter is considerable, the antero-posterior small, and the other, in which the antero-posterior diameter is long, the lateral comparatively short; the latter variety includes all forms of pigeon or chicken-breast, and among others the rickety thorax. It is in the former of these two varieties of thorax that these pulmonary artery murmurs can be generated by pressure. In the pigeon-breasted thorax, the sternum is placed at a considerable height above the pulmonary artery, and cannot be forced against it by the physician or the patient; and I have demonstrated to you, that emphysematous lung fills up the space between the receding sternal ends of the ribs and the prominent sternum in the rickety

thorax. I have examined a very, very large number of children with rickety thorax, and other forms of pigeon-breast, but could never even modify the cardiac sound; while it is rare to examine a flat flexible thorax, and not be able to produce a basic systolic murmur. The three conditions, then, favouring the production of a murmur by pressure, whether exercised by the physician or patient, over the base of the heart, are:—1. Narrowness of the chest from before backwards. 2. Flexibility of the thoracic parietes. 3. Anæmia. In anæmia, remember, 1st, that the walls of the vessels are more flaccid than in health; 2nd, that the blood is poor in solid constituents, and therefore more readily thrown into sound-generating vibrations than is healthy blood.

Although I have no case to illustrate the fact, I ought not to omit to tell you that, when the abdomen is enormously distended, the relation between the ventricles and the orifice of the pulmonary artery and aorta is altered, and a murmur may be the consequence. A well-marked case of this kind fell under my observation some time since; the patient was the subject of ovarian dropsy; at the base of her heart a loud blowing murmur was heard, whether generated in the pulmonary artery or aorta could not be determined; perhaps a murmur originated in both. She was tapped, and all murmur disappeared. In such cases the apex of the heart is elevated.

The lungs, again, may compress the great arterial trunk in the thorax. The man in ward 4, suffering from emphysema and bronchitis, illustrates my present point. You remember that when that man coughs violently, the radial artery becomes full and hard, and the only movements perceptible in the artery are those communicated to it at each tussive succussion; during the cough the artery seems to be so full that the heart's beats no longer distend and elongate the vessel; it appears filled to its utmost. Now, it is not that the artery is distended because the blood cannot pass freely through the capillaries of the compressed lung, or through the compressed large veins in the thorax. It is not because the flow of the venous current is impeded, that the arterial current is stayed; but it is that the blood is driven during the violent expiration in greater quantity into the artery; it is the *vis à tergo* that is increased; and I say so,

because if you press the artery between your finger and the bone, so as to stop the current at that spot, you still feel the powerful influence exerted by the cough in increasing the quantity of blood in the part of the artery between your finger and the heart.

The degree of tension of the artery during coughing is, in some degree, a measure of the strain on the lung in powerful sudden expiration, and hence of the probability of the occurrence of emphysema.

There is one vessel so powerfully compressed during the violent expiratory efforts of coughing, straining, etc., that I must specially direct your notice to it; I mean the internal jugular vein. This vessel is so situated at its termination as to be compressed, during the efforts in question, between the lung and the clavicle, and the clavicular origin of the sterno-cleido mastoideus. The more bulky the apex of the lung, the more completely is the flow of blood out of the jugular vein prevented. In vesicular emphysema, the apex of the lung is often increased in size; and I have repeatedly pointed out to you the supra-clavicular bulging when the patient was coughing, and also the distension of the jugular that accompanied it. Now, if you call to mind what I told you a few minutes since, respecting the distension of the arteries in consequence of compression of the great arteries in the thorax during coughing, and what I have now told you about the special compression of the jugular, you will not wonder that a vessel now and then gives way in the brain, when the patient is making a violent expiratory effort, either with complete closure of the glottis, as in violent muscular efforts, or with incomplete closure of the glottis, as in coughing.

I am now about to call your attention to another class of phenomena—to an altogether different effect of pressure on tactile and sound-generating vibrations of the heart and great vessels.

The girl Elsworthy, lately a patient in ward 3, because the subject of chorea, some time before admission into the hospital suffered, as so many choreic children do, from acute rheumatism, and, as a consequence of her old attack of rheumatism, has an organic mitral regurgitant murmur,—*i.e.* a systolic murmur, having its point of greatest intensity at

the left apex, or a little above and outside the apex, audible under the angle of the left scapula, and not heard at the top of the sternum. You remember how often I have told you, that to determine the seat of a murmur you must consider—the time of the heart's beat at which it occurs, the point of the præcordial region at which it is the loudest, and the direction in which it is carried. When the hand was placed over the spot where the apex of the heart impinged, provided no pressure was exerted on the part, a thrill was perceptible; this thrill ceased when the hand was firmly pressed on the cardiac region. The woman in an adjacent bed suffered from the same disease; the thrill in her also was systolic, and imperceptible when the hand was firmly pressed on the præcordial region. The same signs were observed in the man Searle, on whose case I lately lectured.

Further, when the stethoscope was gently applied over the apex of the heart, we heard a long, deep-toned, loud systolic murmur, conveying to the mind the idea that it was generated near to the ear. When *firm pressure* was made with the stethoscope on the thoracic parietes, over the point where the apex impinged, the murmur was at once modified, its duration was considerably shortened, it was not so loud as before, its tone was less deep, and, at the same time, it seemed as though generated at a greater distance from the ear than before. All of you appreciated the variations produced in the murmur by pressure.

In the man and the woman the effect of pressure on the mitral regurgitant murmur was the same in kind, and almost in degree, as in the girl.

As to the cause of this modification of the murmur by pressure: it seems probable, at least, that the determining cause of the long murmur at the apex, in such cases as these, is twofold. The blood driven by the contraction of the left ventricle through the partially-closed mitral orifice, throws its valve, and is itself thrown, into sound-generative vibrations; the vibrations of the valve and of the blood are communicated to the walls of the ventricle and by them to the thoracic parietes.

The effect of pressure is to diminish the extent of the vibrations of the ventricular walls; hence they are no longer

appreciable by touch, and so the thrill ceases. Over the directly-induced vibrations of the blood it probably exercises comparatively little power, and they are still conveyed to the ear. Some of the sound-generating vibrations then are continued, because the pressure is not perfect; all the palpable vibrations and some of the sound-generating vibrations are stayed. If you fill a glass with water, place your ear near to it, strike it sharply, and then place your finger on it, you will find a modification in the ringing of the vessel similar in kind to what was induced in these cases by the pressure of the stethoscope—the ringing of the glass is less long continued, less loud, and the ringing sounds more distant than before the finger was applied.

The more superficial and the more lax the coats of the vessel in which vibrations are to be detected by touch, the more readily are they stayed by pressure. This was well illustrated by the ease with which the strong vibrations perceptible by a light touch in the jugular veins of Searle were lost when the finger was more firmly placed on the vein. Friction, tactile vibrations, are usually, as in the woman with pericarditis now in the ward, stronger the more powerful the pressure exerted.

To pass from the circulatory to the respiratory organs: I cause a man with a healthy chest to say 'round,' and place my hand gently on his inferior dorsal thoracic region. I feel a fremitus. I press firmly with my hand, and the thrill or fremitus is scarcely perceptible, or perceptible only at the margin of the hand where the pressure is little or nothing.

Again, I make the man say 'round,' and, by my hand, placed very gently on his thyroid cartilage, I endeavour to appreciate the strength of the vibrations of his larynx. I now have a pillow pressed pretty firmly against his dorsal thoracic region, so as to check, to a considerable extent, when he speaks, the vibrations of this part of his thorax. I bid him say 'round,' placing my finger on his thyroid cartilage as before, and I find its vibrations are less strong than they were before. This seems to demonstrate that the vibrations of the air produced in the larynx are, in persons with healthy chests, strengthened in the larynx itself by the vibrations of the thoracic parietes.

DEFORMITIES OF THE CHEST

1882

BYRONIANS OF THE CHURCH

1862

DEFORMITIES OF THE CHEST¹

UNDER the head of deformities of the chest are included all deviations in shape from the normal chest.

Deviations from the shape of the typical thorax are appreciable by careful physical examination. Of the various methods employed for this purpose, by far the most valuable are inspection and mensuration. Although in some few cases it may be important to determine the exact amount of deformity by mensuration, there are very few deviations in shape or size of the thorax, the degree of which cannot be sufficiently estimated for clinical purposes by the eye and hand, without the aid of any special instruments for measuring.

Deformities of the chest may be due either to abnormality of the parietes, or to disease of internal structures.

DESCRIPTION.—Deviations from the form or size of the typical thorax may be either *general or local*; *i.e.* the abnormality may involve the whole thorax, or a part only.

I. GENERAL DEFORMITIES.—1. *General Diminution*.—The chest may be too small—that is, diminished in all its diameters without being in other respects deformed. Diminution of the thorax simultaneously and uniformly in its antero-posterior and lateral diameters is effected mechanically by an increase in the obliquity of the ribs. The smaller the chest (having regard to the height of the person) the more obliquely are the ribs arranged, and the more acute the angle formed between each of the true ribs (excepting the first) and its cartilage. The intercostal spaces of the true ribs are widened about the junction of the ribs with their cartilages, and at the same time the ribs posteriorly are approximated more closely to each other, the closeness of

¹ Contributed to the *Dictionary of Medicine*, edited by R. Quain, M.D. (1882), and reprinted by kind permission of the publishers, Messrs. Longmans.

the approximation being in proportion to the diminution in the size of the thorax. The vertical diameter of the thorax is lessened by an increase in the height of the arch of the diaphragm. The very oblique position of the false ribs, and the height to which the diaphragm rises into the chest, cause several of the false ribs to lie in contact with the diaphragm, and thus no portion of lung is under these ribs. They are, practically speaking, no longer part of the chest walls.

The costal angles are diminished in proportion to the diminution of the size of the thorax, *i.e.* to the obliquity of the ribs. The obliquity of the ribs also causes the shoulders and the sternal ends of the clavicles to droop, and at the same time to incline forwards; the upper part of the scapula is carried by the shoulder forward, the inferior tilted backward. General and symmetrical diminution in the size of the thorax has one and only one cause, namely, small size of the lungs. Small lungs may be congenital, *i.e.* due to original conformation; or the consequence of atrophic degenerative changes incident to age. In both these cases the lungs are, in relation to the length of the ribs, disproportionately small, and, as a necessary consequence, the relatively too long ribs are arranged more obliquely than they are in a well-formed chest, and the diaphragm is pushed by the abdominal organs higher into the thorax. When the small size of the lungs is due to atrophy, the supra-clavicular fossæ are deepened and the vertical diameter of the chest proportionately diminished. In advanced life the congenitally small lungs are frequently reduced still further in size by the supervention of atrophous emphysema. The congenitally small lungs and the consequently small chest is one of the characteristics of tuberculosis, *i.e.* of that congenital organisation in which tubercle is likely in subsequent periods of life to occur. Atrophous emphysema is especially common in those who have either manifested symptoms of tubercle in their youth, or belong to tubercular families. It is the congenitally small lungs of childhood which are prone to become the seat of tubercle in youth, and the subjects of atrophous emphysema in old age.

2. *General Enlargement.*—The thorax may be too large, increased in all its diameters, without being otherwise

deformed. It is simply bigger than it should be, having regard to the height of the subject.

When the thorax is abnormally large, the ribs, instead of being more obliquely situated than natural, as they are in the small thorax, are placed more horizontally than they are in the normal thorax. The angle formed between each rib and its cartilage is greater than in health; while the intercostal spaces, especially the lower, are widened, and the ribs less closely approximated, the arch of the diaphragm is lessened in depth, and a considerable mass of lung lies under the lower false ribs, between them and the diaphragm. The chest is increased in all its diameters. The shoulders are raised. The costal angles are greater than natural.

Increase in the size of the whole thorax has but one cause, viz., increase in the size of the lungs. Increase in the size of the lungs generally, and pretty uniformly, is the consequence of disease, and of one disease only, viz., large-lunged or hypertrophous emphysema. When the increase in size of the thorax attending large-lunged or hypertrophous emphysema is moderate in degree, the increase in its size is effected by the altered position of the ribs; but when the lung disease is extreme, then a certain amount of the enlargement is caused by pressure on the inside of the chest during the violent expiratory efforts of severe cough.

3. *Irregular General Deformities.*—In the deformities above described the antero-posterior and the lateral diameters retain more or less perfectly their normal proportion—both are increased or both are diminished; in the former case the chest is on the whole more barrel-shaped than natural, but the deviation from the normal form is not considerable. If, however, the chest walls are from any cause unduly soft or unduly rigid, then the actually or relatively soft portions will recede during each inspiratory act, and local deformity of the chest follows. The diameter of the chest at the part where the absolutely or relatively soft portion of the parietes is placed will be diminished. The special deformities of the chest which result are due, therefore, primarily to the state of the parietes, and are not, as those previously described, secondary to conditions of the lungs themselves.

a. *Diminution in the antero-posterior diameter of the*

thorax.—The antero-posterior diameter of the thorax is frequently less than that of the normal thorax, the lateral diameter being proportionately increased. The chest has an oval form—it is flattened from before backwards.

The thorax flattened from before backwards is usually associated with small lungs, but the mechanical cause of the flattened form is the want of full resisting power in the ribs and considerable strength in the cartilages. These conditions of thorax are common in the subjects of tuberculosis.

The flattening of the thorax is increased by all impediments to the free passage of air through the air-tubes. In some children suffering from even slight bronchial catarrh, the flattening of the chest is seen to be increased at each inspiration; and if the impediment to the entrance of the air to the pulmonary tissue be constant or extreme, not only is the flattening increased at each inspiration, but the sternum is also depressed, especially at its lower half, below the level of the costal cartilages, and thus the antero-posterior diameter of the thorax is still further diminished in the median line.

b. Increase in the antero-posterior diameter of the thorax.—In rickets the cartilages of the ribs are very firm, whilst the ribs themselves are softer than natural, and especially so near to their enlarged growing ends—the softest part of the ribs; that is to say, just outside the nodule formed at the spot where cartilage is in the process of growing into bone. The consequence of the extreme softness of the ribs at this part is that at each inspiration the weight of the atmosphere presses inward the softest part of the ribs, while the sternum is borne forward by the firm cartilages. The result is great increase in the antero-posterior diameter of the thorax, and diminution of the lateral diameter at the parts corresponding to the softest part of the ribs. The depression of the softest part of each rib is increased by the want of resilience of the softened structures.

A groove is thus formed in the thoracic walls just posterior to the rickety nodules; and this groove being deepened at each inspiration, the part of the lung adjacent is compressed in place of being expanded during the inspiratory act. At the same time, in consequence of the cartilages and sternum being thrust forward at each inspiration, air enters with undue

force into the lung-tissue subjacent to these parts. The consequence of the excessive expansion of the anterior part of the lung is vesicular emphysema, and the recession during inspiration of the softened and imperfectly resilient and therefore deeply grooved part of the chest wall leads to collapse of the subjacent pulmonary tissue; and, as the effect of these two conditions, the lungs, when the chest is opened, present a vertical groove corresponding to the groove in the chest walls. The antero-posterior diameter of the thorax in rickets is still further increased by the curvation of the spine. The muscles are weak, the child is unable to sit upright, that is to say it is unable, in consequence of the weakness of its muscles, to support the weight of the upper part of its body, the bones of the spine are, in common with the other bones of the body, softened, and the result of the weakness of the muscles and the softness of the vertebræ is the dorsal bow.

When deformity of the chest is the result of undue softness of the chest walls, the position of the solid organs subjacent to the parietes is frequently perceptible to the eye. The liver supports the lower ribs on the right side, the heart supports the ribs and cartilages over it on the left side, and thus these organs cause local prominence of the chest walls without being themselves in any way abnormal.

In the so-called *pigeon-breast*, the antero-posterior diameter of the thorax is increased in the middle line, the lungs are small, the ribs and cartilages are firm, the ribs are placed obliquely and the chest walls are flattened laterally, and the sternum as a consequence is thrust forwards; thus the chest in the pigeon-breasted has a triangular form, the apex of the triangle being the sternum. Impediment to the free entrance of air into the lower lobes of the lungs will favour the production of and increase the deformity. The chests of children who suffer from repeated attacks of bronchitis, but are otherwise healthy, are commonly the subjects of this deformity while there is increased expansion and subsequent enlargement of the upper part of the chest, the lungs being more or less collapsed below and emphysematous above.

c. Transverse anterior constriction of the lower part of the thorax is the consequence of small size of the lung, or of imperfect inspiratory expansion, permanent or frequently

recurring in youth. In these cases the lower ribs are little used in respiration, while below they are borne outwards or supported by the liver, stomach, and spleen, and thus an imperfectly formed transverse depression is produced in the front of the chest on a level with the base of the ensiform cartilage.

The deviations from the type of the normal thorax hitherto described are bilateral, and more or less symmetrical.

II. LOCAL, UNSYMMETRICAL, AND UNILATERAL DEFORMITIES.—1. *Fulness of the supra-clavicular region*.—The supra-clavicular region, corresponding to the portion of the thoracic cavity above the clavicle, may be fuller than natural. The causes of this local bulging are—*a*. Development of adipose and cellular tissue. *b*. Distension of the deep-seated veins. *c*. Large-lunged emphysema, in which disease there is occasionally distension of that part of the cavity of the thorax which lies above the level of the clavicle; the distension is due to pressure on the inside of this part of the thoracic cavity; air being forced violently into this part of the lung during the powerful expiratory effort of cough.

2. *Depression of one supra-clavicular fossa* is caused by any pathological condition of the apex of the lung which produces diminution of its bulk, *e.g.* atrophous emphysema or chronic consolidation of the apex.

3. *Elevation of one shoulder*.—Occupation is a common cause of elevation of one shoulder; thus in clerks, who sit much at the desk, the left shoulder is permanently a little higher than the right, and the upper portion of the spine is slightly curved, the convexity being to the left; so in those who carry heavy weights on one arm, the opposite shoulder is elevated and the spine curved. Whatever necessitates an increase in the capacity of one side of the thorax causes elevation of the shoulder on the same side; thus, considerable dilatation of the heart, fluid in the pericardium, fluid in the pleura, aneurism of the arch of the aorta or of the innominate, all lead to elevation of the shoulder. The shoulder is depressed and carried forward when, from any cause, the whole or upper part of one side of the chest is diminished in size, *e.g.* when the apex of the lung is the seat of chronic pneumonia or chronic phthisis.

4. *Uniform dilatation of one side* of the thorax is due, with one exception, to fluid or air in the pleura; the exception is those rare cases of encephaloid cancer of the lung, in which the formation of cancer is uniformly diffused through the lung-tissue, and in amount so great that the lung 'infiltrated' with cancer very decidedly exceeds in bulk the healthy lung inflated with air by inspiration.

In uniform dilatation of one side of the thorax, the shoulder is raised, the ribs are placed more horizontally than on the healthy side, the intercostal spaces are widened, and the spine slightly curved. When the enlargement is moderate in amount, the increase in capacity is effected by the altered position of the ribs; but when the increase in size is very considerable, then it is due in part to the pressure exercised by the air, fluid, or cancer-loaded lung on the inner side of the chest wall.

5. *Uniform contraction of one side* of the thorax is the consequence of any pathological condition which leads to general and uniform reduction in the size of the lung, *e.g.* cirrhosis of the lung, infiltrated cancer of the lung, chronic tubercular disease of the lung, chronic pneumonia, or the change in the texture of the lung which follows long-continued compression by fluid in the pleura. When the whole of one side of the thorax is reduced in size, the shoulder on that side is depressed, the ribs are placed more obliquely and are more closely approximated than on the opposite side, the intercostal spaces are narrowed, and the spine is curved, often considerably, the concavity of the curve being towards the contracted side.

6. *Lateral curvature of the spine*, instead of being the consequence, may be the cause of deformity of the thorax: the ribs are then approximated on the side and at the part where the concavity of the curvature is placed, while they are separated and the shoulder raised on the side of the convexity.

7. In *angular curvature of the spine* the deformity of the thorax varies with the seat and the extent of the vertebral disease; but, speaking generally, it may be said that in angular curvature of the spine the antero-posterior diameter of the thorax is increased in proportion to the amount of

destruction of the bodies of the vertebræ, and that the ribs are in a corresponding degree approximated.

8. *Extreme depression of the lower part of the sternum* is the consequence of softness of the cartilages of the ribs and impediment to the free passage of the air to the pulmonary tissue. This deformity is never congenital, although the subjects of it often affirm it to be so; it may, however, commence to be formed directly after birth if there be a congenital impediment to the entrance of air into the lungs, *e.g.* atelectasis.

This deformity may be the result of direct pressure. In certain occupations pressure has to be exerted on the lower part of the sternum—thus, some shoemakers use a wooden instrument which has to be kept in its place by pressure against the lower part of the sternum. For direct pressure to produce this deformity it must have been applied in early youth, while the parts are still flexible, and have been exerted frequently over a long period of time.

9. *Congenital deformities* of the thorax are few in number and are due to arrest of development—for example, cleft sternum, and defective formation of one or more ribs or cartilages.

10. *Unsymmetrical diminution in size of a part* of the thorax is produced by any pathological change which reduces the size of the subjacent part of the lung. All chronic inflammatory or congestive conditions of the apex of the lung, whether primary or the consequence or the concomitant of the formation of tubercle, are attended by diminution of the bulk of the part of the lung which is the seat of the lesion. Considerable loss of pulmonary tissue is usually accompanied by falling inwards of the chest wall over the cavity.¹ The formation of a cavity is almost invariably attended by chronic inflammatory condensation, and this increases the local depression of the chest wall. In chronic thickening of the pleura, the chest wall at the part is, by the contraction of the fibrin, drawn inwards, and, the lung subjacent to the thickened pleura being condensed, the chest wall is also forced in during inspiration by atmospheric pressure. Hence,

¹ It is said that a very large air-containing cavity may give rise to local bulging.

after pleurisy limited in extent, it is common to find permanent flattening of the thoracic parietes at the base of the chest on the side affected.

In cancerous infiltration of the lung, limited in extent, the lung-tissue is sometimes so much condensed that the bulk of the cancer and lung are less than that of the healthy lung, and the chest walls as a consequence are flattened over the seat of disease.

11. *Unsymmetrical localised bulging*.—If the ribs are, in relation to the size of the lungs, disproportionately long, and their cartilages soft, then one or more of the cartilages may be knuckled forwards; the cartilage, being compressed between the end of the rib and the sternum, bends in an angle outwards. Although the prominence is trifling, it often causes anxiety to parents and its subject. Local deformity of this kind is occasionally the result of repeated lateral compression of the chest wall in the athletic sports of young boys, *e.g.* cricket.

All the diseases of the chest which are accompanied by general enlargement of both or one side of the chest, when localised, are attended by local bulging; thus, a common cause of abnormal fulness of the lower part of the left side of the thorax, posteriorly, is emphysema of the corresponding part of the lung; a moderate amount of fluid in the pleura is attended by fulness of the lower part of the chest on the same side. In both these cases the ribs are raised into an abnormally horizontal position: the chest walls are not pushed outwards, but the ribs are raised, and the intercostal spaces are to that extent widened. The ribs are put into the position which gives the greatest capacity to the thoracic cavities containing the fluid or the enlarged lung. Local bulging may be produced by aneurism of the arch of the aorta or of the innominate artery; by growths, malignant or other, within the chest; by chronic pleurisy with effusion circumscribed by dense false membrane; by hydatids; or by abscess; and in all these cases the prominence is due to direct pressure on the inner side of the chest wall, and to changes in the chest wall itself.

Hypertrophy and dilatation of the heart, and fluid in the pericardium, are attended by fulness of the præcordial region,

The bulging from these diseases is much greater in the child than in the adult. In these cases a little of the fulness is produced by a more horizontal arrangement of the ribs; but when the prominence of the præcordial region is at all considerable, it is the result of the pressure exercised by the fluid or by the large and powerfully acting heart on the inner surface of the corresponding part of the chest wall.

At the part corresponding to the junction of the first and second bones of the sternum, opposite the cartilage of the second rib, the sternum projects forward. This prominence is called the angle of Ludovicus. Any impediment to the free entrance of air into the lungs may cause depression of the lower part of the sternum; if the ossification of the sternum is not complete at the junction of the first and second bones, undue prominence of this part is the result. Subsequently a formation of bone takes place at this spot and increases the prominence.

ON THE DETERMINING CAUSES
OF
VESICULAR EMPHYSEMA OF THE LUNG

*Read at the Royal Medical and Chirurgical Society of London, June 13, 1857,
and published in vol. xl. of the Society's 'Transactions.'*

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ON THE DETERMINING CAUSES OF VESICULAR EMPHYSEMA OF THE LUNG

SINCE the attention of physicians was fixed on vesicular pulmonary emphysema by Laennec, and the anatomical character of the disease demonstrated by that pathologist, the question as to the determining cause of the dilatation of the air-cells has been a fruitful source of controversy. The question to be decided is one of much practical interest; for as vesicular emphysema is incapable of cure, its prevention is of the highest importance, and to determine its cause is to determine the possibility of its prevention, and to some extent the means to be employed for its prevention.¹

That vesicular emphysema may occur in lungs, the texture of which is otherwise perfectly healthy, is beyond doubt; we see examples of this daily in the acute vesicular emphysema of children. But then it is equally beyond doubt that in certain varieties of vesicular emphysema, *e.g.* that in which one or more vesicles at the apex or margin of the lung are greatly enlarged—the texture of the distended vesicles is constantly abnormal. Mr. Rainey² has rendered it

¹ If the advocates of the theory now generally received, and which has been termed the inspiratory theory, be correct, then powerful expiratory efforts, so far as concerns the production of emphysema, are invariably to be encouraged as essentially preservative, the inspiratory efforts only are to be moderated. 'Die Causalindication besteht . . . in Vermeidung inspiratorischer Anstrengungen, welche die gewaltsame Ausdehnung der Luftzellen steigern können.' (Canstatt's *Specielle Pathologie und Therapie*, Band ii. p. 636.) If the expiratory theory, which I advocate, be correct, then the great object of the physician, so far as concerns the prevention of emphysema, must be to moderate, in many cases at least, the violence of the expiratory efforts. Persons disposed to emphysema or affected to a slight degree with that disease, must avoid occupations which necessitate expiratory efforts with a closed glottis, *e.g.* carrying great weights, powerful pushing or pulling.

² *Medico-Chirurgical Transactions*, vol. xxxi.

highly probable that fatty degeneration of the tissue of the lung is one of the anatomical conditions on which loss of its elasticity and contractility depend. While my own observations have led me to the conclusion that the most frequent anatomical change of the lung having this result is fibrous degeneration, the consequence of the exudation of that variety of lymph which escapes from the capillaries where they are the seat of slight but long-continued congestion, in a person of tolerably healthy constitution. So common is the exudation of this variety of lymph in the congestions of old persons, that the changes of tissue which result from its presence are to be reckoned among the degenerations incident to old age. The loss of elasticity and contractility and permanent dilatation of the part which is its seat, if subjected to a distending force, is well illustrated by a reference to the large size so often attained by varicose veins when their walls are the seat of this degeneration.

The contractile power of the lung being impaired, it is evident that every single forcible distension of the air-cells must be followed by a certain degree of permanent dilatation; that an amount of expansion of the cells which would be instantly removed by the normal contractile power of the lung-tissue, must, if that contractile power be seriously diminished, require only to be frequently repeated to lead to extreme vesicular emphysema. The air-cells, whether their walls be or be not healthy, can be distended, so far as we know, by one power only, viz., the pressure of air on their inner surface, but then the air may be driven against their walls, both during inspiration and during expiration. Hence two theories of the determining cause of emphysema of the lung, termed, respectively, the inspiratory and the expiratory theory.

The inspiratory theory.—Atmospheric pressure may be brought to bear with undue force on the inner surface of the air-cells by the enlargement of the capacity of the thorax during inspiration being disproportionate to the capacity of the air-cells. Some portions of the lungs, being from collapse or other cause diminished in volume and incapable of dilatation, then as the lungs are kept in contact with the thoracic parietes during inspiration by the pressure of the atmosphere

expanding the air-cells, and so increasing the bulk of the organs, the obliteration and diminution in size of one or more air-cells necessitates (*supposing* the enlargement of the capacity of the thorax in inspiration to continue the same as with healthy lungs) abnormal expansion of the unobliterated air-cells.

This theory [of the determining cause of emphysema] is essentially that of Williams, Elliotson, Watson, Hasse, Rokitansky, and others. It was moulded into its present exact form by Dr. William Gairdner, and strongly supported by him in a series of very able papers published in 1850. Dr. Gairdner maintains, that the inspiratory theory admits of universal application, and that no other reasonable explanation can be offered of the mode in which the dilatation of the air-cells is effected. The views of Dr. Gairdner on this point have been adopted almost without question by the latest writers on the subject, *e.g.* Rilliet and Barthez (*Maladies des Enfants*, p. 601), Sieveking (Jones and Sieveking's *Pathological Anatomy*, p. 411).

Nor do I deny that in some cases a certain amount of dilatation of the air-cells is determined by inspiration.

The expiratory theory.—The object of this paper is to show that by far the most common and efficient cause of vesicular emphysema of the lung is the pressure of the air contained in the lung brought to bear on the inner surface of the air-cells by the expiratory efforts.

The expiratory theory I am about to advance is altogether different from the erroneously termed expiratory theory of Laennec.¹

Powerful expiration is, it seems to me, infinitely the most

¹ Laennec's is in reality an inspiratory theory. Starting with the assertion that the inspiratory force is greater than the expiratory, he states his theory thus, 'L'air, après avoir forcé la résistance que lui opposait la mucosité ou la tuméfaction de la membrane muqueuse bronchique, ne peut la vaincre dans l'expiration, et se trouve emprisonné par un mécanisme analogue à celui de la crosse du fusil à vent. Les inspiration suivantes, ou au moins les plus fortes d'entre elles amenant dans le même lieu une nouvelle quantité d'air, produisent nécessairement la dilatation, des cellules aériennes auxquelles se rend la bronche oblitérée.' (*Traité de l'Auscultation Médiate*, tom. i. p. 302.) As the temperature of the air in the lungs is considerably above that of the external air, its dilatation after entering the air-cells must tend still further, Laennec fancied, to over-distend the air-cells.

frequent determining cause of acute vesicular emphysema and of the chronic vesicular emphysema which accompanies chronic bronchitis. It is probably the constant determining cause of the vesicular emphysema which supervenes on chronic congestion of the lungs and bronchial tubes, and on diseased heart, and of the atrophous emphysema of the aged. In like manner, expiration appears to be the invariable determining cause of vesicular emphysema whenever it is considerable in degree and general, or occupies chiefly or only the apex and border of the lung, and whenever the dilatation of one or more vesicles is extreme.

It has been asserted that during expiration every part of the lung is equally compressed, and therefore, that the cells of no one part can be over-distended. Dr. Gairdner observes: 'But the most serious objection to the expiratory theory is that the expiratory act is mechanically incapable of producing distension of the lung or of any part of it. The act of expiration tends entirely towards emptying the air-vesicles by the uniform pressure of the external parietes of the thorax upon the whole pulmonary surface; and even when the air-vesicles are maintained at their maximum or normal state of fulness by a closed glottis, any further distension of them is as much out of the question as would be the further distension of a bladder blown up and tied at the neck by hydrostatic or equalised pressure applied to its entire external surface. The air-vesicles can sustain no distending pressure from the column of air within the tubes, as that air only becomes compressed in virtue of a force which opposes exactly as much resistance without as it creates pressure within. It is singular that a theory so radically unsound and so devoid of proof should have been allowed to maintain a place in medical literature.' (*Monthly Journal of Medical Science*, vol. xiii. p. 10.)

But is it true that during expiration every part of the lung is equally compressed? Is it true that every part of the thoracic walls are during powerful expiratory efforts equally unyielding?

We have only to watch a person whose chest is exposed during a fit of coughing to see what a consideration of the anatomical constitution of the thoracic parietes would à

priori lead one to assert, viz., that the answer to both these questions must be in the negative.

Before coughing a person makes a deep inspiration, *i.e.* he distends as far as possible the air-cells, he then closes the glottis and compresses forcibly the lungs by the thoracic and abdominal parietes, the moment the compression of the lungs has attained a certain point, he opens more or less the glottis, and the air is driven forward by the muscular effort and the elasticity and contraction of the lungs and of the thoracic walls with a force proportionate to the compression to which it was subjected before the opening of the glottis.

Now it is manifest that if there be parts of the thoracic walls which are more yielding, or which during powerful expiratory efforts with a closed glottis contract less than others, that the air immediately before the opening of the glottis will be driven from the compressed portions of the lung into the air-vesicles of the lung situated under such parts of the walls with a force proportionate to the general muscular and other powers in play, to the local want of compression and to the degree of yielding of the walls at those particular spots. That there are such parts, and that they are exactly those which are most frequently the seat of vesicular emphysema, and the sole seats of extreme dilatation of the air-cells, is demonstrable.

Above the first rib the lung is covered only by soft tissues, and certainly, compared with the part of the lung situated below the level of the second rib, it is subjected during ordinary respiratory efforts to very little external compression.

The air-cells of the lung above the first rib being then surrounded by comparatively yielding walls, and being comparatively or absolutely uncompressed during expiration, ought, one would *à priori* suppose, to be distended to the utmost when powerful expiration is made with a closed or imperfectly opened glottis; the air being necessarily driven from the compressed to the comparatively uncompressed parts of the lungs. And that during violent expiration with a more or less closed glottis, the air is actually driven into the lung-vesicles of the apex with power enough to distend them to the utmost, is demonstrated by the supra-clavicular

bulging which may be seen during a fit of coughing; and the hand has only to be placed upon the same part to prove that the lung-tissue subjacent is, during strong expiration, distended by a considerable force. Percussion proves that the bulging to which I refer is pulmonary. If the apex of the lung be the seat of emphysema, then the supra-clavicular bulging during violent expiration is extreme, and percussion on the bulging part elicits an almost tympanitic sound.¹

The cells of the anterior margin of the lung are, like those of the apex, forcibly distended when violent expiratory efforts are made with the glottis closed, or imperfectly opened. The expiration of coughing is performed chiefly by the abdominal muscles forcing up the diaphragm, the ribs compressing the lungs laterally, and the sternum and the cartilages of the ribs anteriorly. But the cartilages are to some degree yielding, and the anterior margin of the lungs is to a considerable extent protected from direct pressure by the heart and great vessels; the consequence is, that the air is driven during violent expiration from the parts most compressed (*i.e.* the central, basic, and lateral parts) into the air-cells of the anterior margin, as well as into the air-cells of the apex.

Again, the margin of the base of the lung, the part of the lung in the vicinity of the root of the organ below the entrance of the bronchus, and the little ridge of lung which lies behind the trachea on the right side forming the posterior margin of what may be termed the tracheal groove on the lung, are in a like manner imperfectly supported and comparatively uncompressed during violent expiration. The base, too, of the left lung generally is less firmly supported than is the corresponding part of the right lung—the liver being a more unyielding organ than the stomach, and

¹ The force which is exerted on the walls of the air-cells when the glottis is closed and powerful expiratory efforts made is also shown by the fact that during the expulsive efforts in parturition one or more air-cells occasionally give way, and the air finds its way through the posterior mediastinum into the cellular tissue of the neck and face. A case of this kind fell under my observation when a student. The woman, after a violent detrusive effort, in which necessarily the glottis was closed and the muscles of expiration called into action, suddenly exclaimed, 'I am unable to see.' Her face and neck were, I now found, the seat of subcutaneous emphysema.

compressing the base of the right lung more uniformly than the spleen and stomach do that of the left lung. The consequence of this want of support and of compression of the parts of the lung last enumerated is, that they, like the apex and the anterior margin, are the chosen seats of emphysema;¹ the air contained in the parts of the lung most powerfully compressed during expiration with a closed glottis, being forced in undue quantity into such parts of the lung as are less compressed and less perfectly supported.

It is manifest that what has been above advanced, as to the effect on the air-cells of powerful expiratory efforts made when the glottis is partially closed, is equally true of the same efforts when made with one or more bronchial tubes partially closed. Dilatation of some of the air-cells beyond the tubes must ensue, if those cells be not uniformly compressed during inspiration.

While, therefore, collapse of the lung is the consequence of complete occlusion of a bronchial tube, dilatation of the air-cells may be the result of imperfect occlusion.

The objection to the theory of Laennec, urged by Louis, viz., that the ordinary seat of catarrh is the base of the lung posteriorly, that of emphysema the apex and anterior margin, is unanswerable. The objection of Dr. Gairdner to the same theory, viz., that impermeability of a bronchial tube is followed by collapse of the pulmonary tissue from which it leads, is equally fatal to it. But then the conclusion of Louis from his facts, viz., that there is no relation between catarrh and vesicular emphysema by no means necessarily follows. The relation between the two is, that catarrh excites frequently repeated violent expiratory efforts, with a more or less perfectly closed glottis, and therefore is one of the exciting causes of repeated over-distension of these air-cells, which during these efforts are the least supported and compressed. Catarrh, then, of the base of the lung posteriorly, may be the exciting cause of vesicular emphysema of the apex and anterior margin, and Louis's conclusion as to the

¹ Louis found the lower lobe of the left lung twice as often the seat of emphysema as the corresponding lobe of the right lung; while the apex of the right lung was more frequently affected with emphysema than the apex of the left lung.

necessary want of relation between catarrh and vesicular emphysema, because they affect different parts of the lung, is manifestly illogical.

Dr. Gairdner's conclusion, viz., that obstruction of a bronchus, because it is sometimes followed by collapse of the air-cells, cannot be a determining cause of rarefaction of the part of the lung from which it leads, is equally untenable. For incomplete occlusion of the bronchi may have, and in some cases I believe does have, a result the reverse of perfect occlusion, and in catarrh and bronchitis imperfect occlusion of the tubes is the rule, perfect occlusion the exception.

The notes of three cases will suffice to illustrate the points dwelt on in this paper—the facts are so common and so patent to every observer, that it would be useless to give more.

Mrs. L., æt. 63, tall, thin; suffered from winter cough; was 'asthmatical on exertion'; the chest was small in circumference, but long; the intercostal spaces were wide, and fell in during ordinary inspiration; there was some recession of the supra-clavicular regions during inspiration. On coughing there was great bulging of the supra-clavicular regions, especially the right. On percussion, the bulging part gave out an almost tympanitic sound. Two days after the foregoing observation the lady died suddenly of fatty degeneration of the heart. The apices of the lungs were the seat of extreme local emphysema.

H. C., æt. 46, a patient under my care in University College Hospital, suffering from dry pleurisy and emphysema. The subjoined note was taken a day or two after admission. The cough is more troublesome, and there is a large amount of sonorous râle, audible during expiration, over both sides of the chest anteriorly. Below the prominence opposite the second costal cartilage the sternum recedes during each inspiration; in the act of coughing it advances with each expiratory succussion; there is marked depression of the supra-clavicular regions during each inspiration, and marked bulging during each expiration. The infra-clavicular regions also advance during the expiration of coughing. The expiration of coughing is chiefly effected by the abdominal muscles, and these are called into play even as low as the pubes.

Catherine C., æt. 1 year 9 months, was admitted under my

care into the Hospital for Sick Children suffering from croup. The following note was taken in the morning as she died in the evening.

Respirations 36, noisy; inspiratory and expiratory sound, as heard by the bedside, husky, hoarse. Expiration much longer than inspiration; cough frequent, hoarse, croupy, singing. During inspiration there is recession of the supra-clavicular, infra-clavicular, and supra-sternal regions of the sternum and costal cartilages below the second, and of all the intercostal spaces. During expiration there is bulging of the supra-clavicular region, and prominence of the infra-clavicular and upper sternal regions. No physical signs of bronchitis. The child died the same evening; and on examination of the body, there was found, in addition to the false membrane in the larynx and trachea, vesicular emphysema of the anterior margin of both lungs, especially strongly marked in the parts overlapping the heart and great vessels, and at the apices. There was scarcely a trace of collapse of any part of the right lung, but several small patches of collapsed tissue in the inferior lobe of the left lung. The emphysema was as great of the right as of the left lung.

During the cough, which takes place in a fit of whooping-cough, the same supra-clavicular bulging, advance of the sternum, etc., as were observed in the above case, are well seen, as are during the whoop, the recession of the supra-clavicular region, of supra-sternal region, etc.

I may sum up the position I have taken in regard of the question as to the determining cause of vesicular emphysema of the lung, thus:

1. The lung during expiration is compressed at different parts with different degrees of force.

2. The parietes of the thorax, in consequence of their anatomical constitution, yield to the same force at different parts with various degrees of facility.

3. The chosen seats of emphysema are exactly those parts of the lung which are the least compressed during expiration, and the least affected by inspiration, and which are situated under those portions of the thoracic parietes that give way the most readily before pressure.

4. The power possessed by man of dilating the apex of the lung by inspiratory effort is altogether insufficient to distend the air-cells to the degree to which they are often found to be distended in local emphysema of the apex.

5. That the air-cells of the apex can be distended very considerably and with force during expiration is matter of demonstration; for in violent coughing we may see the elevation of the supra-clavicular region, the advance of the upper part of the sternum, and of the intercostal spaces generally during each expiratory succussion, the bulging parts being resonant on percussion.

6. When an impediment exists to the entrance of the air into the lungs and to its egress from the lungs, as in croup and in whooping-cough, the thoracic walls over the parts of the lungs, which after death are found emphysematous, recede during inspiration and advance during expiration.

7. In the cases last mentioned the more violent the inspiratory efforts the greater the recession of the parts of the parietes referred to, and the more violent the expiratory efforts the greater their advance.

8. When the apex is the seat of calcified or of obsolescent tubercle, lymph is exuded around and into all the tissues of the apex. Some air-cells are obliterated and the elasticity and contractility of the tissues generally is either diminished or lost.

9. Under the circumstances last specified, dilatation of the unobliterated cells, by the air driven into them during violent expiration with a closed glottis, attains its maximum.

10. Under the same conditions in the same situation, the inspiratory force is altogether insufficient to cause dilatation of the air-cells.

11. The atmospheric air moved by the inspiratory effort can exert comparatively little pressure on the inner surface of the air-cells situated at the extreme margin of the base, the root of the lower lobe (*i.e.* that part immediately next the spine and below the primary bronchus) or at the part of the apex situated in the furrow posterior to the trachea on the right side. While violent expiration, being chiefly performed or greatly aided by the abdominal muscles forcing upwards the liver, etc., drives the air (in consequence of the highly arched form of the diaphragm in violent expiration) from the central part of the lung, not only through the bronchi towards the larynx, but also towards the circum-

ference of the lungs, *i.e.* towards those parts which are the least compressed during expiration.

12. In the parts just named dilatation of the air-cells is extremely common, and the dilated cells are often of large size.

13. While complete obstruction of a bronchial tube is followed by collapse of the pulmonary tissue on its distal side, partial closure of a tube has as its consequence dilatation of some of the air-cells of the part of the lung from which it leads, provided that part be unequally supported and compressed during expiration.

14. Vesicular emphysema is the invariable and necessary consequence of whatever impedes the free exit of air from the lungs, and at the same time excites powerful expiratory efforts; because by such efforts the air is driven from the more powerfully compressed parts of the lungs into those parts which are less powerfully compressed and the walls over which yield more or less readily to pressure.

Postscript.—If the view of the determining cause of emphysema advanced in this paper be correct, then in the lower animals subject to pulmonary vesicular emphysema the disease ought to be found to affect especially those parts of the lungs which in them are, during expiratory efforts with a closed glottis, the least compressed, and which are situated under those parts of the thoracic walls that are the least capable of offering resistance to pressure. To ascertain whether it is so or not, I examined, a day or two since, with the assistance of Mr. Henderson, the veterinary surgeon of Great Scotland Yard, five horses, the lungs of which were emphysematous. In four of the five horses the anterior lobes only, and that part of the inferior lobe of the right lung which is protected by the trachea were affected; in the fifth, in addition to these parts, the margin of the inferior lobe.

Now the anterior lobe of the lung in the horse is situated above and over the heart, under the four upper ribs. It is placed, that is to say, in a part of the thorax where it can during expiration be subjected to very little external compression. So the part of the right lung, which in all five

horses was emphysematous, was protected from compression by the trachea. In the lungs which were the most emphysematous there was no evidence of other disease, so far as I could judge, no trace of recent or of old collapse.

The frequency with which horses suffer from emphysema is of course attributable to their being so often compelled to make (in pulling, etc.) powerful expiratory efforts with a closed glottis. One would expect to find all animals (man among the others) engaged in occupations necessitating continued and repeated expiratory efforts with a closed glottis, more subject to pulmonary vesicular emphysema than others of the same class, *e.g.* draught oxen rather than milch cows.

EMPHYSEMA OF THE LUNGS

1871



EMPHYSEMA OF THE LUNGS¹

DEFINITION.—Relative excess of air in a part, or the whole of the lungs.

The relative excess of air may be the result of increase in the quantity of air in the vesicles, of diminution in the solid tissues of the lung, or of the presence of air in lung structures which in health do not contain air.

Pulmonary emphysema may be divided into—

1. Interlobular, extra-vesicular, or extra-alveolar emphysema.

2. Vesicular or alveolar emphysema.

This division, first made by Laennec, has been adopted by all subsequent writers on the subject.

INTERLOBULAR, EXTRA - VESICULAR, OR EXTRA-ALVEOLAR EMPHYSEMA.

DEFINITION.—Air in the connective tissue of the lung.

The connective tissue of the lung is seated, chiefly at least, between the lobules and under the pleura. The air in extra-vesicular emphysema occupies the meshes of this connective tissue.

When air is present in the connective tissue between the lobules it accumulates in small bubbles of tolerably equal size, separated from each other by bands of tissue, so that the surface of the lung looks as if streaked or crossed by rows of small beads. When air is in the sub-pleural tissue it forms air-blebs, sometimes of very large size.

Air may be formed after death in the connective tissues of the lungs by decomposition; it may be generated there during life by gangrene; and it may be extravasated into the same tissue in consequence of rupture of the normally

¹ An article contributed to Reynolds's *System of Medicine*, vol. iii. (1871), and now republished by kind permission of Messrs. Macmillan.

air-containing structures of the lung. When formed by decomposition after death, the gas is usually seated in the inter-lobular tissue; when generated by gangrene, in the sub-pleural tissue; and when extravasated from the air-vesicles, it commonly occupies both situations.

Air extravasated into the connective tissue of the lung occasionally finds its way into the posterior mediastinum, and thence into the subcutaneous tissue of the neck, face, trunk, etc.

Rupture of the normal air-vesicles may be the result of injury inflicted from without, or of the pressure of the air on their inner surface during violent expiratory efforts made when the glottis is closed, *e.g.* during cough and parturient efforts. The distension of the air-vesicles by inspiratory efforts is never great enough to cause their rupture.

Interlobular emphysema is a condition of little importance. When the air finds its way through the connective tissue of the posterior mediastinum into the subcutaneous tissue, the air is quickly absorbed, and in a few days no trace of the emphysema is to be detected.

With the exception of the cases in which the air reaches the subcutaneous tissue, the diagnosis of pulmonary extra-vesicular emphysema is impossible, and even in these cases there are no pulmonary signs or symptoms to indicate the existence of the local lesion.

Should the existence of extra-vesicular emphysema be ascertained, no treatment is needed.

PULMONARY VESICULAR EMPHYSEMA.

DEFINITION.—Increase in the capacity and size of the air-vesicles of the lungs.

Pulmonary vesicular emphysema is a very common, and frequently a grave disease.

Causes of increase in the capacity and size of the air-vesicles.—All forms and degrees of pulmonary vesicular emphysema have their origin either in destruction of the partitions between the air-vesicles, or in over-distension of individual air-vesicles. In the former case, two or more air-vesicles are, by the primary lesion, thrown into one; in the latter, each air-vesicle is, by an over-distending force, increased in capacity and size.

It is improbable that nutritive changes in the tissue of the walls of any hollow viscus ever lead directly to expansion of its cavity. But changes in the walls of a hollow viscus may weaken their resisting power and so favour the expansion of its cavity; and, again, changes in the walls of a hollow viscus may cause a dilatation to be permanent, which otherwise would have been temporary.

Changes in the walls of a hollow viscus, which strengthen their resisting power, may, at the same time, weaken their contractile power. Walls so changed may resist a dilating force longer than healthy walls, but should the dilating force be sufficient to stretch them, the dilation of the cavity they enclose is permanent. The walls are indeed stronger, but then the cavity is more likely to suffer *permanent* dilatation.

The causes of increase in the capacity and size of the air-vesicles of the lungs are then divisible into:—

1. The forces which determine their over-distension;
2. The conditions which favour their over-distension;
3. The conditions which render their over-distension permanent;
4. The lesions of structure by which two or more vesicles are thrown into one.

Although this division should always be kept in view when considering the causes of pulmonary vesicular emphysema, it will be better, in an article such as this, to consider the causes included in the second and fourth divisions incidentally, as occasion arises, when treating of the causes included in the first and third divisions.

Determining causes of over-distention of the air-vesicles.—Pressure of air on the inside of the air-vesicles is the force which directly causes their normal expansion; increase in that pressure is the immediate cause of their over-distension.

Excess of pressure of air on the inside of the pulmonary air-vesicles (of the whole or of a part of the lung) may be brought to bear,

- (a) By excessive expansion of the chest walls.
- (b) By normal expansion of the chest walls, when disseminated portions of the lung are shrunk, and no longer admit air;
- (c) By unequal compression of the lung at the moment

when there is impediment to the free escape of air from its air-containing parts.

(a) In health, inspiration is effected by muscular effort, ordinary expiration chiefly by the elasticity of the thoracic parietes and of the lung textures. The muscular effort of inspiration overcomes the resistance to the entrance of the air into the air-vesicles, offered by the elasticity of the lungs and of the walls of the thorax; the muscular effort ceasing, the elasticity of these parts is sufficient to accomplish the ordinary expiratory act.

The elasticity of the ribs and of their cartilages diminishes considerably as age advances, while in a large number of cases the muscles of inspiration continue as powerful as, and are sometimes more powerful than, in early life. The result of inspiratory expansion of the chest being constantly accomplished by the action of muscles undiminished in power and activity, and of expiratory diminution of the chest being constantly performed incompletely and imperfectly by thoracic parietes the elasticity of which is diminished, is gradual expansion of the chest walls, increased capacity of the chest, and dilatation of the air-vesicles of the lungs. The capacity of the chest not being reduced to its normal size during expiration, the inspiratory effort is made on a chest retaining too much air in the lung-vesicles, and thus, especially if there be repeated and powerful calls on the inspiratory power, as from cough or great muscular effort, the result is considerable over-distension of the air-vesicles.¹

In the same way lessened elasticity of the lungs from age-degeneration, or other cause, without loss of power in the muscles of respiration, leads to increase in the capacity of the thorax, and over-distension of the air-vesicles. The excessive expansion of the thorax, and therefore the dilatation of the air-vesicles in this, as in the last case, is determined by extreme muscular inspiratory action—the necessary result of deficient ordinary expiratory power.²

Another cause of increased expansion of the thorax has

¹ Dr. G. Budd, in a paper on Pulmonary Vesicular Emphysema, published in the *Med. Chir. Soc. Trans.* for 1840, clearly pointed out the part which loss of elasticity of the lungs plays in the production of emphysema.

² See p. 159.

been described by Freund. He says that persons of all ages, from twenty years upwards, the well nourished as much as the withered and decrepit, are liable to a chronic disease of the cartilage of the ribs, which results in their hypertrophy and increased firmness and rigidity, and in diminution of their elasticity. As this increase in the size of the cartilages takes place in all directions, the ribs and sternum are separated from each more than they are in health; the ribs being forced outwards and upwards, and the sternum forward and a little upwards. The capacity of the thorax is thus (Freund says) increased, and the lungs proportionately over-distended. It has been contended by later writers that Freund exaggerated the frequency at least of this affection; that he supposed changes in the cartilages resulting from their stretching to be the primary affection—in fact, that he took the effects of the action of the determining cause for the determining cause itself. For the capacity of the thorax to be increased under the conditions named by Freund, the diaphragm must continue to be at the termination of ordinary expiration on as low a level as in health. Usually, however—and perhaps always when the cartilages lengthen—they bend so as to form an angle, with its concavity upwards about their centre.

(b) It is evident that if disseminated portions of lung are from any pathological condition diminished in size and no longer admit air, and that if, at the same time, the chest walls expand during inspiration to the same amount as in health, the air-admitting vesicles must be over-distended in proportion to the number of cells into which no air enters, and the degree to which the airless vesicles are diminished in size. Thus, in certain cases of bronchitis, disseminated lobular collapse is common. The collapsed lobules are smaller in bulk than are the air-containing lobules—their vesicles admit no air during inspiration. The necessary result is, that if the chest walls expand to the same degree as before the establishment of collapse, and so inspire an equal quantity of air, the capacity of all the air-vesicles still pervious must be increased.¹

(c) If a lung removed from the body be moderately

¹ This point has been excellently well brought out by Dr. W. Gairdner.

inflated, and the bronchus leading to it be tied, and then the substance of the organ be compressed at one part, over-distension of the air-vesicles of the uncompressed part is produced. Should the compressed part be large, and the compression considerable, even rupture of the air-vesicles of the uncompressed part may result. Under the conditions supposed, air is forced from the compressed parts of the lung into the air-admitting structures of the uncompressed parts of the lung.¹

The conditions essential to the over-distension of the air-vesicles here present are:—

- (a) Inflation of the lung.
- (b) Closure of the natural passage for the escape of air from the lung.
- (c) Unequal pressure on the lung.
- (d) Unequal support of different parts of the lung.

During violent cough and great muscular effort these essential conditions are fulfilled:—

(a) Preparatory to cough and to great muscular effort, a deep inspiration is taken, *i.e.* the lungs are inflated.

(b) Then the glottis is closed, *i.e.* the air is prevented from escaping by the natural channel.

(c) Then, by the action of the expiratory muscles, the lungs are strongly compressed, and an examination of the structure of the thoracic walls at once shows that the compression must be unequal.

(d) Examination of the structure of the walls of the chest also shows that the support offered to the lungs by those walls is very different in degree at different parts.²

Again, when blowing a wind instrument the chest is expanded to its utmost, and then the chest walls compress the inflated lungs—the air cannot escape as freely through the instrument as through the open glottis, and the

¹ This expiratory theory was first advanced by Mendelssohn, in a very able paper, 'Der Mechanismus der Respiration und Circulation,' 1845. The writer of this article was unacquainted with Mendelssohn's paper when he advanced the same theory in 1857, and so far as he knows the existence of Mendelssohn's paper was unknown in this country, and rarely if ever referred to abroad till Biemer's article appeared in 1867.

² For details on this point see the author's paper on the determining causes of Pulmonary Vesicular Emphysema, in the *Medico-Chirurg. Soc. Trans.* 1857, and here reprinted—*ante*, p. 135 *et seq.*

mechanical effect is over-distension of the air-cells of the least compressed and least supported parts in proportion to the impediment to the escape of air and the force with which it is attempted to drive the air forward.

The over-distension of the air-cells thus affected will be in proportion to the amount of inflation of the whole lung, to the firmness with which the glottis is closed, or the smallness of the aperture of the wind instrument, or other obstacle to the free escape of air;¹ to the extent, degree, and difference in the force and compression exercised on the several parts of the lung at the same moment; and to the deficiency of support afforded to the less compressed parts by the thoracic parietes. The greater and the more extensive the compression of one part of the lung, and the less the compression of the other, the greater will be the distension of the air-cells in the less compressed parts; and the less the imperfectly compressed parts are supported by the thoracic parietes, the greater will be the distension of their air-vesicles.

It would at first sight appear that the over-distension of the vesicles should be in all cases limited to the less compressed and the less supported parts of the lungs, but on further examination it will be seen that this opinion is erroneous. Thus, if from some change in the walls of the chest or of the air-vesicles, the latter continue over-distended after the force which directly determined their over-distension has ceased to act, or, in other words, if there be permanent dilatation of the air-cells, then the size of the chest and of the lungs is permanently increased.

The portions of lung corresponding to the intercostal spaces are less compressed and less supported just before violent expiration than are the parts immediately under the ribs themselves. Now with every increase in the size of the lungs, or thorax, or both, the relative positions of the lungs and ribs are changed. As the chest enlarges, the ribs assume a more horizontal position, the lower intercostal spaces become wider, and their supporting power by so much diminished.

By these changes in the lungs and in the chest walls their relative positions are being constantly shifted, and fresh portions of the lungs are being constantly brought to correspond to the ribs and to the intercostal spaces, etc., and

¹ Dr. Budd's case.

thus, ultimately, the air-vesicles of the whole lung may be over-distended. But when the air-vesicles of the whole lung are thus over-distended, the dilatation of the vesicles at the apex and margin of the lung is in excess of the dilatation of the vesicles of other parts. Strong expiratory effort, while there is impediment to the free escape of air from a part or whole of the lung, is now admitted to be the most common efficient determining cause of over-distension of the air-vesicles.

Pulmonary vesicular emphysema is very common in horses, and for this reason, viz., that they are constantly making powerful muscular efforts with closed glottis. No one who watches a horse draw a heavy load up a short steep incline on a damp cold day can doubt this. While making the effort, the horse holds its breath, having previously inflated its lungs. No sooner, however, does the animal cease its effort than the glottis is opened, and the air is suddenly expressed from the lungs. The degree to which the air was compressed during the powerful effort (and the consequent strain on the less compressed and less supported part of the lung) may be judged by the distance to which, and the sudden violence with which, the cloud of breath-vapours is seen to be driven forth.

Permanence-securing causes, or, the conditions which render over-distension, or increase in the capacity and size of the air-vesicles of the lungs, permanent.—Whatever destroys the partitions between adjacent air-vesicles, and whatever permanently diminishes the ordinary or habitual respiratory power, must, to a like degree, be a permanence-securing cause of increase in the capacity and size of the air-vesicles. The permanence-securing causes, therefore, are:—

1. Direct injury to the elasticity of the walls of the air-vesicles.

2. Permanent diminution of the power of supporting or compressing the lung, at any one part, during violent expiratory effort.

3. Changes in the structure of the parietes of the thorax, which permanently diminish their elasticity, and therefore their ordinary or habitual expiratory power.

4. Chronic changes in the structure of the lungs, which permanently diminish their elasticity, and therefore their expiratory power.

5. Atrophy of the septa between the air-vesicles of the lungs, by which two or more vesicles are thrown into one.

1. If the forces which expel the air from the air-vesicles, viz., the elasticity of the thoracic parietes, and the elasticity of the walls of the air-vesicles, are at the termination of over-distension of the vesicles in a state of health, then the force determining their over-distension ceasing to act, the air-vesicles return to their natural size; but if, as very rarely happens, the air-vesicles have been very greatly over-distended, or kept for a very long time over-distended, or have been very repeatedly over-distended, then the elasticity of the walls of the air-vesicles may be permanently injured, and the over-distending force ceasing to act, they do not recover their normal dimensions. They are under the circumstances supposed permanently over-distended. The elastic structures of their walls have been directly injured by the over-distending force. So great even may be the force by which their over-distension has been effected, that the partitions between adjacent vesicles may be destroyed, and two or more vesicles thrown into one; or even, as has been previously mentioned, the destruction may have reached further, and the air have been extravasated into the interlobular tissue.

2. The observations of Ziemssen on a case in which there was loss of muscular power in the four upper intercostal spaces, proves that this loss may be a cause of vesicular emphysema. In Ziemssen's case, during violent expiratory effort, these intercostal spaces no longer affording their normal support to the lung, were forced outward so much as to stand above the level of the ribs. When the muscles of either intercostal spaces were stimulated to contract by faradisation, then the bulging during expiratory efforts of that intercostal space ceased, thus proving that want of muscular contraction at any part during expiratory effort is a cause of over-distension of the air-vesicles of the lung at that point; and if the want of support be permanent, then certainly the over-distension will be permanent.

3. The degeneration of the ribs and cartilages incident to age diminish their elasticity, and consequently diminish the expiratory power of the chest walls. If, as was previously pointed out, the inspiratory muscles act perfectly when the expiratory force resulting from the resilience of the ribs and

cartilages is diminished, dilatation of the thorax, over-distension of the air-vesicles, and enlargement of the lungs are determined.

As age-degeneration is a permanent lesion, the loss of elasticity resulting from it is permanent; the dilatation of the thorax, over-distension of the air-vesicles, and the enlargement of the lungs is permanent. Age-degeneration of the ribs and their cartilages is with perfectly acting inspiratory muscles, therefore a permanence-securing cause of large-lunged vesicular emphysema.

The disease of the cartilages of the ribs described by Freund, once established, is permanent, and therefore the over-distension of the air-vesicles, due to the expansion of the chest resulting from it, is also permanent.

4, 5. Whatever changes in the lungs diminish their elasticity, to the same degree render permanent over-distension of the air-vesicles determined by any of the forces previously enumerated. Diminished elasticity of the lung may be the consequence of those changes in texture which result from repeated or long-continued congestion. After a part has been the seat of long-continued or of repeated congestion, it is, if an organ, indurated and toughened; if a tissue, toughened and thickened. If death occur long after the outset of the congestion, then a certain amount of wasting of the original structures is found to have taken place. In some cases, certainly, these changes are due to the formation among the normal anatomical elements of the part, of imperfectly developed connective, fibrous, or fibro-cellular tissue.

All degenerations of texture incident to age are attended by more or less loss of elasticity.¹

The degenerations incident to age,² as they affect the lung, may be divided thus:—

¹ Diminution of elasticity is one of the most marked effects of the changes in nutrition incident to advancing age, *e.g.* of the skin, giving the aged look; of the arteries, causing them to become tortuous or S-shaped, at first when the part in which they are placed is shortened, as in flexion of the limbs, and then permanently; of the inter-vertebral cartilages, of the elastic structures in the sole of the foot, the joints, the bones, etc.

² Those changes of nutrition which are the characteristics of age, and in fact constitute old age, may occur, generally or locally, at an unusually early age. Thus one man grows prematurely old as regards his jaws, another as regards his hair, another as regards the heart, etc.

(a) Atrophy, or waste of all the anatomical constituents of the lung, with general diminution in its size. As the partitions between the air-vesicles atrophy, two or more vesicles are thrown into one. This form of atrophy has been supposed to be preceded by fatty degeneration.

(b) Thickening of the fibrous element of the lung, with more or less waste of some of its anatomical constituents. When the subject of this form of degeneration the size of the lung is often increased, and it may be considerably so.

In this latter form of age-degeneration there is, at the outset at least, no atrophy of the inspiratory muscles; while in the former, the muscles on the outside of the chest are wasted and pale, and the diaphragm is thin, lax, and in folds. In both the ribs and cartilages are the seat of degenerative changes attended by loss of elasticity.

So, also, when the ribs and cartilages lose elasticity from age-degeneration, the lungs rarely preserve their normal elasticity; they too, commonly, like the ribs and cartilages, are suffering from age-degeneration.

The conjunction of diminished elasticity of the lungs and of the parietes, reduces the ordinary or habitually employed expiratory force to a minimum. Now, this being the case, if the muscles of inspiration and of expiration retain their normal power, then frequent cough, habitual straining at stool, moving heavy weights, climbing hills, blowing wind instruments, or other causes of repeated and powerful inspiratory efforts, followed by violent expiratory compression of the inflated lungs, with impediment to the escape of air, will be followed by great and permanent increase in the size of the thorax, and corresponding over-distension of the air-vesicles.

Changes in the lung, attended by loss of elasticity, said to be independent of age and of congestion, have been described by various authors.

M. Villemin thinks that the true anatomical structure of the walls of the air-vesicles is a network of capillary vessels, with a nucleus filling each intercapillary mesh, and elastic fibres on the inside of the vesicles crossing over the capillaries and intercapillary nuclei. 'In pulmonary vesicular emphysema,' M. Villemin says, 'the nuclei in the meshes of the capillary network hypertrophy, compression and atrophy

of the capillaries follow; then the enlarged nuclei undergo fatty degeneration; they fall from their places in the walls of the air-vesicles, destruction of the elastic tissue and of more capillaries occurs; apertures are formed between adjacent vesicles, and finally, two or more vesicles are thrown into one.

'There is then,' M. Villemin says, 'a first stage of emphysema, a true hypertrophy of the elements of the vesicular membrane; from this there naturally results an extension of that membrane and an increase in the capacity of the vesicles.'

It does not, however, necessarily follow, even though M. Villemin's anatomical observations be correct, that there is an increase in the size of the lung, as he supposes, because the walls of the air-vesicles are lengthened, for they might, under such circumstances, be folded on themselves. Moreover, the accuracy of these observations has been doubted. The so-called intercapillary nuclei are said by some observers to be epithelium on the inside of the air-vesicles.

'Changes in the nutrition of the lung,' Freund says, 'necessarily follow on the changed conditions of the respiratory movements due to the lengthening of the rib-cartilages, and these changes are attended by loss of elasticity and the other changes in the walls of the air-vesicles which follow on their continued over-distension.'

Dr. Waters, while admitting that his investigations do not enable him to say what is the nature of the degeneration which leads to emphysema, and that his microscopical researches on this point have yielded no results, adds, 'I do not entertain the slightest doubt that the disease in its severer forms is of a constitutional nature.'

Varieties of Pulmonary Vesicular Emphysema.—As over-distension of the air-vesicles may occur in perfectly healthy lungs, and in lungs the seat of any of those pathological changes which impair their elasticity, and as, moreover, the dilatation may affect the air-vesicles of the whole, or of a great part of the lung, or may be limited to the air-cells of a small part of the lung, pulmonary vesicular emphysema has been divided into varieties.

The various forms of pulmonary vesicular emphysema may be described under the four following heads:—

Acute vesicular emphysema.

Chronic local emphysema.

Large-lunged (or hypertrophous) emphysema.

Small-lunged (or atrophous) emphysema.

Although perfect and uncomplicated specimens of each variety are common, cases of pulmonary vesicular emphysema are frequently seen in practice and in the dead-house, in which these several varieties are conjoined in the same lung; and, again, cases which cannot at the time when they come under observation be referred absolutely to the one or the other group. The reasons for this are manifest when the etiology and the pathology of the affection are considered.

ACUTE VESICULAR EMPHYSEMA.—By acute pulmonary vesicular emphysema is signified acute over-distension of previously healthy air-vesicles.

The part of the lung, the air-vesicles of which are over-distended is puffed up, is paler than it should be; the vesicles themselves, seen through the pleura, are manifestly larger than natural. The pallor is due solely to the excess of air in the vesicles stretching their walls, and so separating the capillaries further than should be from each other. The meshes of the capillary network on the walls of the air-vesicles are widened. Acute vesicular emphysema may be produced by too much air being drawn into the over-distended air-vesicles by inspiratory effort; or by too much air being driven into the air-cells of parts of the lungs by extreme compression of other parts by expiratory efforts, while the escape of the air by the natural outlet is prevented or retarded, *e.g.* by closed glottis, narrowing of the trachea or bronchi.

Both these forces conspire to determine the occurrence of acute over-distension of the air-vesicles in acute bronchitis. In that disease disseminated collapse, and the consequent diminished bulk of lung, and increased desire for breath, lead to violent inspiratory efforts and over-distension of the previous air-vesicles; while the frequent and violent expiratory efforts with closed glottis (preparatory to cough) determine over-distension of the air-vesicles of the less compressed and less supported parts of the lung.

When the ribs are greatly softened, as in some cases of rickets, the anterior margin of the lungs is the seat of acute

vesicular emphysema. The over-distension of the air-vesicles is produced partly by the compression of the lung at a little distance from its margin by the recession during inspiration of the ribs at their junction with their cartilages, but chiefly by the great advance of the sternum and rib-cartilages during inspiration, these parts being thrust forward by the impressing ribs.

In acute pulmonary vesicular emphysema, the rule is that the air-vesicles resume their normal size as soon as, or very soon after, the over-distending force ceases to act. The walls of the air-vesicles and the adjacent tissues being healthy, they contract to their normal dimensions.

In comparatively rare cases the over-distension is so great, so long-continued, or so frequently repeated, that the over-stretched walls of air-vesicles are injured, their elasticity is impaired, and the air-vesicles continue permanently larger than they should be.

It is in this way that severe and prolonged whooping-cough in children appears to produce chronic pulmonary vesicular emphysema. The over-distension especially affects the air-vesicles of the apex and anterior margin of the lungs, the air being forced into those parts during the violent expiratory efforts which precede the cough.

Symptoms.—If widely spread and extreme acute pulmonary vesicular emphysema causes increased resonance of the chest; the symptoms due to the lesion are masked and altogether lost in those proper to the disease to which it is secondary.

It requires no special treatment.

CHRONIC LOCAL EMPHYSEMA is characterised by extreme permanent over-distension of a few vesicles. The large vesicles are formed by the coalescence of several smaller. The largest may be as large as a poulet's egg, are not unfrequently the size of hazel-nuts, though more commonly not larger than peas. In the same group vesicles are often found varying in size from a pin's head to a hazel-nut.

The walls of these large vesicles are never healthy; they are thick, opaque, wanting in elasticity, and vessels of some size frequently ramify on the larger. Threads composed of obliterated bronchi, the remains of vessels or of lung tissue,

cross the cavity of the larger vesicles. Sometimes these vesicles communicate with a small bronchus; at others the bronchus leading to them is occluded.

The most common seat of chronic local emphysema is the apex of the lung, then the anterior margin, and the margin of the base of the lung. At the apex the emphysema is often conjoined with the remains of old tubercle.

The pathology and mechanism of the production of chronic local emphysema is best studied as it occurs at the apex of the lung, when that part is the seat of obsolescent or of calcified tubercle. When tubercles obsolesce or calcify at the apex of the lung, a considerable portion of lung tissue in their vicinity is usually the seat of chronic congestion and exudation of lymph. This portion of lung loses its porosity, becomes tough, inelastic, and puckered, *i.e.* irregularly contracted. Here and there, however, portions of the lung textures are damaged, not destroyed, so that some air-vesicles still admit air.

In health the inspiratory and expiratory forces are at a minimum at the apex. But during expiratory efforts with closed glottis, as in severe cough, the air is driven from the more compressed parts into the little compressed apex, and thus the vesicles still pervious to air are over-distended; and as their walls have, from the previous changes in the tissues of the apex of the lung, lost much of their elasticity the over-distension is permanent. Every paroxysm of cough must add to their dilatation. The diminution in size of the apex assists, as a permissive cause, in the production of extreme chronic local emphysema at the apex. Thus in proportion to the loss in the elasticity of the air-admitting textures, to the frequency and the violence of the expiratory efforts with closed glottis, and to the permanent diminution in the size of the apex, will be the degree and the rapidity with which local emphysema at that part will be established.

The anterior margin of the lung, the margin of the base, the anterior inferior angle of the superior lobe of the left lung are, like the apex, very imperfectly compressed and supported during expiratory efforts, and so air is forced powerfully into the vesicles of those parts; and should the texture there be damaged at any time so as to diminish its elasticity the

result will be great dilatation of a few vesicles. The margins of the lungs are thus sometimes fringed with large vesicles.

Chronic local emphysema is always a secondary lesion. Its formation at the apex is the consequence, not the cause (as some have fancied), of the obsolescence of tubercles. Coincident with the obsolescence is damage to the air-admitting textures of the lung, and it is that damage which renders the chronic local emphysema with large vesicles possible.

Symptoms.—The development of emphysema at the apex of the lung, when that part is the seat of chronic consolidation with contraction, diminishes the depression of the shoulder, and of supra- and infra-clavicular regions, and increases the resonance of the same parts; the dilated vesicles often projecting above and surrounding the solid textures. The dilatation of the vesicles may be so extensive and considerable as to cause supra-clavicular bulging either permanently or during cough.¹ It is unattended by other symptoms.

Treatment.—From the nature of the lesion it will be understood that no treatment is required.

LARGE-LUNGED VESICULAR EMPHYSEMA.—By this name it is proposed to designate those cases in which there is over-distension of the air-vesicles of the whole, or of a large section of one or of both lungs, great increase in bulk of the lungs, or of the affected part of the lungs, and corresponding increase, local or general, in the capacity of the thorax. The term hypertrophous pulmonary vesicular emphysema has been used to describe the same set of cases.²

¹ The bulging part is resonant, and cannot therefore be confounded by a tolerably careful observer with the prominence of the same part due to distention of the veins during severe cough.

² Large-lunged is by far the better of the two names, because it involves the expression of no opinion in regard of disputed facts. Many observers regard pulmonary vesicular emphysema as atrophic from its outset, no matter how it originates. And it must be admitted that, even when the disease has been hypertrophic when first established, the lungs may be greatly wasted in regard of their essential anatomical constituents before death. And, again, in some cases of large-lunged vesicular emphysema, as in those in which the occurrence of the disease is determined, and its continuance secured by increase in the capacity of the chest from age-degeneration of the ribs and cartilages without diminution in the power of the respiratory muscles, the wasting and rarefaction may not be preceded by hypertrophy of any anatomical constituent of the lung.

General large-lunged vesicular emphysema is a very serious disease. The symptoms directly due to it are grave; the diseased conditions dependent on it for their origin are very frequently fatal. Thus a large proportion of cases of heart-disease have their starting-point in large-lunged vesicular emphysema.

It rarely occurs in a marked form before the middle of life, and it more commonly affects those disposed to accumulation of fat in the subcutaneous tissue and internal parts. Lungs, the subject of this form of vesicular emphysema, are larger and drier than healthy lungs.¹

The parts uncoloured by pigment are paler than healthy lung. The lungs overlap the pericardium to a considerable extent, and meet above it even to near the top of the sternum; they have a down-cushion-like feel, and retain the impression of the fingers. When the thorax is opened they contract less than healthy lungs do under like circumstances.

Large-lunged vesicular emphysema is, in the great majority of cases, preceded by attacks of bronchitis, by congestion of the lungs, by dry winter cough, or by chronic bronchitis; that is to say, by diseases having as immediate consequences toughening and thickening of the tissues of the lung,² and severe cough; in other words, diminished elasticity of the lungs, and powerful expiratory efforts with closed glottis.

By far the most common determining cause then of the over-distension of the air-vesicles in large-lunged vesicular emphysema is powerful expiratory effort with closed glottis; and the most common permanence-securing cause is the

¹ When vesicular emphysema follows on bronchitis, congestion of the lungs, and similar pathological conditions, the lungs, at the very outset of the disease, weigh more than in health, and would continue to do so were it not for the waste of the normal anatomical constituents of the lung—blood, blood-vessels, epithelium, or intercapillary nuclei—which follows on over-distension of the air-vesicles, and on the lesions which secure the permanence of their over-distension.

² To comprehend the relation between bronchitis, the changes following it in the walls of the air-vesicles, and the frequency with which bronchitis supervenes on pulmonary vesicular emphysema, it must be remembered that the blood of the bronchial arteries is returned to the heart chiefly through the pulmonary veins, and that many good observers affirm that the bronchial mucous membrane is in great measure nourished by the blood of the pulmonary artery, and that anastomoses exist between the finest divisions of the bronchial and pulmonary arteries.

changes in the texture of the walls of the air-vesicles resulting from excess of blood in their capillaries.

The next most frequent determining and permanence-securing causes of large-lunged vesicular emphysema are diminished ordinary or habitual expiratory force, dependent on age-degeneration of the bones and cartilages in the thoracic parietes, without loss of full muscular inspiratory power, occurring alone, or more commonly conjoined with thickening of the tissues and diminished elasticity of the lungs, changes also due to age-degeneration.

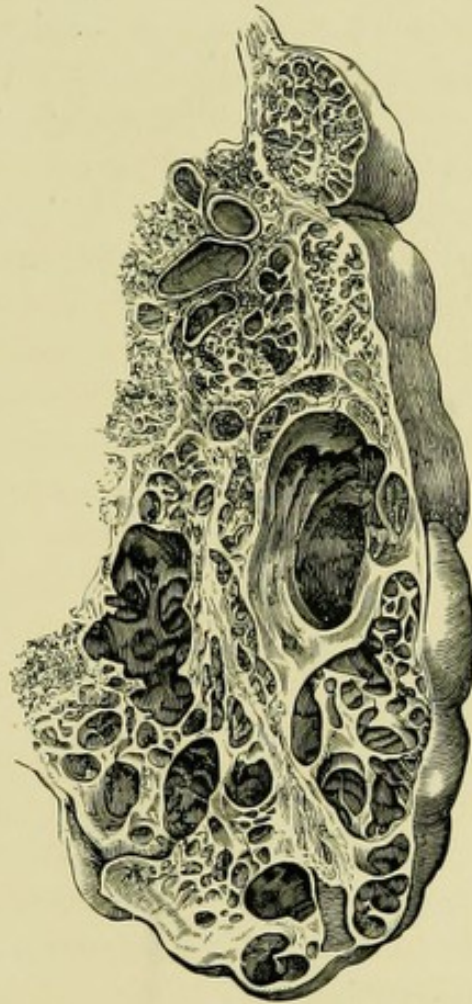
As bronchitis, winter cough, and congestion of the lungs are common at advanced periods of life, *i.e.* at the period of life when, without loss of muscular inspiratory power, age-degeneration of the bones and cartilages of the thorax and of the lungs is common, it is manifest that violent expiratory efforts with closed glottis, habitually defective expiration, and therefore excessive inspiratory dilatation, changes in the lung due to congestion, and changes in the lung-tissue due to age-degeneration, must in a very large number of cases conspire to produce large-lunged vesicular emphysema.

The changes which occur in the texture of the lung, in consequence of continued congestion, have been admirably described and figured by Rokitansky. (See Fig. p. 167.)

When the lung is congested, as from disease of the left side of the heart, an increase in the quantity of the connective tissue occurs, the walls of the air-vesicles are thickened, the parenchyma appears thicker and swollen, and unusually resistant.

On section of the lung the margins of the lung-vesicles are thicker than in health, and the cavity of each vesicle more visible than it should be, because its thickened walls prevent collapse. Sometimes the cavity of each vesicle is increased, and the lungs are larger than they should be; in other words, the substance of the lung is toughened and thickened from the formation of tissue, and enlargement of the lung, with dilatation of the vesicles, follows when any of the determining causes of over-distension of the air-vesicles come into action.

But, however produced, permanent over-distension of the air-vesicles is followed by various pathological changes in



SECTION OF LUNG IN ADVANCED STATE OF CHRONIC VESICULAR EMPHYSEMA.

(From Rokitansky, *Lehrbuch der Pathol. Anatomie*, B. iii. 1861.)

SECTION OF THE IN ALPHABETICALLY OF CHRONIC IN THE
(The History of the United States, 1776-1876)

their walls. Some of these changes are the direct mechanical result of their over-distension; some are the result of degenerative changes in the structures thus mechanically injured; some of defective nutrition consequent on the injury inflicted on the capillaries of the walls by their stretching; some of altered nutrition due to the alterations in structure; some are due to the pathological states to which the permanence of the over-distension is owing. So that when the disease is far advanced, and has existed for some time, not only are individual air-cells enlarged, but the partitions between many are perforated; between others they are reduced to mere ridges; at places they have altogether disappeared; and at places they are greatly, though it may be irregularly, thickened by imperfectly constituted fibrous tissue formed in and about the normal structures. And so ultimately, atrophy of some structures is conjoined with increase in size and thickness of others; and rarefaction and condensation may affect adjacent parts of the same lung.

If a portion of lung in an advanced stage of vesicular emphysema be inflated, dried, and then cut across, the cut surface appears to be made up of spaces varying in size from a millet-seed to a hemp-seed, while near to the apex and margin of the lungs may be a few much larger spaces. These spaces or small cavities are separated and intersected by septa and by threads of very variable degrees of thickness. (See Fig.)

Black pigment accumulates in considerable quantity on the inner surface of the dilated vesicles, and amid the fibrous and other solids. This black pigment owes its origin in part to the conversion of the hæmatin in the partially destroyed capillaries into melanin.

When the whole of both lungs are emphysematous, the changes just described are much more advanced at the margins and apices than they are elsewhere; and, as a rule, they are more advanced at the base of the left, than they are at the base of the right lung; these being, in the great majority of cases, the parts first to suffer in large-lunged vesicular emphysema, because they are the parts least compressed and least supported during expiratory efforts with closed glottis.

Effect of over-distension of the air-vesicles on the circula-

tion.—The capillaries of the pulmonary artery distributed on the walls of the air-vesicles are at first stretched in proportion to the over-distension of the vesicles, and then, the over-distension continuing, some of the stretched vessels give way and are obliterated.

The passage of blood through the capillaries lengthened by stretching must be attended by increased friction, in proportion to the lengthening and narrowing of the vessels.

Destruction of the capillaries diminishes the number of channels through which the blood can pass, and so impedes, in proportion to the number of capillaries torn, the passage of the blood from the right to the left side of the heart.

Impediment to the flow of blood through the lungs is the cause of the greater number of the primary and secondary symptoms of large-lunged visicular emphysema. The several causes of impediment to the flow of the blood through the lung and their modes of action are—

- 1 Deficient extent of chest-movement in ordinary respiration; especially deficient ordinary or habitual expiratory movement.

- 2 Violent expiratory efforts with closed glottis; by the pressure brought to bear on the heart and great vessels, as well as on the air in the interior of the air-vesicles, and so on the capillaries in their walls.

- 3 Diminished resistance from loss of elasticity of the lung; by disturbing the normal proportion borne by the pressure of the air on the inner to that on the outer chest-walls.

- 4 Lengthening of pulmonary capillaries; by increasing the friction.

- 5 Destruction of pulmonary capillaries; by diminishing the channels for the passage of the blood from the pulmonary artery to the pulmonary vein.

As the establishment of an efficient collateral pulmonary circulation is anatomically impossible, any impediment to the flow of blood through all, or nearly all, the pulmonary capillaries must have as direct result impediment to the escape of blood from the right ventricle.

The first effect of difficulty to the passage of blood through the pulmonary capillaries must be, in accordance with general laws, increased efforts, and so hypertrophy of the

walls of the right ventricle; increased pressure on the inside of the right ventricle; and so dilatation of its cavity.

At the outset, the impediment to the onward passage of the blood may at parts of the lung be trifling, compared with the impediment at other parts; in such case these parts suffer from the increased blood-pressure, become hyperæmic, and, it may be, œdematous.

The impediment to its onward passage is soon felt by the blood in the right auricle and in the whole systemic venous system, of which the right heart is merely the terminus. When the auricle and ventricle are dilated, the right auriculo-ventricular orifice is dilated, and the result of increase in its circumference, without corresponding increase in the size of the tricuspid valve, is incompetence of the valve to close the dilated opening, and regurgitation of blood during the ventricular systole, from the right ventricle to the right auricle, and veins opening into it.

But the impediment to the flow of blood through the pulmonary capillaries is not only followed by over-distension of the venous system, but ultimately the blood passes from the systemic capillaries into the veins with difficulty, and so an impediment arises to the escape of blood from the arteries, and from the left side of the heart, which is merely the head of the general arterial system.

That such impediment to the escape of blood from the arteries does exist when there is strong impediment to the flow of blood through the lungs, is manifested by placing the finger on an artery when a patient suffering from general pulmonary vesicular emphysema coughs violently, the artery instantly becomes full and tense, and, for the second of violent expiratory effort, ceases to pulsate.

Over-filling of the capillaries of an organ or tissue with retardation of the flow of blood through them never continues for any length of time, and is never repeated frequently without inducing changes in the structure of that congested organ or tissue.

The changes of the several organs, resulting from mechanically-induced congestion, are considered at length in the articles on diseases of special organs. Only such changes of special organs as give rise to the more important symptoms

in bad cases of large-lunged pulmonary vesicular emphysema, will be here considered.

Speaking generally, if an organ be the seat of mechanically-induced intermitting congestion, the earliest result is increased nutrition and enlargement of the organ. When the dilatation of the capillaries has reached a certain degree and becomes permanent, then wasting of the structures of the part with increase in connective tissue, especially of imperfectly-formed connective tissue, may result. The formation of the latter may precede, and greatly preponderate over the wasting of the natural structures of the part. A large number of free granules, of oleine and proteine, are found among the proper anatomical elements of the part almost from the very commencement of the congestion; and fatty degeneration of the normal structures frequently precedes their disappearance.

The parts that especially suffer in large-lunged vesicular emphysema are—

The Heart.—First and most certainly, the heart.

The first effect of the impediment to the passage of the blood through the lungs is increase in the muscular tissue of the right side of the heart; then follow accumulation of blood in the ventricle, and some dilatation of its cavity. The right auricle next suffers in the same way, and soon the whole venous system: the veins of the heart suffering over-distension in common with the other veins.

Mechanically-induced congestion of the walls of the heart, with increased action of the organ, leads not only to hypertrophy, but ultimately to induration and toughening of the walls. When these changes have occurred in its muscular tissue, the heart loses its power of close contraction, and permanence of the dilatation, produced by the pressure of the blood on its inner surface, is the result.

Free granules of oleine and proteine are found between the muscular fibres; and, after or shorter time, fatty degeneration of the damaged muscular tissue follows.

When distension of the veins has reached a certain point the blood escapes from the systemic capillaries with difficulty, and increased action of the left ventricle follows. As the walls of the left side of the heart suffer from the same

mechanically-induced congestion as the walls of the right side, when impediment to the escape of blood from the left ventricle is established, its walls and cavity experience, though in a less degree, the same changes in texture, etc., as the right side of the organ, viz., hypertrophy, induration, toughening, and permanent dilatation.

The Liver.—The radicles of the hepatic vein, then the terminal twigs of the portal vein, and finally its radicles suffer congestion from the same cause as the systemic capillaries, *i.e.* from the impediment to the escape of blood from the inferior vena cava.

In consequence of the impediment to the circulation, the liver is first enlarged from mere congestion, and in this stage a variety of 'nutmeg liver' is found after death.

When the congestion has continued for some time, the organ is more or less enlarged, indurated, and toughened, and free granules of oleine and proteine infiltrate all its tissues, then its natural structures waste, especially, it is said, the cell element, and a certain amount of granular atrophy is the final result.

Ascites very rarely occurs before the hepatic structure is organically injured, and rarely to any great amount from those changes only which follow directly from the impediment to the circulation here considered.

The Kidneys, in common with other organs, suffer congestion in cases of extreme large-lunged vesicular emphysema.

This extreme congestion is evidenced during life by the presence of albumen, and sometimes of blood, in the urine.

The kidney suffering from mechanically-induced congestion is at first larger, darker, and moister than in health. Granules of oleine and proteine are scattered through all its structures. After a time, induration and toughening of the organ follow. A slight amount of granular atrophy of the previously enlarged kidney is the ultimate result.

The Connective or Cellular Tissue throughout the body suffers from its mechanically-induced congestion. Its texture is toughened and thickened, and serosity is effused into its meshes.

Anasarca is one of the earliest consequences of over-filling of the venous system from impediment to the flow of blood

through the lungs. The anasarca is frequently attributed to the regurgitation of blood through the right auriculo-ventricular orifice; but both the regurgitation and the anasarca are really due to a common cause, *i.e.* to the state of the pulmonary capillaries. As a rule, however, before over-distension of the veins is so great as to relieve itself by letting out serosity into the cellular tissue, the pressure on the inside of the right ventricle and auricle is sufficient to dilate the auriculo-ventricular orifice to such an extent, that the tricuspid valve is incompetent to its closure, pulsation in the jugulars is perceptible, and the anasarca is then erroneously attributed to tricuspid regurgitation, as it is often called.

Blood and General Nutrition.—Niemeyer has pointed out that congestion of the venous system from mechanical impediment to the onward flow of blood through the lungs, or right heart, cannot exist without causing impediment to the escape of its contents from the thoracic duct. To this Niemeyer attributes the deficiency of fibrine and of albumen in the blood in cyanosis dependent on mechanically-induced over-filling of the venous system, and to it he also attributes the general emaciation which occurs in advanced cases of pulmonary vesicular emphysema.

The Vessels of the Lungs.—In the last stages of the disease, after the left ventricle has suffered hypertrophy and dilatation, secondary lesions of the lung not unfrequently occur, thus the lungs may become greatly congested, and œdema of the lungs or congestive pneumonia follow. The mechanical impediment to the flow of blood through the pulmonary capillaries has told back through the systemic capillaries on the left side of the heart, and so on the radicles of the pulmonary veins.

Symptoms of Large-lunged Vesicular Emphysema.—The chief direct symptoms of large-lunged vesicular emphysema are—

- (a) Increase in the size of the thorax.
- (b) Increase in the resonance of thorax, and prolonged expiration.
- (c) Shortness of breath.
- (d) The lungs are larger than in health, and the capacity of the thorax is in proportion to the size of the lung.

The increase in the circumference of the thorax is effected chiefly by diminution in the natural obliquity of the ribs. By this alteration in the direction of the ribs, the lower intercostal spaces are very considerably widened.

The sternum is carried forward.

The lower latero-dorsal bulging of the thorax is increased.

The enlargement of the circumference of the chest thus gained is made still greater by posterior curvature of the lowest cervical, the dorsal, and upper lumbar part of the spinal column. The patient stoops, he grows round-shouldered and round-backed.¹

Increase in the capacity of the thorax from above downward is produced by lowering of the diaphragm. At the termination of expiration in extreme cases of large-lunged vesicular emphysema the diaphragm lies very low, so that it is not in contact with the inner surface of even the lowest rib.

When the air-vesicles of the upper half of the lungs are the first to suffer over-distension, or are much more affected than are the air-vesicles of the lower part of the lung, the upper part of the thorax is disproportionately larger. When the determining cause of the over-distension has been violent cough from bronchitis, then the disproportion in size between the upper and lower part of the thorax is sometimes increased by imperfect expansion of the lower part of the lungs; the condition of the bronchial mucous membrane and the contents of the bronchial tubes preventing the free and ready entrance of the air into the air-cells of these parts of the lung.

The increase in the capacity of the thorax is determined by the forces which determine the over-distension of the air-vesicles, viz., by repetition of full inspiratory efforts, expiratory efforts with closed glottis, and diminished elasticity of the thoracic parietes, or of the lungs, or more commonly of the two conjoined.

(b) Of the physical signs, after those furnished by inspection of the thorax, by far the most constant and important in regard of diagnosis, is increased resonance on percussion—clear full sound. The abnormal clearness on percussion is

¹ Whenever the depth of the chest, from before backwards, requires to be increased, *e.g.* in dilatation of the heart—effusion into the pericardium—the patient instinctively rounds his back and elevates his shoulders.

due to the relative increase in the quantity of air in the chest, and to the tension of the chest-walls.

As the large lungs overlap the heart, the region of pre-cordial dulness is diminished, and as the diaphragm is flattened, the hyper-resonance extends posteriorly even to the twelfth rib, and in front often as low as the margin of the thorax, even on the right side, the liver lying altogether under the abdominal parietes.

Expiration is, in extreme cases, considerably prolonged in consequence of the diminution in the resilience of the chest-walls and lungs, and of the large size of the latter. At the same time, the inspiratory murmur is short and feeble. But when this form of pulmonary vesicular emphysema is limited to a part of the lung, the only physical signs are local bulging and hyper-resonance.

(c) Shortness of breath is always present in large-lunged vesicular emphysema. At first, the shortness of breath is only felt on exertion; the patient cannot mount a hill as he did. Then, when walking on level ground, he requires to stop from time to time to take in breath—he breathes too frequently, and pants a little; or it may be that he ‘suffers with his breath’ after a full or an indigestible meal, when the descent of the diaphragm is impeded by a distended stomach. However the shortness of breath is induced, the subject of large-lunged vesicular emphysema is, from a very early period, conscious that his ‘wind’ is no longer what it was.

As the disease advances, the shortness of breath is experienced on the least exertion, *e.g.* ascending a few steps or a gentle slope; and finally, even when sitting on a chair. The patient is always panting.

By the altered position of the ribs and the diaphragm, a considerable increase in the capacity of the thorax is, as has been shown, obtained, but it is obtained at the expense of the inspiratory capability. The chest-walls are constantly expanded, and when the disease is far advanced, the capacity of the chest may be greater at the termination of expiration than, in the normal condition of the lungs and chest-walls, it should be at the termination of inspiration. As Dr. C. J. B. Williams has tersely expressed it, ‘Breath is taken as it were on the top of the breath.’

The lowering of the diaphragm may be so considerable, it is said, as to cause its physiological action to be reversed. In place of increasing the capacity of the thorax by its contraction, the diaphragm may draw, it has been said, the lower ribs inwards, and so diminish to a slight extent the capacity of the lower part of the chest at the end of inspiration.

The diaphragm may be forced downwards by the expiratory efforts, which determine over-distension of the air-cells, but it probably never lies very low till the elasticity of the lungs is considerably impaired.

The great natural agent in effecting the ascent of the diaphragm, after it has been lowered by its own contraction, is the elasticity of the lungs. The muscles relax at the termination of inspiration, and the diminution in the size of the lungs resulting from their resilience, greatly aids in determining the passive ascent of the diaphragm. When the lungs, from loss of elasticity, no longer diminish in size as much as they should at the termination of inspiration, the ascent of the diaphragm is less than it should be, and it begins to act at the commencement of each inspiration from a lower and lower level; consequently, the increase in the capacity of the thorax obtainable by its contraction is always lessening, till finally it is perhaps just possible that its normal physiological action may be, as above stated, reversed.¹

When the ordinary muscles of inspiration are, in consequence of the permanent expansion of the chest, unable to dilate it sufficiently to take in a proper supply of air, all the extraordinary muscles of inspiration are habitually employed in breathing, hence the muscles of the neck, back, etc., capable of aiding inspiration are, after a time, considerably hypertrophied, the shoulders are raised, and the enlargements of

¹ The common cause of recession of the lower part of the chest during inspiration is some impediment to the free entrance of the air into the lungs, and the pressure of the external air for this reason being brought to bear with undue force on the outside of the thorax by the powerful action of the inspiratory muscles. The lower parts of the chest walls are there most yielding, and are therefore pressed inwards by the weight of the atmosphere. On this and other points connected with the deformity of the chest in pulmonary vesicular emphysema, the reader is referred to Dr. Sibson's elaborate and most able paper in the thirty-first volume (1848) of the *Medico-Chir. Soc. Trans.*, 'On the Movements of Respiration in Disease.'

the muscles of the neck, the scaleni especially, give a peculiar breadth to the neck.

Imperfect aëration of the blood resulting from the damaged state of the pulmonary capillaries, and the changes which take place in the walls of the air-vesicles after they have been long over-distended, add greatly to the shortness of breath; while the dilution of the air taken in at each inspiration, by the large quantity of residual air left after expiration, must still further distress the breathing by interfering with aëration of the blood.

The shortness of breath, then, in uncomplicated large-lunged vesicular emphysema, is due to the small extent of movement of the chest-walls, including the diaphragm, during respiration, to the impurity of the air in the thorax at the termination of inspiration,¹ to the state of the capillaries of the pulmonary artery, and to the structural changes in the substance of the walls of the air-vesicles.

If bronchitis in any form or asthma supervene, the distress of breathing is greatly increased; and in some cases in which the distress of breathing has been unusually great fatty degeneration of the heart has been found after death (Virchow).

General description of the symptoms in a case of advanced Large-lunged Vesicular Emphysema.—The thorax is barrel-shaped; the antero-posterior, lateral, and vertical diameters are increased; the sternum is arched; the lower cervical, dorsal, and upper lumbar spine is curved, concavity forward; the ribs are too horizontal; the intercostal spaces are widened, and but little, if at all, depressed below the level of the ribs; the posterior bulgings on either side of the vertebral column are greater than they should be; the costal angle is larger than in health, and as the diaphragm is flattened and the lower part of the sternum is forced forward, at the same

¹ Although the capacity of the chest is greater in large-lunged vesicular emphysema than it is in health, spirometrical observations show that its vital capacity, as measured by the quantity of air that can be expelled after deep inspiration, is diminished. The residual air must, therefore, be much greater than it should be. Speaking of the difficulty of breathing in Emphysema, Magendie (*Leçons*, 1825, tom. i. p. 169) observes:—‘The tissue of the lung has lost some of its elasticity, and no longer reacts with sufficient force on the air which has penetrated into its parenchyma.’

time that both lungs are enlarged, the heart is at once less covered than in health by the sternum, thrust downwards by the forces that over-distended the air-vesicles, and carried downwards by the contraction of the diaphragm, and can, in consequence, be felt and seen beating below the ensiform cartilage. The heart, and especially the right ventricle, is dilated and hypertrophied, its impulse is heaving, and its dilatation and hypertrophy render the epigastric pulsation very perceptible.

The shoulders are raised, and the muscles of the neck and shoulders, especially the sterno-cleido-mastoidei, the scaleni, the omo-hyoid, and the trapezii stand prominently out.

The fossa behind the clavicle is frequently deepened; when, however, there is excess of emphysema above the level of the first rib, there may be post-clavicular bulging. Under all circumstances, when the patient coughs there is undue prominence, or bulging even of the post-clavicular fossa, and of the intercostal spaces, the air being forced from the more to the less compressed and supported parts by the expiratory efforts preceding the opening of the glottis.

The neck is broad from hypertrophy of its muscles, and its veins are unduly prominent. As the obstruction to the circulation increases, the veins of the neck pulsate synchronously with the beat of the right ventricle, and fill from below when emptied by the pressure of the finger. The whole venous system is manifestly dilated, the larger veins of the upper extremities have a knotted appearance from over-distension just above their valves, the hæmorrhoidal veins are enlarged, thickened, etc., and often bleed—it may be to the great relief of many of the discomforts from which the patient is suffering. The face has a coarse, bloated, dusky, and, on exertion, even livid aspect; the alæ of the nose and the lips, especially the lower lip, are thickened. The eyes are prominent, the conjunctivæ injected, occasionally yellowish, and the eyelids puffy—drowsiness, mental dullness, and headache are common symptoms. Emaciation is sometimes very considerable. The legs are œdematous, or the whole cellular tissue the seat of anasarca. Orthopnœa is often present, because in the recumbent position the

extraordinary muscles of inspiration can have only imperfectly supported points, in place of fixed points, from which to act; and again, because the weight of the body in the recumbent position interferes with the expansion of some part of the chest-walls, and the position and weight of the abdominal viscera with the descent of the diaphragm.

In this stage of the disease the urine frequently contains albumen, and now and then blood and blood-casts of tubes.

The abdomen is generally fuller than natural. The spleen and liver are increased in size, and the latter organ is frequently so much depressed by the determining cause of the distended lungs, by the enlarged and distended heart, and by the flattened diaphragm, that its upper convex surface can be distinguished by eye and touch through the abdominal walls.

When from supervention of bronchitis, or other cause, the impediment to the pulmonary capillary circulation is temporarily increased, the liver and spleen may be proved, by percussion and touch to be larger than before, and to resume their former size, as the circulation through the lungs becomes freer, and the mechanically-induced congestion is in consequence lessened.

The pulse in large-lunged emphysema is often small and weak, from the small quantities of blood which pass through the lungs and therefore into the left ventricle.

The urine is, speaking generally, that of imperfect respiration, and of congestion of the kidneys and liver. At times it is very abundant, pale, clear, and of low specific gravity; at others it is scanty, high-coloured, and loaded with lithates, which, as the urine cools, form a heavy brick-dust-like sediment. This deposit is not in all cases due merely to the concentration of the urine, there may be an absolute increase in the quantity of uric acid. Owing to the imperfect aeration of the blood there is a scanty supply of oxygen distributed through the system, hence the products of tissue metamorphosis are in a lower state of oxidation, and uric acid is formed to some extent in place of urea. Parkes thinks it is only when bronchitis is superadded to emphysema that there is such deficient oxidation as to lead to excess of

uric acid in place of urea. J. C. Lehmann, in a carefully observed case, found the urine after each attack of difficulty of breath deficient in urea and very acid. It contained oxalic acid and allantoin. To this Parkes objects that in Lehmann's case bronchitis complicated the emphysema, and refers to a case of uncomplicated emphysema so severe as to cause cyanosis and constant dyspnœa, observed by Ranke and himself, in which very little uric acid and a full quantity of urea were present in the urine. Biemer says, after quoting the observations of Lehmann and Ranke, that he has more often been able to detect small quantities of bile pigment in the urine.

A trace of albumen may, when the disease is far advanced, be constantly present in the urine; the quantity being increased with every increase of the impediment to the flow of blood through the lungs. When the congestion of the kidneys is suddenly greatly increased, or attains, even slowly, an extreme degree, the urine contains blood and blood-casts of tubes. Much albumen with little evidence of impediment to the flow of blood through the lungs, renders it probable that organic disease of the kidney is present.

It is not uncommon for the symptoms to be very trifling for some years, and then for a year or more, to see the graver symptoms only when the patient has an attack of bronchitis; with the cessation of the bronchitis the œdema of the legs the albumen in the urine, and the jugular pulsation frequently disappear. The over-distended heart and veins having their walls, as yet, to any serious degree, undamaged, contract nearly to their normal dimensions, when the extra impediment to the flow of blood through the lungs, due to the acute attack, has passed away. But the improvement is only for a time, another attack of bronchitis renews the serious symptoms, and after one or more such renewals, they are permanently established.

The variations in severity of the chief symptoms of large-lunged vesicular emphysema may be summed up thus:—the increase in size of the thorax varies from that obtained by a slight diminution in the natural obliquity of the ribs, or trifling local bulging, to the utmost expansion of the chest-walls; the hyper-resonance on percussion from slightly

increased clearness to the fullest clear sound; the prolongation of expiration, from an amount difficult to appreciate, to that in which it considerably exceeds in length the inspiratory sound. The impediment to the flow of blood through the lungs varies, from just enough to give, when the patient coughs, undue prominence to the great veins of the neck, to sufficient to cause hypertrophy and dilatation of the right side of the heart, jugular pulsation, and knotting and enlargement of all the superficial veins, anasarca, albuminuria, enlargement of the liver and spleen, dilatation of the systemic capillaries and arteries, hypertrophy and dilatation of the left side of the heart, and finally organic changes in the structure of all the organs in the body, and of the connective tissues generally. The shortness of breath varies from a mere 'touch in the wind' to inability to move without great distress of breathing.

The imperfect aeration of the blood varies from just sufficient to cause a slightly dusky hue of the lips on exertion, to enough to give the patient the purple or leaden hue of cyanosis.

When large-lunged vesicular emphysema is limited to a lobe or part of a lobe, as not unfrequently happens, then local fulness or bulging, and hyper-resonance with trifling shortness of breath, are commonly the only evidences of the disease.

SMALL-LUNGED (OR ATROPHOUS) PULMONARY VESICULAR EMPHYSEMA.—After large-lunged vesicular emphysema has lasted some time, and the over-distension of the vesicles is extreme, a certain, it may be considerable, amount of wasting of the tissues of the lung ensues; and thus a form of atrophous emphysema is established.

But in the variety of emphysema designated small-lunged or atrophous pulmonary vesicular emphysema, atrophy of the lung-tissue is the primary disease, or it supervenes on trifling primary over-distension.

Small-lunged vesicular emphysema is confined to persons well past middle life. Those who suffer from it are commonly thin. Withered-looking, shrivelled old persons frequently have their lungs damaged by this form of emphysema. It is

a far less troublesome and less grave affection than is large-lunged vesicular emphysema.

In primary general small-lunged vesicular emphysema the whole of both lungs suffer. There is waste of tissue, true atrophy. In some cases fatty degeneration has been said to precede absorption, or the final disappearance of tissue.

In this form of emphysema the separate vesicles are not dilated; but the partitions between adjacent vesicles with their pulmonary capillaries and other structures disappear or are reduced to mere threads, and two or more vesicles are thus thrown into one. No over-distending force is necessary to determine the increased capacity of the vesicles.

Lungs the subject of this disease are smaller, lighter, and drier than are healthy lungs. They would, from the destruction of the capillaries, be pale, but the pallor from this cause is commonly concealed by the large amount of black pigment spread through them. They are much less than healthy lungs, because they have lost much of their natural structures. The air-vesicles are large, but the lungs themselves are small.

The division between the superior and inferior lobe is more vertical than in health. The elasticity of the lungs is in a great measure lost—there is no resilience in them—they pit on pressure, and the pit remains. Their small size, their lightness, and the very small space into which they may be compressed, are often most remarkable. They are occasionally so much wasted, that, on opening the thorax, they sink back at once toward the spine and posterior part of the thorax. When the lungs and air-passages are in health, death takes place at the termination of expiration. On opening the thorax, healthy at the moment of death, and permitting the pressure of the air to bear on the outer surface of the lungs, there is at once a slight diminution in their size. This diminution is due to the elasticity of the lungs. Before the opening of the thorax the complete elastic contraction of the lungs was opposed by the pressure of the air on the inner surface of the air-vesicles.

In hypertrophous pulmonary vesicular emphysema the resilience of the lungs is diminished, hence when the thorax is opened there is less contraction of the lungs, and therefore less separation of the lungs from the chest-walls, than there

is when the lungs and the air-passages are healthy. The quantity of solid tissue constituting the walls of the air-vesicles, etc., and the irregular thickening of that tissue, prevent any mere collapse of the lungs.

When the lungs are in a state of extreme atrophous vesicular emphysema, they have not only lost in a great measure their elasticity, but a large quantity of their solid tissues has disappeared. The consequence is that, when the thorax is opened, and the pressure of the air on the internal and external surfaces of the lung is equalised, although little or no diminution may occur from its resilience, the weight of the lung may be sufficient to cause it to fall in like an inflated bag of wet paper.

If the subject of extreme atrophous pulmonary vesicular emphysema suffer from cough, then local emphysema with large vesicles is frequently superadded to the general atrophous emphysema. The elasticity of the lungs being diminished, the vesicles of the parts least compressed and least supported during expiratory efforts being permanently and greatly over-distended, atrophy of their walls throws several air-vesicles into one, and air being forced into the large cells so formed may lead to their extreme dilatation. For reasons previously assigned, these vesicles are found at the margins and apex of the lung.

Coincidentally with the occurrence of the changes in the textures of the lungs which constitute atrophous vesicular emphysema, the ribs and their cartilages experience degenerative changes by which their elasticity is diminished, but at the same time also the inspiratory muscles shrink and lose strength.

The chief direct symptoms of small-lunged or atrophous pulmonary vesicular emphysema are:—(1) shortness of breath; (2) diminution in the size of the thorax.

Shortness of breath in small-lunged emphysema is never felt to any notable degree, unless the patient makes exertion; and as the disease usually occurs in the aged, or in those wasted from other chronic diseases, persons indisposed and incapable of moving quickly, exertion sufficient to cause distressing shortness of breath is rarely made.

Primary atrophous vesicular emphysema is commonly

attended by general waste, and is therefore accompanied by waste of blood as well as of tissues ; so that the capillary pulmonary vessels, although reduced in number, still suffice for the passage of the diminished quantity of blood.

Again, the lungs being small, the expiratory power is enough to drive out the air, and the play for inspiration is considerable. The patient, in place of, as in large-lunged vesicular emphysema, always 'taking in breath on the top of breath,' in small-lunged vesicular emphysema inspires from the bottom of his breath.

The chest in small-lunged vesicular emphysema is diminished in capacity, and all its diameters are less than in health. The diminution in the antero-posterior and lateral diameters is obtained by a great increase in the obliquity of the ribs. The upper intercostal spaces next the sternum are widened and depressed below the level of the ribs. So obliquely placed are the lowest ribs, that their cartilages almost reach the crest of the ilium, and the intercostal spaces are lost, the ribs themselves really touching. The cartilages between the ribs and the sternum, as the ribs become abnormally oblique, bend so as to form an obtuse angle. Respiration is short, the thorax moves as a whole in inspiration, and the expiratory recoil quickly follows. The inspiratory murmur is short and feeble—the expiratory not prolonged. From the loss of elasticity in the ribs and cartilages, and the imperfect tension of the chest-walls, the resonance on percussion may be even less than in health, although the solids in the chest are diminished. As the lungs are small, the heart is less covered than it should be, and so the extent of precordial dulness may be increased, and that, although the heart itself may be partaking of the general atrophy. As atrophous emphysema is usually accompanied by waste of blood, and as the general muscular power of the patient forbids active exercise, there is commonly in small-lunged emphysema not only little distress in breathing, but no damming back of the blood in the right ventricle, overdistension of the venous system, dropsy, or hypertrophy, or dilatation of the heart.

Complications of Pulmonary Vesicular Emphysema.—The frequent conjunction of bronchitis and pulmonary

vesicular emphysema has been admitted from the time the latter was recognised as a special disease. Laennec considered the emphysema to be in all cases the consequence of bronchitis, and especially of that form of bronchitis which was designated dry catarrh. And Louis, while denying the relation of the two diseases as cause and effect, admitted the frequency of their co-existence.

It is now established that bronchitis is the most common cause of the direct production of pulmonary vesicular emphysema, and also that emphysema may be developed independently of bronchitis; and that when so established, the subjects of emphysema are very prone to suffer from bronchitis. So that although, as a rule, bronchitis is the cause of pulmonary vesicular emphysema, it may be the consequence; and the emphysema, in rare cases, may be unaccompanied from first to last by bronchitis.

One cause of the frequency with which bronchitis supervenes on pulmonary vesicular emphysema is, that when a part of the lung only is emphysematous, and the passage of the blood through the capillaries of that part is consequently impeded, hyperæmia of the non-emphysematous tissues is the result—the blood passing into the vessels of the part which offer it the less resistance.

Chronic general catarrhal bronchitis, with much violent cough and little expectoration, is the most common complication of general large-lunged vesicular emphysema. Acute capillary bronchitis is one of the most common causes of death in the same form of the disease. If the heart be, as it so often is, dilated and hypertrophied, then the acute capillary bronchitis is usually attended with much congestion of the substance of the lung.

Chronic bronchitis with profuse purulent expectoration is less common in large-lunged than it is in atrophous pulmonary vesicular emphysema. When the purulent discharge is considerable, the so-called amyloid degeneration of various organs is said to occur pretty frequently. In this case, amyloid degeneration is connected with the profuse supuration and not with the pulmonary vesicular emphysema.

Dilatation of the bronchial tubes is common in all forms of pulmonary vesicular emphysema.

In severe large-lunged vesicular emphysema, it is common to find an excess of fluid in the pericardium after death. It is only when congestion and œdema of the lungs have complicated the disease that an excess of serosity is found in the pleurae.

The subjects of large-lunged vesicular emphysema frequently suffer from disturbance of the digestive organs. The liver is loaded with blood, and the bile formed is thick and dark. The walls of the stomach are congested, and ultimately thickened. The result is notable disturbance of the stomach digestive processes. The patient suffers from visible distension of the epigastric region, and also from a sense of weight and fulness in the same part, especially after food, altogether disproportionate to the quantity and the quality of the food taken. Flatulence and acidity of stomach are troublesome symptoms.

The distension of the stomach is frequently so great as very decidedly to intensify, by the impediment it offers to the descent of the diaphragm, the habitual dyspnoea. The derangement of the stomach is also a not infrequent exciting cause of asthma.

Spasmodic asthma is a very common complication of pulmonary vesicular emphysema, especially of the large-lunged form of the disease. Attacks of extreme difficulty of inspiration supervene suddenly in the early morning, or at uncertain times, on the habitual shortness of breath. In addition to attacks of ordinary spasmodic asthma, the subjects of pulmonary vesicular emphysema often suffer for weeks together from increased dyspnoea, out of all proportion to any catarrhal bronchitis which may happen to be present. This dyspnoea is, in a great measure at least, due to spasm of the bronchial muscular fibres.¹

Phthisis.—One of the most marked anatomical characters of congenital tuberculosis, *i.e.* of an inherited disposition to the deposit of tubercle, is the small size of the lungs. It is by no means uncommon for a deposit of tubercle to take

¹ It is possible that in many cases asthma precedes pulmonary vesicular emphysema, and the violent efforts to inspire are the determining causes of the emphysema, that in this as in so many other cases, the effect has been mistaken for the cause.

place in the apex of such lungs, and then for the tubercle to obsolesce or to calcify. The subjects of these changes frequently live to an advanced age. Chronic solidification with contraction of the apex of the lung, much black pigment in the solid tissue, and local emphysema with large vesicles, frequently follow the obsolescence or calcification of the tubercles.

After passing middle life, the subjects of these lesions frequently become affected with general atrophous pulmonary vesicular emphysema, rarely if ever with large-lunged vesicular emphysema. Subsequently, the solid tissue of the apex of the lung may undergo molecular disintegration, and a cavity be formed; death, in such cases, is said to have occurred from tubercular consumption, when, indeed, there are no tubercles present. This is a form of senile phthisis.

Headache and drowsiness are common consequences of large-lunged vesicular emphysema. These symptoms are due to congestion, the consequence especially of the mechanical impediment to the passage of blood through the lungs, aided in some cases by the imperfect aeration of the blood, and by those changes in the coats of the vessels so commonly seen in the subjects of the disease here considered. The veins and arteries, from changes in their walls, partly due to the congestion of their *vasa vasorum*, lose some of their normal reactive force, and hence the pressure of the blood on the inside of their walls leads here, as elsewhere, to their permanent dilatation. After death, we find not only increased capillary vascularity and excess of serosity in the meshes of the pia mater, but the arteries and veins in the same structure manifestly larger than they should be.

Diseases which in their progress are frequently complicated with Pulmonary Vesicular Emphysema.—All diseases accompanied by severe cough, by diminution of the whole or part of a lung, or by impediment to expiration, are accompanied by over-distension of the air-vesicles.

All diseases or changes in nutrition attended by damage to the elasticity of the lung, render permanent what would otherwise be transient over-distension of the air-vesicles.

Winter cough, catarrhal, and other forms of bronchitis,

are undoubtedly the diseases most frequently conjoined with pulmonary emphysema.

Diseases of the left side of the heart, by damming back the blood in the lungs, and so inducing changes in their texture, leads to permanent dilatation of the air-vesicles when their over-distension has once been determined by cough, etc.

Pneumonia is sometimes attended by acute vesicular emphysema of the air-admitting vesicles, but as the walls of the over-distended vesicles are healthy, and their over-distension is neither extreme nor of long duration, they return to their natural size when the pneumonia subsides. After solidification has passed away, the textures of the lung damaged by the pneumonic exudation may be the seat of permanent chronic emphysema.

When one pleura contains sufficient fluid to render the lung altogether useless, the opposite side of the thorax expands more than it should, and is for the time larger than in health, and its air-vesicles are enlarged in proportion to the degree of the expansion of the side. Should the impervious lung be, from long compression or other cause, so damaged as never again to admit any quantity of air into its vesicles, then permanent over-distension of the vesicles of the opposite lung is the consequence. It rarely happens that the distension of the air-vesicles in this case is sufficient to interfere with the capillary circulation on their walls, or to induce atrophy or other serious changes of the vesicular septa.

In chronic phthisis, the bases of the lungs very frequently suffer from vesicular emphysema; this is especially likely to happen when there has previously been dry pleurisy with adhesions at the same point. Hyper-resonance of the bases of the lung is, therefore, frequently conjoined with tubercular dulness of the apices. The expiratory efforts of cough are the determining cause; the damage inflicted on the textures by the pleurisy is a common cause of the permanence of the over-distension. The vesicular emphysema in this and similar cases, when tolerably limited in extent, is not attended with notable disturbance of respiration or circulation. It must, it is true, add a little to the shortness of breath, and

a little to the impediment to the circulation through the lungs, but these additions are insignificant in comparison with the primary disturbances of respiration and circulation resulting from the original disease. Diseases attended by incomplete occlusion of the air-passages frequently have, as consequence, over-distension of the air-vesicles of the whole or of part of the lung.

In accidental occlusion of the larynx, acute general vesicular emphysema is frequent. Thus, in the case of a woman, who in a state of drunkenness choked herself by thrusting the food with her finger from the overfull pharynx into the larynx, the lungs were the seat of extreme general acute emphysema.

In such cases, supposing the obstruction to be at first incomplete, the sufferer does what he would do if the obstacle were removable—he makes the deepest possible inspiration, and then coughs. The violent expiratory effort drives the air into the less compressed and supported parts. The local obstacle to the escape of air being irremovable, causes an excess of air to be retained in the lungs. A second deep inspiration follows, and so finally general over-distension of the air-vesicles is established.

The full normal distension of the lungs with air may be mistaken for acute general emphysema, if death occur while the lungs are distended by a deep inspiration, and a foreign body in the larynx prevent the expulsion of the air from the lungs by the natural death expiratory act.

In croup, the false membrane in the larynx may, in rare cases, act for a short time as a valve, admitting the air to pass into the lung, but opposing its escape, and so cause acute vesicular emphysema.

In croup, again, pulmonary lobular collapse and lobular pneumonia are both common, and, when present, some of the acute vesicular emphysema found after death may be secondary to those lesions of structure.

But more commonly than in either of the modes just enumerated, the acute vesicular emphysema found after death in croup is produced during the expiratory efforts of coughing; that it is so produced is proved by the situations it occupies, viz., those parts of the chest which in these cases

are seen during life to recede during inspiration, and to advance during expiration.

In whooping-cough, vesicular emphysema is a constant result of the violent expiratory efforts of that disease. When the over-distension is extreme, and is frequently repeated, the elasticity of the walls of the vesicles may be diminished, and then a certain amount of over-distension remains a permanent lesion. As bronchitis is a frequent complication of whooping-cough, disseminated lobular collapse may in some cases aid to a slight extent in the production of the vesicular emphysema.

Hereditary nature of Pulmonary Vesicular Emphysema.—Jackson¹ found that eighteen of twenty-eight subjects of pulmonary emphysema were born of parents, one of whom was affected with the same disease; while of fifty non-emphysematous patients, three only came of emphysematous parents; and Niemeyer remarks, 'I have known at least one family in which, without catarrh preceding, all the members for three generations suffered from emphysema.'

These facts afford some support to the theory that pulmonary vesicular emphysema is far too common a disease for Jackson's few oft-quoted observations, or for solitary baldly stated facts, such as that of Niemeyer, *to prove*, or even to render it highly probable that it is really hereditary.

If the cases be excluded in which the disease owes its origin to inherited predisposition to bronchitis, heart-disease, asthma, premature age-degeneration, etc., the facts adduced in support of the hereditary nature of pulmonary vesicular emphysema will be reduced to an insignificant figure. It is not denied that the disease may be hereditary, but it is without question in the writer's mind that the evidence adduced in support of its being hereditary in the sense in which tubercle and cancer are hereditary, is altogether insufficient for the proof.

Diseases of the lungs to which Pulmonary Vesicular Emphysema indisposes.—Pneumonia with exudation of

¹ Quoted by Louis.

lymph, croupous pneumonia, as it has been called, rarely occurs in the emphysematous parts of a lung. The destruction of the capillary vessels which occurs in chronic vesicular emphysema is supposed to afford a certain degree of exemption from this form of inflammation. Although a streak or two of blood in the sputa is not uncommon in the bronchitis from which emphysematous patients suffer, hæmorrhage in quantity from emphysematous lungs is said to be rare. Those suffering from atrophous vesicular emphysema alone enjoy a practical immunity from pneumonia and from hæmorrhage.

Tubercle has been said to be excluded by pulmonary vesicular emphysema.

No doubt active congestion of a part accompanies the formation of tubercle, and active congestion rarely affects the emphysematous parts of a lung, and to the same extent vesicular emphysema of the lung indisposes to the deposit of tubercle. It is to the wasting of the vessels in emphysema of the lung that the immunity, so much as it is, from tubercle is due.

The blood in pulmonary vesicular emphysema is less fully aerated than in health. To this venosity of the blood, Rokitansky attributes the infrequency of tubercle in the subjects of pulmonary vesicular emphysema. His theory, however, is opposed by facts.

TREATMENT OF CHRONIC PULMONARY VESICULAR EMPHYSEMA.—The treatment of pulmonary vesicular emphysema may be divided into curative, palliative, including the treatment of its direct consequences, and preventive.

Curative Treatment.—Some therapeutists have supposed that, by the lengthened administration of small doses of strychnine, and others, that by the skilful employment of electrical power, permanent contraction of the walls of the dilated air-vesicles may be obtained. But, although powerful agents for exciting muscular contraction, neither strychnine nor electrical action have any influence in restoring and increasing the elasticity of a tissue. In chronic pulmonary vesicular emphysema, it is the elasticity of the walls of the air-vesicles which is damaged. Experience, as might have

been anticipated, has afforded no evidence in support of the value of the drug or the battery in the cure of pulmonary vesicular emphysema.

Again, some therapeutists have placed persons suffering from chronic pulmonary vesicular emphysema in a chamber supplied with condensed air, in the hope that the breathing of the condensed air would cure the disease. Others have alleged that great attention to diet, and the administration of iron, and other blood, nervine, and stomach tonics, will, by improving the nutritive powers, cure the disease. But, if it be remembered, that to cure chronic pulmonary vesicular emphysema of severity sufficient to cause trouble to the patient, is not only to renew the elasticity of the walls of the air-cells, but also to restore the stretched vessels to their normal length and to their natural tone, to repair the apertures in the walls of the air-vesicles, and to replace the torn and otherwise destroyed capillaries by healthy vessels, it will be at once admitted that the cure of chronic pulmonary vesicular emphysema is impossible. Persons suffering from emphysema have been greatly relieved by breathing condensed air; but the relief, as might be anticipated, has been temporary only; and while attention to diet, etc., is of no avail to cure the disease, it is of great importance in staying the progress of the disease and relieving distress.

Preventive Treatment.—The great factors of pulmonary vesicular emphysema being:—Excess of pressure of air on the inside of the air-vesicles; age-degenerative changes of the parietes of the thorax; changes of the texture of the lung, from excess of blood in it; age-degenerative changes of the lung, in order to prevent the disease, and to stay its advance when established, care must be taken to guard against these, its determining and permanence-securing causes. Catarrhal, and all other forms of bronchitis being beyond question the most frequent excitors of the pulmonary vesicular emphysema, the prevention of these diseases is of the very highest importance. To secure immunity from bronchitis, and to prevent its recurrence, clothing must be adapted to the season, and it is necessary that cold and wet, especially fog and cold winds, be avoided. A mild climate has a

marked influence in preventing the attack of bronchitis, to which so many are subject during the winter in this country. Hence, it is most important for those whose lungs are the seat of vesicular emphysema to spend the winter in a mild, and not too dry, air.

When chronic or sub-acute bronchitis is present, the freer the secretion from the bronchial mucous membrane, and the less violent the cough, the less likely is chronic emphysema to follow. Expectorants and opiates combined are the great medicinal agents. The expectorants selected when the cough is dry, should be those that promote secretion; when the secretion is abundant, those that favour its expulsion.

Violent and irritative cough—that is, cough out of all proportion to the matter to be expectorated—should be restrained by sedatives: opium, belladonna, stramonium, conium, and prussic acid are the chief sedative agents in this class of cases. These drugs are more efficacious when given with little water, and in a small quantity of mucilage and syrup. Sedative inhalations are particularly useful. The sedative should be placed on the sponge of Maw's inhaler, and the steam of hot water passed through the sponge. Chloroform vapour exhibited in this way is sometimes very serviceable.

When the secretion from the bronchial mucous membrane is too abundant and purulent the mineral acids, quinine, iron, especially the tincture of the perchloride, and cod-liver oil, are invaluable; as is also the inhalation of mild stimulants, *e.g.* iodine diffused in small quantity through the room. In these cases, a change to dry sea-air is often very serviceable.

All efforts which try the muscular powers, as carrying heavy weights, are injurious. All exertions which induce panting, or oblige the person to stop frequently to recover his breath, are calculated to inflict permanent injury. Rapid walking, hill climbing, and violent exertions of all kinds, are to be carefully avoided. Walking exercise should, as much as possible, be limited to level ground.

Many an old gentleman has been hurried to his grave by attempting to follow the birds as he did in his earlier days

and by striving to improve his health by active exercise. It is a great gain for length of life to take old age pleasantly. Those predisposed to the disease and, *a fortiori*, subjects of pulmonary vesicular emphysema, should never attempt to play wind instruments.

When urging these points on a patient, it must never be forgotten that the permanence-securing cause being established, every single over-distension of the air-vesicles permanently increases their size. The increase on each occasion is indeed insignificant; but as every repetition of the over-distension adds to that previously existing, it follows that, should the over-distension be frequently repeated, a considerable amount of dilatation must be the ultimate result.

All the foregoing means which are of importance in preventing the occurrence of the disease are practically still more important as preventing its increase when established. Many a man whose wind was merely touched, has become dropsical, etc., by attempts to renovate himself, by endeavours to climb, hunt, and shoot as he did before his 'wind' began to go. Old age has commenced on his chest; he is but between fifty and sixty, and he won't admit the existence of it. He strives against its inevitable consequences, and dies from the effects of the struggle years before he would have done had he shunned the contest.

All measures which oppose the supervention of the degenerative changes of age are to be sedulously employed, with the hope not only of specially retarding age-degeneration of the lungs and thoracic parietes, but of the body generally.

Diet, carefully regulated exercises, and of drugs, iron and cod-liver oil, especially the former, are among the most potent means for effecting the object in view.

Palliative Treatment.—The distress from which the subjects of large-lunged vesicular emphysema suffer, is due—

1. To shortness of breath.
2. To congestions of distant organs produced mechanically by the impediment to the flow of blood through the pulmonary capillaries.
3. To the abnormalities of blood which result from the functional and structural changes of the liver and kidneys especially, consequent on their congestion.

1. The remedies for the shortness of breath vary according to its direct cause. Having regard to treatment, the causes of shortness of breath may be summed up thus:—

(a) Organic changes in the walls of the thorax, in the walls of the air-vesicles, and in the capillaries in the walls of the air-vesicles, and dilution of the air received into the air-vesicles at each inspiration, by the excess of air retained in them at the termination of expiration.

Breathing condensed air, it appears probable, temporarily relieves the distress of breathing due to the dilution of the air. Whether it does more than this is doubtful.

(b) Catarrhal and other forms of bronchitis.—For the shortness of breath arising from these affections, expectorants which both favour free secretion and expectoration are the great remedies. Ipecacuanha and carbonate and chloride of ammonium, squills and senega are the most potent remedies.

(c) Asthma.—Free secretion and expectoration from the bronchial mucous membrane affords the most efficient relief in continuous shortness of breath from this complication. Ipecacuanha, squill, ammonia, and senega alone, or combined with sedatives and anti-spasmodics, are the agents best calculated to attain the desired end.

It must not be forgotten that disturbances of the digestive organs, the liver, stomach, and bowels are common in large-lunged vesicular emphysema, and are also frequent exciting causes of attacks of spasmodic asthma in that disease.

(d) Congestion of the liver, accumulation of flatus in the stomach and bowels, and loaded bowels by interfering with the descent of the diaphragm, are common causes of shortness of breath.

It is in consequence of this that a full dose of blue pill, or calomel and colocynth, followed by a brisk, warm, saline aperient, so often affords marked relief to the dyspnoea of large-lunged vesicular emphysema.

Blue pill occasionally, aromatic saline, antacid aperients, taraxacum with soda, or nitro-hydrochloric acid with aromatics, and attention to diet, are the means best calculated to ward off shortness of breath from these causes.

2. In treating the congestions of organs, two objects have to be kept in view.

1st. To remove the impediment to the flow of blood through the lungs.

2nd. To relieve directly the local congestions.

The impediment to the flow of blood due directly to organic changes in the walls of the air-vesicles and in the pulmonary capillaries, is irremediable. Catarrhal and other forms of acute and chronic bronchitis increase the impediment to the capillary circulation through the lungs; and, therefore, to relieve those affections, is to relieve the congestion of the venous system. Free secretion and expectoration from the bronchial tubes is the most efficient agent for affording relief in these cases.

Violent cough again impedes the flow of blood through the lungs, and so produces congestion of the venous system.

Sedatives, therefore, by checking cough, become means of relieving local congestions.

Free secretion from the kidneys, liver, and intestinal mucous membrane, relieve the local and general over-distension of the capillaries and veins of those organs, resulting from impediment to the flow of blood through the lungs.

Of diuretics, the ordinary salts of potash, with small quantities of iodide of potassium, are, as a rule, the most efficacious. This class of remedies should be preceded by one or more doses of blue pill, with squill and digitalis.

It is common for diuretics not to act till the tension of the venous system has been, to some extent, taken off by other means. Hence, should diuretics fail when first given, aperients may be employed, and their use be followed by diuretics with advantage.

Blue pill, and other cholagogues, followed by hydragogue aperients, such as cream of tartar with jalap, effect the desired object by promoting a full flow of secretions from the liver and intestines, and so especially relieving congestion of the portal radicles and terminal branches.

A natural relief is occasionally afforded to a congested organ by spontaneous hæmorrhage from it. Cerebral congestion is relieved by epistaxis; congestion of the lungs by hæmoptysis; of the liver and intestines, by hæmorrhoidal bleeding; of the kidneys, by hæmaturia; of the stomach by hæmatemesis. The blood thus lost may not only relieve

the vessels of the organs from which it escapes, but the venous system generally.

When congestion of an organ is extreme, the application of dry-cups, or the removal of a small quantity of blood by cupping-glasses, is sometimes very useful. When the distension of the whole venous system is extreme, the removal of a little blood from the arm gives marked and sudden relief when judiciously performed.

The stomach dyspeptic symptoms are due chiefly to congestion of the stomach following on congestion of the liver. They are best treated by occasional doses of mercurials, saline aperients with mineral acids and mustard poultices to the epigastric region. These remedies may be followed by small doses of strychnine, and light aromatic bitters.

Aromatics, with alkalies, afford temporary relief to the sense of distension and weight.

3. The congestion of the kidneys is sometimes accompanied by the retention of urinary elements in the blood, congestion of the liver by slight jaundice, and finally by organic diseases of those organs, and then all the abnormalities resulting from those diseases follow.

As regards the special treatment of the conditions of blood dependent on the diseases of the liver and kidneys, it is here only necessary to say that the treatment before recommended for the relief of the congestion of these organs is that best calculated to secure the removal from the blood of the elements retained in it.

In cases of atrophous pulmonary vesicular emphysema the great object is to support the failing general powers. Iron is one of the most important tonics in this class of cases. A moderate supply of stimulants is useful.

When accompanied with profuse purulent expectoration, mineral acids, especially the sulphuric, with small doses of quinine, tincture of the sesquichloride of iron, cod-liver oil, and mild sea-air, are the great remedial agents. Stimulating inhalations are sometimes serviceable.

ON CONGESTION OF THE HEART
AND ITS
LOCAL CONSEQUENCES

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ON CONGESTION OF THE HEART AND ITS LOCAL CONSEQUENCES.

THE principal object of this paper is to call the attention of the Fellows of the Society to the occurrence of congestion of the muscular tissue of the heart; to the most common and direct consequences of that congestion, viz., induration, toughening, and thickening of the walls of the heart; and to the influence which those changes of texture exercise (as predisposing causes) on the development of permanent dilatation of the heart.

The expression, congestion of the heart, has been hitherto used to signify extreme distension of the cavities of the heart with blood; few pathologists have even mentioned congestion of the substance of the heart, and none have, I believe, so much as adverted to its most important consequences.¹ When the right cavities of the heart are over-distended with blood from an impediment existing to its onward flow, there is not only engorgement with blood of the *venæ cavæ* and of the veins opening into them, but also of the coronary sinus, veins, and branches. Now, it is a pathological law, that mechanically induced congestion, if long continued, slowly formed, and intermitting altogether or in degree, has

¹ Bertin notices the dilatation of the veins of the heart which accompanies engorgement with blood of the right auricle: 'Il (le sang) s'accumule donc dans leur cavité, distend leur parois, et les engorge jusqu'à leurs dernières extrémités.' He observes also that rupture of the distended veins may occur. (*Traité des Mal. du Cœur*, 1824, p. 41.) Rokitansky has the following remarks on the subject at page 267 of the second volume of the last edition of his *Path. Anat.* § 7: *Krankheiten der Textur.*—*Hyperämie*: 'Ein Strotzen der Gefässe des Herzens, zumal seiner Venen und kleine Blutaustretungen in Form von Hirsekorn—Linsengrossen Ecchymosen unter dem Pericardium in der Nähe des Sulcus transversalis, an den Verhöfen und an den Ursprungsstücken der Arterienstämme kommen bei Stenosen des Herzens und bei Asphyxien Neugeborner und Erwachsener häufig vor.'

for effect induration. and also (when the fibrin exuded is not of the powerfully contractile variety) permanent increase in bulk of the organ or tissue.

Thus, when the inferior vena cava is unable to pour its blood freely into the right auricle, the liver and kidneys are at first enlarged and softened from over-distension of their vessels and effusion of serosity into their structures. If the impediment to the onward flow of the blood be quickly removed, those organs soon recover their normal size and consistence; but, if the impediment continues for any length of time, then the organs in question are found to be indurated, toughened, and (except under the conditions previously specified) permanently enlarged. The induration, toughness, and enlargement, are due, chiefly at least, to an interstitial exudation of lymph, which is found after death, either amorphous, or in the form of granules, or more or less organised into fibrous tissue. It may be that, in particular cases, there is also some true hypertrophy of the proper structure of the congested part.

When the contractile coats of a hollow viscus are indurated, toughened, and thickened by an interstitial exudation of lymph, their normal contractile power is diminished. The consequence is, that although the resistance to over-distension of the walls of the viscus is greater than natural, yet, if the distending force be sufficient to overcome that resistance, and be repeatedly applied at short intervals over a length of time, permanent and extreme dilatation of the viscus will be produced.

What is true in these respects of other organs and tissues is true of the walls and cavities of the heart. When there is long-continued obstruction to the passage of the blood out of the right ventricle, and consequently impediment to the free entrance of the blood from the coronary sinus into the right auricle, congestion of the walls of the heart follows. If the impediment to the exit of the blood from the sinus be slowly produced, be moderate in degree and permanent, or be frequently repeated, then induration, toughening, and thickening of the walls of the heart will ensue, and permanent dilatation of its cavities be the final result; the over-distension of the walls of the cavities being the immediate cause of the

dilatation—the induration, toughening, and thickening being the cause of the permanence of the dilatation.

The peculiarities of the indurated and toughened walls of the heart are as follows:—

When divided across, the outer wall does not fall inwards, the form of the cavity being still retained, even though, as is sometimes the case, the walls are (in consequence of the extreme dilatation of the cavity) thinner than natural; the cut surface of the muscular tissue has a very smooth, compact, homogeneous appearance; the columnæ carneæ stand firmly out; the tissue is harder than natural, and singularly tough. This tough, leather-like quality is one of the most marked characters. The colour of the indurated, toughened tissue may be paler or darker than natural.¹

The microscopical appearances of the indurated and toughened tissue are—

1st. The striæ of the muscular fibres are generally indistinctly seen, though here and there they are as perfect as in health.

2nd. The muscular fibres are more firmly united to each other than in the healthy heart.

3d. Both between and within the muscular fibres are innumerable molecular granules, chiefly proteine.

4th. Lymph which has a granular form exhibits in the heart, as elsewhere, a tendency to undergo fatty degeneration, and the muscular fibres of the heart, damaged by the congestion and the presence in and among them of the lymph, are prone to the same change; the consequence is, that it is common to find here and there a considerable number of particles of free oleine as well as muscular fibres which have undergone fatty degeneration.²

5th. In some cases the cellular or connective tissue seems to be increased in quantity.

When the exit of the blood from the right auricle is suddenly or greatly impeded, it is not unusual to find small

¹ Laennec described several of these characters as proper to the dilated and hypertrophied heart. *De l'Auscult. Med.*, vol. ii. p. 154.

² When the fatty metamorphosis of the muscular tissue and lymph is extensive and extreme, softening of the walls of the heart may ultimately occur; the parietes of the heart being first indurated, and subsequently softened, in consequence of the indurated tissue undergoing fatty degeneration.

crimson spots studding the external surface of the heart, and less commonly the internal surface. These spots are evidently extravasations of blood, the result of capillary hæmorrhage from mechanical obstruction to the onward flow of the blood.¹ Under like conditions there is often found a considerable excess of serosity in the pericardium, and œdema of the loose cellular tissue at the base of the ventricles; the latter especially, if the patient is much emaciated.

When the congestion has been extreme and long-continued, the coronary sinus is found to be more capacious than natural.

In studying the effects of congestion on tissues and organs, it is necessary to separate not only slight from extreme congestion, but also continuous from intermitting congestion, suddenly formed from gradually formed congestion, and congestion of organs the action of which is normal or increased from congestion of organs, the functions of which are less actively performed than in health.

It is the very gradually developed, long-continued, intermitting congestion of organs, the functions of which are over-actively performed notwithstanding their congestion, which is especially followed by induration, toughness, and hypertrophy.

The causes which lead mechanically to an accumulation of blood in the right cavities of the heart have been attentively studied, and are well known. They are referable either to disease of the arteries of the heart itself, or to some cause interfering with the free passage of the blood through the vessels of the lungs.

The following cases are sufficient to *illustrate* the main facts adverted to in the foregoing remarks; viz., the most common causes of mechanical impediment to the passage of the blood through the right side of the heart; the relation between those impediments, engorgement of the right cavities of the heart, and congestion of the walls of the heart; the result of that congestion of the cardiac parietes, viz., induration and toughness, especially affecting the parts most in action, and the influence of induration and toughening of the walls of the heart in the production of permanent dilatation of its cavities.

¹ See Rokitansky, in previous note, p. 199.

CASE.—A woman, æt. 50, suffered during life from general anasarca, symptoms of extreme congestion of the lungs, liver, kidneys, etc. After death, the walls of the heart were found greatly thickened, and the cavities dilated. When the heart was opened the divided walls still retained their rounded form; they did not fall inwards; the substance of the organ was remarkably tough and hard; the mitral orifice was much constricted; the left auricle was scarcely larger than natural, the right was very capacious; the foramen ovale was so patent as to admit the points of three fingers; the right auriculo-ventricular orifice was very large ($5\frac{1}{4}$ inches in circumference); the coronary sinus and veins were considerably dilated. The orifice of the aorta seemed of normal size, it measured $2\frac{5}{8}$ inches in circumference: the orifice of the pulmonary artery was so much dilated as to measure $4\frac{1}{2}$ inches in circumference. There was a large amount of subendocardial and subpericardial extravasation of blood in very thin layers, varying in extent, at some places forming mere specks, at others large patches.

The kidneys were hard, tough, and granular. The liver was remarkably tough and granular, and fibrous at the margin.

The lungs were the seat of pulmonary apoplexy.

When examined by the aid of the microscope, every fibre of the heart was found to be studded, both within and without, with minute granules—the majority proteine, a few oleine. The fibrous tissue seemed to be more abundant than natural. The cross-markings of the fibres were less distinct than are those of the fibres of a health heart.

Remarks.—The primary disease in this woman was evidently narrowing of the left auriculo-ventricular orifice. The blood, thus prevented from readily passing into the left ventricle, must have exerted abnormal pressure on the septum auricularum; as a consequence, it is probable that an obliquely patent foramen ovale, such as is so often found when no admixture of the blood occurs, became dilated. The pressure on the two sides of the septum being unequal, the blood must have passed from left to right auricle, thus accounting for the difference in the relative size of the two auricles. The large size of the pulmonary artery was, doubtless, due to the impediment to the escape of blood from the pulmonary veins, and to its passage through the lungs.

These changes, as well as the condition of the liver and

kidneys, point to the long continuance of the impediment to the circulation; the large size of the veins of the heart, and the extravasation of blood under the visceral pericardium and under the endocardium, show the degree to which the substance of the heart must have been congested during life; the toughness and hardness of the tissue of the walls of the heart were singularly well marked.

CASE.—J. S—, æt. 35, a man of intemperate habits, by trade a carpenter, was admitted into University College Hospital, November 14th, 1860.

On September 6th he had an epileptic fit. On the 16th he was seen by Dr. Coghlan, of Notting Hill, who found, in addition to much cerebral disturbance, a loud, soft, blowing systolic cardiac murmur, having its point of greatest intensity at the apex; the heart's impulse abrupt and irregular in force and frequency; the pulse at the wrist so rapid, weak, and irregular, that it could not be counted. No anasarca.

Against the advice of his medical attendant the man resumed work, and when again seen he was suffering from extreme dyspnoea and extensive dropsy.

On admission into the hospital, about six weeks after the supposed commencement of his illness, the most prominent symptoms were extreme dyspnoea, anasarca, and ascites.

All the superficial veins were distended with blood. The jugular and subclavian veins became distended to the utmost when the man coughed; they pulsated synchronously with the heart's beat; a strong thrill was perceptible by the finger placed over the point of junction of the right internal jugular and subclavian veins. On slight pressure both the pulsation and the thrill ceased.

Physical signs indicated the existence of hypertrophy and dilatation of the heart, and regurgitation of blood through the right and left auriculo-ventricular orifices. The urine contained a considerable quantity of albumen. The congestion of the various organs and tissues diminished very greatly during the first few weeks of the man's stay in the hospital; so much so that the anasarca almost disappeared; there was scarcely a trace of albumen in the urine; the pulse fell to 96, and was more regular in force and frequency, and the man could lie on his back, and walk about the ward without difficulty.

For a fortnight before the man's death, on January 4th, 1858, however, the distension of the venous system was even greater than on his admission.

The larger veins were abruptly dilated at intervals, indicating the situation of their valves. There was distinct pulsation of

cardiac rhythm in the veins of the arm ; pressure on a vein stopped the pulsation on the distal, but not on the proximal side of the point of pressure ; the pulsation, therefore, was not transmitted through the capillaries. The thrill in the veins at the root of the neck was very perceptible to touch, it was synchronous with each beat of the heart. A loud systolic murmur, evidently generated in the veins, was audible at the spot where the thrill was to be felt. There was neither thrill nor murmur in the carotid or subclavian arteries.

At two points, where the bulging of the brachial vein attained the size of half a broad bean, the pulsation of the vein was perceptible to touch as well as eye.

There was orthopnoea, extreme anasarca, and ascites.

After death about eight ounces of reddish serosity was found in either pleura, and ten ounces in the pericardium.

The heart was much larger than natural. The right auriculo-ventricular opening measured $6\frac{1}{4}$ inches in circumference. The left auriculo-ventricular opening was so much contracted from old disease of the mitral valve, that even the points of two fingers passed through it with difficulty. All the cavities of the heart were much dilated ; that of the left ventricle, however, less so than the others.

The walls of the heart were thicker and much harder and tougher than natural. When cut across they did not fall inwards, but the cavities still retained their rounded form unchanged.

The coronary sinus was very capacious ; the right internal jugular vein was enormously dilated ; the valves at its orifice were perfect, as were the valves of the veins of the arms. There was no disease of the aorta itself, and only adhesion to a trifling extent of two adjacent aortic sigmoid valves to each other.

The aortic orifice measured 3 inches in circumference. The pulmonary artery measured $3\frac{1}{2}$ inches in circumference.

The liver was small, but singularly tough ; it was strongly granular ; the tissues of the portal canals were especially thick and tough ; the acute margin of the organ was reduced to fibro-cellular tissue. The kidneys were exceedingly hard and tough, and finely granular. The lungs were the seat of pulmonary apoplexy and congestive pneumonia.

Remarks.—The starting-point of the disease of the heart in this case evidently was at the mitral orifice ; to this all the other diseased states of the organ were mechanically referable. The disease of the mitral orifice was doubtless, notwithstanding the history, of long standing.

How extreme must have been the impediment to the passage of the blood through the right side of the heart, was shown by the remarkable fulness of the superficial veins. The distension of the right jugular vein was so great that the valves at its opening into the subclavian vein no longer sufficed to close the aperture, and regurgitation was permitted; in the same way as regurgitation was permitted through the dilated right auriculo-ventricular orifice from insufficiency of its healthily sized valves to close the abnormally large aperture. The thrill felt at the root of the neck, and the systolic murmur heard at the same point, were evidently due to the same cause, viz., to the flow of blood past the margin of the valves into the dilated jugular vein.

That the same cause which had led to the distension of the visible veins, viz., the impediment to the escape of blood from the right side of the heart, had also led to distension of the veins of the heart, was shown by the dilatation of the coronary sinus and the amount of serosity in the pericardium. The condition of the liver and kidneys indicated that they were the seat of exudation of contractile lymph, favoured by, if not the direct result of, their mechanical congestion. The induration and the toughness of the heart were so great that the attention was at once arrested by them.

CASE.—T. B—, æt. 9. This boy had from a very early period of his life been the subject of well-marked cyanosis, and some time before death suffered from a considerable amount of anasarca.

After death, in the *pericardium* was found rather more than two ounces of transparent serosity, and its tissue was more opaque and thicker than natural. Here and there beneath the visceral pericardium were crimson spots due to extravasation of blood; there was a good deal of serosity in the loose cellular tissue at the base of the heart, i.e. between the auricles and ventricles.

The *right auricle* and *venæ cavæ* were filled with recent clots and fluid blood. In the appendix of the auricle was an old clot, and a clot also of some age was interlaced among the *columnæ carneæ* of the *right ventricle*.

The *left auricle* contained much fluid blood, and in its appendix was an old clot.

The *left ventricle* was almost filled with old clot; at first sight it looked as if a fungous growth was sprouting upwards from the apex. A recently formed clot extended from the old clot into the aorta.

The right and left sides of the heart, both auricles and ventricles, were greatly dilated, and their walls thickened. The *valves* were quite healthy.

Dimensions of orifices—aortic, $1\frac{7}{8}$ inch; pulmonary artery, $2\frac{1}{2}$; left auriculo-ventricular, $3\frac{1}{8}$; right auriculo-ventricular, $4\frac{1}{8}$. The weight of the heart, with the old clot in the left ventricle, $7\frac{3}{4}$ ounces.

The *coronary sinus* was of very large size.

The liver, spleen, and kidneys were all remarkable for their hardness and toughness.

As far as could be ascertained this boy had been the subject of cyanosis from his earliest infancy. The long duration and the extreme degree of the impediment to the circulation through the lungs and right side of the heart were shown by the large size of the pulmonary artery and tricuspid orifices and of the coronary sinus, as well as by the state of the liver and kidneys. That there was no great cardiac impediment to the circulation at the time of birth was proved by the closure of the foramen ovale. The sub-pericardial hæmorrhage, and the œdema of the cellular tissue at the base of the heart, indicated a sudden extreme increase in the obstacle to the circulation.

Remarks.—It is probable that the primary affection was endocarditis of the apex of the left ventricle occurring very soon after or shortly before birth; that coagulation of blood on the roughened surface followed, and that to the clot so formed the impediment to the circulation was due. The enlargement, hardness, and toughness of the abdominal viscera evidently resulted from their mechanical congestion. Can we doubt that the extreme induration and toughness of the walls of the heart were due to the same cause?

CASE.—J. R—, æt. 47, a cabman, of intemperate habits, was admitted into University College Hospital, November 17th, 1857, suffering from extensive and extreme hypertrophous emphysema of both lungs, capillary bronchitis, and hypertrophy and dilatation of the heart, more especially affecting its right side.

The man stated that he had been for many years subject to winter cough, and that he had previously had five or six attacks of bronchitis, the last about a year ago; that his breath was always short, and his face and lips dusky purple in colour; that a year since he first observed that his legs were swollen, and shortly after that his abdomen was larger than natural; that he vomited a little blood two years ago, had often coughed up mucus streaked with blood, and had suffered repeatedly from epistaxis.

He dated his present illness from the 9th of November, when he had rigors, followed by heat of surface, thirst, increase of cough, and dyspnoea, and was obliged to take to his bed.

When I saw him after he entered the hospital he was suffering from extreme orthopnoea. The whole surface was livid. The vessels of the conjunctivæ were dilated and filled with blood; the eyeballs were prominent, the lower lip swollen and everted. The large veins of the neck, which during inspiration were scarcely perceptible, were distended during expiration, but did not pulsate. The heart's sounds were not audible, though its pulsations were perceptible below the ensiform cartilage. There were present all the physical signs of extreme extensive hypertrophous emphysema. There was some anasarca. The pulse was 132, very small and weak; the respirations were 38 in the minute. The urine contained a large quantity of albumen, and some small waxy casts.

On the 18th of December the colour of the man's face, trunk, and extremities was darker, *i.e.* he was more cyanosed than I ever saw the skin of a patient who was not the subject of congenital heart disease.

The lividity varied in degree during the succeeding fortnight. On the 28th the veins of the neck were constantly much distended, and pulsated; there was a large quantity of fluid in the peritoneal cavity; the anasarca had increased in amount; there was a systolic murmur audible at the base of the sternum, *i.e.* just above the ensiform cartilage.

On the 30th my notes were—Cyanotic symptoms more marked than at any time since admission. Heart's beat lower. Left lobe of the liver considerably depressed by the manifestly over-distended right side of the heart and the emphysematous lungs. Veins of neck extremely full, knotted, pulsating.

The man died on the 31st.

On examination after death there was found extreme hypertrophous emphysema of both lungs; great hypertrophy and dilatation of the right side of the heart, moderate hypertrophy and dilatation of the left side. The walls of the heart were indurated and very tough; this change affected the right side infinitely more perfectly than the left. The muscular tissue of the right side of the heart was very compact, and the cut surface particularly smooth and homogeneous in appearance. The mitral orifice measured $4\frac{1}{2}$ inches in circumference; the tricuspid, $5\frac{1}{2}$ inches; the aortic, 3 inches; the pulmonary artery, $3\frac{1}{2}$ inches. The coronary sinus admitted the little finger with facility; it was evidently much dilated. There was general anasarca, and a considerable amount of fluid in the peritoneal cavity.

The liver was enlarged, it weighed 52 ozs. It was uniformly and finely granular; it was hard and very tough. The

kidneys were large and irregularly contracted, so as to be coarsely, unevenly granular on the surface. They weighed 6 ozs. each.

Remarks.—This case affords a good example of the most common causes of over-distension of the right cavities of the heart, and therefore of congestion of the walls of the heart, and, as a consequence, of induration, toughening, and permanent dilatation of the heart: viz., repeated attacks of bronchitis and hypertrophous emphysema. The congestion of the whole venous system showed the great impediment that existed to the flow of blood through the right side of the heart; and the extent to which the capillary system had suffered dilatation proved the length of time that the impediment had existed. The anasarca and the large size of the coronary sinus supported the same inferences.

The liver and kidneys were all enlarged, hard, and tough—changes doubtless chiefly due to their long-continued mechanically produced congestion. The fluid in the peritoneum and the albumen in the urine were also, probably, due to the obstacle to the return of blood from the liver and kidneys. The induration of the heart was very decided; the specific gravity of the most markedly indurated part of the right ventricle was taken for me by Mr. Russell, in Professor Williamson's laboratory; it was 1.053.

The very trifling evidence of old pulmonary collapse found after death, and the enormous extent and degree of the emphysema, conjoined with the fact that, during life, there was very forcible protrusion of the intercostal spaces above the level of the ribs, and equivalent prominence of all the soft parts of the thoracic parieties in violent expiration, told strongly in favour of the expiratory theory of emphysema laid before the society by myself in 1857.

CASE.—Robert W—, aged two years, was a healthy child till the commencement of 1858, when, his mother said, he suffered from bronchitis. During that attack she noticed that his feet were swollen. All œdema disappeared in the course of the ensuing spring, but he never recovered his previous state of health.

The child was admitted into the Hospital for Sick Children on January 30th, 1859—his mother stating that his present illness was of only three days' duration.

There were present the physical signs of general but imperfect solidification of both lungs; strongly marked pulsation in the jugular and subclavian veins; a little lividity of the lips; œdema of legs, hands, and face. The child was seized with convulsions on the 1st of February, and died.

After death the whole of both lungs were found to be loaded with grey granulations and yellow tubercles; and here and there, in addition to much collapse, were nodules of lung solid from pneumonia.

About two drachms of serosity were found in the pericardium; the veins on the surface of the heart were filled with blood.

The pulmonary veins, the venæ cavæ, both auricles, the right ventricle, the pulmonary artery, and the aorta contained a large quantity of dark, loosely coagulated blood. The walls of the right ventricle were only slightly thicker, but were much tougher and harder than natural. The walls of the left ventricle were much less tough and hard than were those of the right ventricle, and its cavity was not dilated.

Examined by the aid of the microscope, the muscular fibres of both ventricles were found to be studded within and without by proteine granules. A few of the muscular fibres were in a state of fatty degeneration. The most completely degenerated fibres were found in the muscular band which passes from the right wall to the septum ventriculorum.

The spleen, liver and kidneys, were all loaded with blood, and tougher than natural.

Remarks.—Here the primary impediment to the circulation of the blood was seated in the lungs. Bronchitis, pulmonary collapse, emphysema, and an enormous accumulation of tubercles in every part of the lungs, were the causes of the obstacle; the degree and duration of the impediment were indicated by the dilatation of the right auriculo-ventricular orifice, by the state of the spleen, of the liver, of the kidneys, and of the veins, and by the anasarca.

The texture of the heart was damaged in the same mode and from the same cause as was the texture of the liver, spleen, and kidneys, *i.e.* from mechanically produced venous congestion.

CASE.—E. B—, æt. 8 years and 9 months, was admitted into the Hospital for Sick Children on September 10th, 1859. In November 1856, she suffered from severe rheumatic fever, and from that time had been obliged to cease her occupation,

viz., that of a tight-rope dancer. When she came under my care she was suffering from extreme anasarca, ascites, great fulness and pulsation of the veins of the neck and upper extremities, and orthopnoea. The chest was in form the type of that which accompanies great hypertrophy and dilatation of the heart in a child. The physical signs indicated very plainly the lesions found after death.

She died October 3d, 1859; the body was examined twelve hours after death. The sternum being removed, the tissues of the mediastinum were found to be much more vascular than natural, and, in common with the cellular tissue at the root of the neck, were infiltrated with serosity, and remarkably tough. There were about four ounces of transparent serosity in the pericardium. Numerous crimson spots, due to sub-pericardial extravasation of blood, were present at the base of the heart, especially about the root of the pulmonary artery; the loose cellular tissue between the ventricles and auricles contained much serosity. Both auricles contained very large coagula; that in the right auricle was continuous with a clot in the coronary sinus. The latter clot, when removed from the sinus, measured 3 inches in length. The veins on the surface of the heart were gorged with dark-coloured blood. The walls of the heart were greatly hypertrophied, especially those of the right ventricle; they were remarkably hard, tough, and dark in colour. The tissue of the walls was more compact, its cut surface smoother, its edges sharper, and its aspect more transparent than natural. All the cavities of the heart, excepting that of the left ventricle, were extremely capacious. The left auriculo-ventricular orifice was greatly reduced in size, from disease of the mitral valve; the right measured $3\frac{3}{8}$ inches in circumference; the aortic orifice, $1\frac{7}{8}$ inch; the pulmonary artery, $2\frac{1}{2}$ inches.

The liver, spleen, and kidneys were all loaded with blood, and very tough.

Examined by the aid of the microscope, the muscular fibres of the heart were found to be paler, tougher, and more closely united to each other than natural; they were covered with very fine molecular granules; their transverse striæ were very imperfect. There appeared to be an excess of fibrous tissue in the walls of the heart.

Remarks.—The primary disease in this case was evidently constriction of the mitral orifice, and the duration of the disease three years. The toughness, etc., of the cellular tissue, the state of the liver, spleen, and kidneys, the dilatation of the pulmonary artery, the serosity in the pericardium, the fluid in the cellular tissue at the base of the heart, the

dilatation and engorgement with blood of the coronary sinus and the veins on the surface of the heart, and the sub-pericardial extravasation of blood, were all evidently due to mechanical impediment to the passage of the blood through the left auriculo-ventricular orifice.

The change in the texture of the heart was, as in the preceding, the result of the same cause that produced the change in the texture of the liver, spleen, and kidneys; and the change was of the same nature; only, in the case of the heart, the call on the organ to act was more powerful than in health, and the impediment to the onward flow of the blood necessarily induced over-distension of the cavities behind the impediment.

CLINICAL LECTURES ON THE
DIAGNOSIS OF EXTRAPELVIC TUMOURS
OF THE ABDOMEN

(1868-9)

CLINICAL LECTURES ON THE
DIAGNOSIS OF EXTRINSIC TUBERCLES
OF THE LUNGS
(1880)

CLINICAL LECTURES ON THE DIAGNOSIS OF EXTRAPELVIC TUMOURS OF THE ABDOMEN.¹



LECTURE I

Methods of Clinical Observation—Physical Examination of the Abdomen—Elements of Diagnosis—Illustrative Cases.

THE aim, gentlemen, of clinical teaching is to make the student practically acquainted with medicine. You are elsewhere taught the theory of medicine; here we strive to make you practitioners. In the course of time, every one who practises his profession acquires a certain amount of individual knowledge—personal knowledge; *i.e.* gains at the bedside a certain knowledge for himself. Knowledge so gained is experience. We say ‘A. is a more experienced man than B.’—meaning that A. has more knowledge acquired at the bedside than has B. Now, the amount and worth of the knowledge so gained is not dependent on the number of sick people of whom A. or B. has had the charge. There are men who have had a great deal of practice, and are still inexperienced. They have failed to acquire from the cases they have had under their care, from the practice they have had, any great amount of certain personal knowledge. On the other hand, there are men who have had the care of comparatively few cases, and yet have learned so much practically from these cases as to be, comparatively speaking, experienced men.

There is a great danger in young men having the care of

¹ *British Medical Journal*, 1869, vol. i.

many patients before they have acquired a certain acquaintance with the general doctrines and principles of medicine, and been well trained to observe at the bedside. Because they have treated many people, and comparatively few of their patients have died, they are satisfied with their imperfect knowledge, speaking confidently of the results of their experience, and of the cures they have effected. In truth, such men mistake self-deception for experience.

Now, clinical teaching should enable the student to start from a higher point of experience than he could attain by his unaided powers. The knowledge which the teacher has acquired by the practice of his profession should be, to a considerable extent, imparted to the scholar. The clinical scholar should be taught by his master to see with his eyes, to touch with his hands, to hear with his ears, to reason with his brain; he should, in fact, be put into possession of that practical knowledge which, in long time, the master has acquired for himself. And should be, by his master's aid, possessed of the method by which practical conclusions, from facts observed, are to be drawn. At the end of his pupil career, then, you will see, the clinical scholar should start from a vantage ground; and not only should he start from the practical position his teacher has gained, but should also have been trained to use his senses so as to gather real facts for himself, and to use his reason so as to draw sound conclusions, to make every case which he subsequently sees add to his real practical knowledge when he attends a case, even though it be of the commonest disease, he not only sees the case, but he learns something, adds something to his experience from it. He has learned to see without his master's spectacles, and to draw conclusions without the aid of his master's brain. Some men have a great store of practical knowledge, though they cannot impart it to others. They cannot impart it for this reason; namely, they have never analysed the process by which they gained it. They have made the conclusion part of themselves; the steps by which they reached that conclusion have long since escaped them, if they ever were cognisant of the separate steps. A very able physician, from whom I learned much, was going round the hospital when I was here a comparatively young man, in

secondary charge of patients; pointing to a woman who lay on a bed very ill, he said, 'Take care of that woman; her back will slough.' I asked, 'Why will her back slough?' 'I don't know,' was the answer, 'but she looks as if it would.' Now, that man had practical knowledge; but he could not impart to his scholar the grounds for his experience or acquired knowledge. He could not analyse the mental process by which he had arrived at the conclusion that the woman's back would slough. As the woman's back did slough, I was much impressed by his remark; but I still wanted to know why, and I analysed the process, by which I subsequently came to the conclusion, that the backs of such like people were likely to slough. Though the patient was young, she was fat, and therefore heavy; she was flabby and anæmic, and therefore of low vital resisting power; she was ill from an illness of a low type, in which sloughing is common; she was lethargic in habit, phlegmatic in temperament, and therefore little likely to suffer from a little pain; sure to lie long on her back, therefore she was likely to slough. He said to me 'such-looking people do slough;' but could not make clear to me the separate links in the chain of evidence which led him to his conclusion. Now, I desire, as one of your clinical teachers, that you and I should note in every case the separate steps by which we reach the top stone of our conclusions—at least, so often as this is possible, which is not always.

There are two ways of teaching clinically, and we shall follow both. One at the bedside; one by special lectures, given either in the ward, in the dead-house, or in this theatre.

Clinical instruction must be given at the bedside. There you must note the aspect of the patient, learn to use your ears, your fingers, your eyes, your nose, all the senses, and all the sense that you have. The clinical teacher must tell you what you are to feel, hear, see, and smell; how you are to use your senses that they may convey to you real knowledge; and he must train you at the bedside to note the daily variations in the symptoms, and tell you what those variations signify—how to draw conclusions from the facts you have noted. He tells you what to observe; he teaches you how to observe;

and he explains to you the signification of the facts you have observed.

In clinical lectures, we review the observations we have made from time to time at the bedside; we weigh the facts noted in the wards; we consider individual cases at length, and, by the progress of the case, or by the facts revealed to us in the dead-house, we verify our first opinions, or we discover our mistakes; and, in the last case, we learn why we were wrong, and so add to our experience, and avoid like mistakes on future occasions; we group cases of the same disease, and note in what points they agree, and how they differ, and endeavour to ascertain the causes of their differences; or we may abstract single symptoms present in a number of different diseases, and consider that special symptom, and endeavour to show why it is common to several pathological lesions—for example, jaundice, convulsions, delirium. Again, in the lecture-room, we bring together remarks made in the course of clinical instruction, arranging them in a more or less systematic manner. And it matters little though one repeats here what has been said at the bedside. It is a good thing for the student if his clinical teacher says the same thing in new words again and again; repetition is the soul of teaching, and especially of clinical teaching. With reference to pathological anatomy to chemical research, to microscopical observations, I shall keep to such points only as I hold to be of practical importance—such, that is to say, as I myself use in daily practice.

To-day, I shall say a few words about the Diagnosis of Abdominal Tumours.

When engaged in practice you will often be consulted because the patient has a large abdomen. Do not forget
✓ that a little child in health should have a big belly. It has often happened to me to have a perfectly healthy child brought to me to be treated because its mother thought its belly large. Those of you who are fond of art may have noticed that the good artist, when painting a little naked child, puts it in such a position as to hide the protuberance of its belly, which is not a pretty object: if necessary to represent it in a position in which the belly is seen, as in

some of Raphael's pictures, you see that the child has a big belly. When an artist paints a naked child and makes a pretty object of its abdomen, you know that he has made a mistake; he has sketched his ideal of what a child should be, and not a child of nature's making. The little child's abdomen is large because its abdominal and intestinal muscles are weak, its pelvis is shallow and small, its diaphragm flat, and its liver and spleen large; and because much flatus is formed in its small intestines, especially during the digestive process. Again, remember that fat-forming persons, at all advanced in life, have big bellies. The large abdomen here is due to fat upon the parietes, fat in the mesentery, omentum, etc.; and again, people whose abdominal parietes are thickly coated with fat, and who have a large quantity of fat in their abdomen, are very liable to have accumulation of flatus in their intestines. Enlargement of the abdomen, due to flatus, increases and decreases suddenly. The size of the abdomen varies from day to day, and at different times of the day. No other enlargements of the abdomen thus vary in degree. ✓

A word here by way of caution. Persons will consult you, saying, 'I have been growing bigger lately,' and referring to their increase in size as if they attached little importance to it. Ladies of middle age are especially likely thus to mislead you; though very anxious in mind because of the increase in size, they do not like to admit even to you, their doctor, that they are anxious or care in the least about it. Do not be misled by this seeming indifference into giving an opinion without an opportunity being afforded to you of forming a correct opinion by examination. It is not long since that a lady, consulting me, referred, as if incidentally, to her increase in size, saying, 'It is really of no consequence, I suppose. The swelling is sometimes trifling, and then considerable.' 'It may be, and probably is, of no moment,' I replied; 'but I cannot say without examining.' 'Oh, I don't think that is necessary,' she said. In a few days, however, she came to me again, and said, 'Well, I think I should like to be quite sure.' I found a tumour of moderate size rising out of the pelvis. Now this lady would have liked me to give her a favourable opinion without examining. I

should have pleased her by such opinion for the moment, but ultimately I should have had great discredit.

Remember that many more mistakes are made from the practitioner not examining, than from his not knowing. The great distension complained of by the patient may be the immediate consequence of accumulation of air in the intestines, and yet the patient be the subject of abdominal tumour. It was so in the lady to whose case I have just referred. A moderately sized tumour in the abdomen may cause great discomfort when the patient has, in addition, an accumulation of flatus. When the flatus disperses, all discomfort ceases. The distension comes and goes quickly, which no other distension than that due to flatus does; and the great swelling thus coming and going, you may be led to overlook a serious disease. It may be that the patient would not have discomfort, would not notice any increase in size, unless she had the solid growth and the accumulation of air. Her trouble is due to the growth *plus* the flatus. Get rid of the flatus, and the patient is free from present suffering; and so for a while, unless you carefully examine the patient, you may be in error as to the disease.

Speaking generally, we may say abnormally large abdomens are due to accumulations of air, to excess of fat, to the presence of fluid, or to solid tumours. Now supposing that you are afforded an opportunity of examining the abdomen, of passing your hand over it, so as to ascertain whether there is anything there which there ought not to be, in what position do you place the patient while making the necessary examination? I see some of you merely raise the legs; you let the patient be in any way on his back, only you carefully draw up his knees. But consider what is the great obstacle to your examination of the abdomen—supposing the patient to be neither very fat nor very tender. The great obstacle is muscular rigidity. The recti, especially, are the great opponents to a physical examination of the abdomen; and by drawing up the legs you relax the recti very little indeed. But if you place the patient on his back, with the shoulders somewhat raised, and the back of his head propped up till the chin falls on the top of the sternum, you will find that the abdominal muscles are relaxed

considerably; then bend the legs on the abdomen, so as to relax the fasciæ, and perhaps also some of the abdominal muscles a little. It is better when you flex the thighs on the abdomen, to let the feet rest on their soles, and to have the knees supported by the hands of an assistant.

Having made the abdominal wall as lax as possible by position, talk to the patient about some interesting subject, and so engage his attention that he may not, by voluntary action of the muscles, resist your manipulations. And if you cannot divert a patient's attention, make him count, make him count up a hundred, and then, when he has counted as far as he can without drawing in his breath, having kept his muscles tight all the time, he takes one deep inspiration, and while he is doing that, and the muscles are relaxed, you put your hand well down, and keep it down. By giving to the parts under the hand a rolling motion, you often can get deeper than you would by simply forcing the hand downwards. You have thus gained a certain point, and at the next pause your hand will go a little nearer to the spine, and so on till you have thoroughly examined the deeper parts. All these means failing, it may be necessary, but it is very rarely so in practice, to put the patient under chloroform.

Now and then a portion of contracted rectus is mistaken for an abdominal tumour. It is not long since I saw a case of this kind. The patient, an old man, was very ill and greatly emaciated. It was feared that he was suffering from malignant disease, and my attention was directed to what it was supposed might be a tumour in the region of the pylorus. Remembering the possibility of mistaking the upper division of the rectus for an internal tumour, I soon found that this body was exactly in the situation of the upper division of the rectus; that I could not find any other upper division of the rectus on that side; that I could just get my finger under the outer border, and that by making the man raise himself in bed I could feel it contract and grow thicker. I thus satisfied myself that the mass about which the question of malignant tumour was asked was really the upper division of the rectus. The progress of the case proved the truth of the opinion. Again, it was this

morning only I saw a gentleman who was supposed to be suffering from a distension of the gall-bladder. The supposed gall-bladder was proved by such an examination as I have just described to you to be the upper division of the right rectus, somewhat larger than usual, and larger on the right than on the left side; the difference in the size of the upper portion of the right and left rectus led to the error in both cases. This enlargement is more common on the right than on the left side. Supposing that you have relaxed, by position, etc., the abdominal walls; that you have passed your hand carefully over the whole abdomen, and have felt in it a body which you think should not be there, the first question you ask yourself should be: 'Is this tumour due to one of the organs of the abdomen more easily perceptible to touch than usual, one of those organs misplaced, or one of those organs increased in size from disease?' To answer this question, and to determine what organ it is which forms the tumour, you should first endeavour to ascertain whether or not the tumour takes its origin in the pelvis. 'Can you pass your hand between the solid body and the brim of the pelvis?' If a person be not very fat and you have relaxed the abdominal muscles, you can very commonly pass your hand back far enough to feel the pulsations of the aorta over the lower part of the spine just where the vessel bifurcates; and this can be done when a tumour formed by an enlarged extrapelvic viscus occupies a great part of the abdomen. Thus you can separate, in the great majority of cases, with facility the tumours which arise within the pelvis from those that have their origin above it; for remember that tumours due to disease of organs placed above the pelvis do not pass down into the pelvis; and though those that have a pelvic origin come up into the abdomen they do not lose their pelvic connections; so that in the one case, you pass your hand well back till you feel the spine and almost the brim of the pelvis and you satisfy yourself that the tumour is above that; or, on the other hand, you feel that though the bulk is above, yet there is a connection with something intrapelvic. That is the first thing I do in examining a patient with an abdominal tumour. Supposing you are still in doubt, the use of the catheter and vaginal or rectal

examination will rarely leave any doubt as to the pelvic or extrapelvic origin of the tumour. I shall direct your attention to this point again when I am speaking of tumours due to increase in size of the spleen.

We will suppose that you determine, by the manipulations I have mentioned, that the tumour is not pelvic in its origin, *i.e.* that it is not uterine, not ovarian, not connected with the bladder. These come, you know, into other departments of medicine. But let me remind you that you may be consulted by a patient suffering from what he supposes to be an abdominal tumour, which supposed tumour is merely an over-distended bladder. I have twice been consulted by men whose sole disease was distended bladder. Both came from the country to see me for supposed abdominal tumour, and both were cured, I was informed, by the use of the catheter only. It is especially in the diagnosis of tumours of the abdomen formed by the organs situate above the brim of the pelvis that I propose to-day and in the next few lectures to direct your attention: to the diagnosis, that is to say, of tumours formed by enlargement of the liver, the spleen, the kidneys, the suprarenal capsules, the pancreas, and the lymphatic glands, and by dilatation of the gall-bladder, and of a circumscribed portion of the aorta.

First, I shall speak of the subject generally, endeavouring merely to make you understand how you are to determine that the tumour is an enlarged liver, spleen, etc.: and subsequently teach you how to distinguish from each other the special diseases of the several organs which cause their enlargement. I shall illustrate this subject by notes of special cases.

In regard of the diagnosis of an abdominal tumour originating above the brim of the pelvis, you first ascertain the position it occupies, *i.e.* its seat in relation to the normal position of the several abdominal viscera. Be sure to note the influence of the respiratory movements on its position. In the woman with an enlarged spleen now in the ward, you have observed how decidedly the position of the tumour alters with the movements of respiration. The spleen, lying in contact with the diaphragm, descends with each inspiration, and ascends with each expiration. The liver also, in

consequence of its relation in position to the diaphragm, rises and falls with each rise and fall of the diaphragm. The position of the kidneys is more changed by respiration than you would expect. When about to ascertain the size of the kidneys by palpation you make the patient inspire deeply, make him cough two or three times, and then take in a deep breath; and in a large number of cases the kidneys will lie lower than they did: not much lower, it is true, but still so much lower as to make a perceptible difference—enough, it may be, to enable you to feel distinctly what you were unable to touch before. The right kidney is more depressed during deep inspiration than is the left, probably from its relation to the liver. The kidney does not rise at once when the diaphragm ascends, as the liver and spleen do. The ascent of the kidney is effected either by the force of your subsequent manipulations, or by the pressure of the surrounding parts, and the resilience of the tissues in which it is imbedded; and, when its ascent is due to the latter cause, it rises slowly.

The position of an aneurism of the abdominal aorta, of tumours of the stomach, colon, mesentery, omentum, or the glands, is not affected, or, if at all, in the most trifling degree, by the respiratory movements.

Only yesterday morning, a medical friend brought an obscure case to me for an opinion. The man was supposed to be suffering from malignant disease. A tumour could be just felt in the left hypochondriac region. The question put to me was, 'What is that tumour? Is it in stomach, colon, omentum?' etc. Now the tumour ascended and descended so decidedly with the respiratory movements, that I said to myself, 'This cannot be stomach, omentum, or colon. Tumours connected with those organs do not thus rise and fall with the diaphragm. The only part in this situation thus affected by respiration is the spleen.' It was a deformed lump, which, by the touch, one could not have felt sure to be the spleen; but, having regard to the situation, and to the degree in which its position was modified by respiration, the diagnosis at which I arrived was, I have no doubt, correct.

As the ascent and descent of the liver, spleen, etc., during respiration, depend on the movements of the diaphragm, you

must not forget that any disease which paralyses its action on the right or left side, will prevent the respiratory ascent and descent of the liver, spleen, etc.: *e.g.*, inflammation of the diaphragmatic peritoneum or pleura; cancerous infiltration of the diaphragm.

We will first consider with reference to their diagnosis generally, abdominal tumours due to abnormities in position, form, or tissue, of the liver.

In health, the liver extends to from one to two fingers' breadth below the nipple. The upper border is to be made out by percussion: and a little below the nipple is the spot at which you commonly find a certain amount of dulness on deep percussion. The liver being of its natural size, and in its natural position, the dulness on percussion passes downwards in the nipple-line¹ to the margin of the thorax, and may extend in health to a finger's breadth or a finger's breadth and a half below the margin. In the middle line the liver reaches, it is often said, to half-way between the ensiform cartilage and the umbilicus: but really the lower edge of the organ is decidedly nearer to the ensiform cartilage than it is to the umbilicus.

The less the circumference of the lower part of the thorax, the more arched is the diaphragm, and consequently the more vertical is the position of the liver: the greater must be the extent of dulness from above downwards: the longer, that is to say, is the line of vertical dulness. About one-third of the liver is overlapped by the lung: for, the lung being healthy, a portion lies between the arch of the diaphragm and the outer wall of the thorax.

It is important to bear in mind that although there is dulness on percussion over the liver, and that the dulness is greater the nearer you approach to the margin of the thorax, yet that everywhere, the liver and lungs being healthy, there is a certain amount of resilience of the thoracic parietes covering the liver, on percussion, perceptible to the hand of the observer. You must, at the bedside, make yourselves practically acquainted with this perceptible-to-touch resilience, and know, from your own observation, that in health

¹ By nipple-line is meant a line drawn vertically downward from the nipple.

there is, as I now tell you, a certain amount of elasticity elicited by percussion even over the whole of the liver.

✓ If the liver is enlarged, as in extreme congestion, albuminoid disease, cancer, hydatids, etc., it may, when it projects very little below the margin of the thorax, come too closely into contact with the chest walls. When it does this, it diminishes the resilience of the parietes, damps their vibration. ✓ On percussion, you feel a sense of resistance, and a want of elastic resilience which is not felt in health. Its projection below the margin of the thorax may be trifling, and still the liver may be so much enlarged as to increase very considerably the sense of resistance, to diminish notably the elastic resilience of those parts of the chest-wall which cover it. Whenever this loss of resilience is perceptible, it is evidence that the liver is too closely in contact with the walls.

✓ In health, the margin of the liver cannot be distinctly felt, even though it be proved by gentle percussion to be a little below the border of the thorax. In some cases, you may perhaps be able just to feel a something which you think is the edge of the liver: but you are conscious that you are not sure of it. Your fingers do not come against a positively resisting substance. If the liver can be distinctly ✓ felt, you may be sure there is something wrong: there is disease. This is a point of practical importance. When attempting to ascertain the lower border of the liver by palpation, you press your hand on the hepatic region, and then pass it downwards, depressing the parietes till you reach a spot where there is a marked diminution of resistance. The solid liver gives a sense of resistance to your hand. The air-containing intestines offer very little resistance. When ✓ the sense of resistance ceases, your hand is below the margin of the liver. Now pass the hand upwards, with its outer edge towards the liver: at the same time keeping the hand on as low a level, with regard to the abdominal surface as possible, till you come to something which offers resistance: and then you will feel the border of your hand ride over the margin of the liver. This is the best mode, at least in the majority of cases, of discovering the lower edge of the liver. There is less resistance to your flat hand than there

would be to the tips of your fingers: and, by the outer margin of your hand, you come into contact with a large part of the margin of the liver at the same instant.

When the liver is enlarged, you determine by percussion its upper limit: by the sense of resistance on percussion of the chest-walls how closely it lies in contact with them: by palpation, if the organ be firmer than natural: and by percussion, if its consistence be natural at its lower border. At the same time that you are defining the lower border by palpation you appreciate the degree of resistance that the liver offers, how hard and how elastic it is: at the same time, you note whether the margin is rounded or sharp, even or nodular: having fixed these points in regard of the margin of the organ, you pass your hand over so much of the upper surface as lies below the margin of thorax and note its characters, especially observing its hardness, if it be smooth or nodular. There is, in reference to this last point, a source of fallacy, against which be on your guard. Some portion of the abdominal muscles lie over the liver, and the little irregularities of the muscles may be mistaken for irregularities of the surface of the liver. ✓

The liver may be of natural size but displaced downwards, depressed so far into the abdomen as to form a veritable abdominal tumour. Displacement to a moderate extent downwards is very common, and considerable displacement downwards is common. Do not forget this. I remember a case illustrating the importance of remembering it, which I saw when I first was physician to the hospital. There was evidently a number of things the matter with the man, but there was a question about this and a question about that. The only point—I tell you to warn you from the mistake I made—the only point about which I felt sure was that his liver was enlarged. The man died, and the only thing that was not there was enlargement of his liver. The liver was of natural size, but it was displaced by the enlargement of an organ which is not usually mentioned as a cause of displacement of the liver, but is a common cause of its displacement, I mean a dilated heart. There is a man in the ward now whose liver is displaced downwards by a dilated heart. He has no fluid in his pleura, nothing in his lungs: the greatly

dilated heart pushes his liver downwards. When the liver is considerably displaced and the patient is at all thin, you may see, especially during an inspiration, as you do in the man now in the ward, the upper rounded border of the liver. This is, I think, decisive evidence of displacement of the liver. Had I known this, and looked for it in the case I have just mentioned, I should not have fallen into the mistake.

The causes of displacement of the liver downwards are various. I will enumerate the commonest. A rickety deformity of the thorax, so common in little children. Large-lunged emphysema is a very common cause of a moderate degree of displacement. Fluid, with or without air, in the pleura, is a common cause of considerable displacement of the liver. Then also, as a common cause, we have a dilated heart: less common, fluid in the pericardium. Tumours of various kinds between the liver and the diaphragm, in the substance of the diaphragm, and between the diaphragm and the lung, are still less common causes of displacement downwards of the liver.

LECTURE II

*The Physical Characters of Tumours formed by the Spleen—
by the Mesenteric Glands—by the Pancreas—by
Aneurism of the Abdominal Aorta and its Branches—
by the Kidney.*

IN health the spleen is not perceptible to the touch. If you can feel the spleen, it is diseased. In health, there is a space of about two inches over the spleen, more or less deficient in resonance: this region of imperfect dulness has its lower border near to the eleventh rib: its inner and outer borders correspond to the inner and outer margins of the exillary lines. When the spleen is enlarged, the resilience of the ribs over the spleen is diminished. In some healthy persons the size of the spleen can be tolerably well defined by percussion, in others it cannot. Whether the size of the spleen can or can not be thus defined by percussion, depends on the shape of the thorax, on the size of the lung, and on the degree to which the diaphragm is arched. For on these points will depend whether the spleen lies in contact with the chest walls, or is, to a great extent, borne away from them.

A moderately enlarged spleen forms a tumour in the abdomen situate on the left side, and passing upwards under the margin of the thorax. Even when of moderate size the tumour lies near the surface: there is no intestine in front of it. In these particulars, it differs from the kidney. It has a sharp anterior margin: and in this also it differs from the kidney. The anterior margin of the tumour passes from above downwards and inwards. Sometimes, but only infrequently, one or more notches can be felt in this border: when felt the notch is quite characteristic. The lower border of the splenic tumour is rounded: the posterior border is to be felt, but not so distinctly as the anterior and lower borders. The fingers can, however, always be passed in behind a

tumour formed by an enlarged spleen. You feel the resistance of the spleen as you pass your hand back and back till you reach the space just outside the mass of muscles that lie in the spinal groove, and there you find a narrow space where there is very little resistance: the anterior border of this space is formed by the posterior border of the spleen. Having one hand on the posterior border of the tumour, and the other in front on the anterior edge of the tumour, you find that it is movable from side to side. You can push the anterior border towards or away from the middle line: you can move the spleen thus when it is enlarged because it is very rarely indeed closely adherent. You know how loosely the spleen is attached by its mesentery: and it is infinitely rare for inflammation of the capsule of the spleen to be followed by adhesions sufficiently short and numerous to destroy its mobility. If a large liver chafe against a portion of the thoracic parietes as it rises and falls during respiration, the irritation usually produced leads to adhesions. The same is the case with the masses of calcareous matter, or even induration-knots, produced occasionally in the lungs, which chafe up and down against the ribs during inspiration and expiration, and give rise to adhesions. But the big spleen has merely a thickened white patch at the spot where it rubs against the ribs or margin of the thorax, as it rises and falls with the rise and fall of the diaphragm. This white patch may be even a quarter of an inch in thickness. The white patch so common on the convex surface of the indurated or enlarged spleen, the white patch less common on the liver, the adhesions over certain lung-indurations, and the white patches so common on the heart, are all equally due to attrition.

Whenever the spleen is considerably enlarged, it passes down below the margin of the thorax. A moderately enlarged spleen may not: for the organ may be kept in place by the costo-colic ligament. You know that fold of peritoneum, which English anatomists talk so little about, but which is so important to us as physicians, the fold of peritoneum which passes from the left angle of the transverse colon to the last rib: so marked in the child, and which remains in some adults as a fold of peritoneum of some

breadth: it did so in the woman whose *post-mortem* examination we made on Saturday. When the spleen enlarges it rides over the anterior border of that ligament, and is thus carried forward more than it otherwise would be. The kidney, as I said, always lies close to the middle line, and there is never any space between it and the mass of lumbar muscles. There is always space between the spleen and those muscles: and one reason for this is that the costo-colic fold of peritoneum bears the spleen a little forwards. It is that also which gives the direction (oblique and inwards) to its anterior edge or, at any rate, assists in doing so when the spleen is sufficiently enlarged to ride over its anterior border.

As the spleen nearly always descends below the margin of the thorax when it is much enlarged, if you have a considerable degree and extent of dulness on percussion in the situation of the spleen, you would examine very long and carefully before you concluded the dulness to be due to the spleen. You would first exclude all other things, because, with such an amount of dulness, the spleen ought to descend and to be perceptible to touch—ought *certainly to be felt at the termination of deep inspiration, i.e.* when it is forced down to its lowest level by the descent of the diaphragm. I have seen cases, in times gone by, in which I should have been saved from falling into error if I had known this. For instance, I remember one case well. A gentleman was supposed to have an enlarged spleen. There was nothing abnormal to be felt in the abdomen, but there was dulness in the situation of the spleen. It proved, on *post-mortem* examination, that the dulness instead of being due to the spleen was caused by a large cancerous mass in the fundus of the stomach. It was the fundus of the stomach which was the dull part. Still, a moderately enlarged spleen may not descend low enough to be perceptible to touch: and in very rare cases a spleen considerably enlarged *may* not descend. I made a *post-mortem* examination in the hospital some time since of such a case: the lowest point of the spleen adhered to the stomach, and so the organ was kept up above the margin of the thorax.

In consequence of the position of the spleen and of its anatomical relation to the diaphragm, the spleen is one of the

organs most affected by respiration: it moves up and down most markedly with each ascent and descent of the diaphragm. It is the friction produced by this movement that so often leads to the white patches on the enlarged spleen from thickening of the splenic capsule. The woman whom we examined the other day had one.

A difficulty is sometimes thrown in the way of diagnosis of splenic tumour by enlargement of the liver. It is commonly said to exist when the left lobe of the liver is enlarged: but whenever the left lobe of the liver is enlarged sufficiently to cause difficulty in diagnosis, the right lobe is also enlarged. The liver then extends very considerably to the left and underlies the spleen: so that there is a little difficulty in feeling the anterior edge of the enlarged spleen. In such cases there may be a practical difficulty in the way of diagnosis. If in doubt endeavour to find the edge of the spleen by passing your left hand from right to left: you will find it ride over something where there is a little elevation. Then, by putting the right hand behind the spleen and tilting it well forward you may move the anterior edge over the surface of the liver. Again, make the patient breathe deeply, and you may possibly find the spleen descending more than the liver. Once have your mind awakened to the possibility of confounding the left lobe of the liver with the spleen and I do not think you will fall into the mistake.

When the spleen is enlarged from acute disease it is almost always tender, as in continued fevers. In splenitis from pyæmia the spleen is tender: but the patient is commonly so ill from the primary disease and the earlier established secondary lesion, that the splenic affection is overlooked. The spleen is very rarely if ever tender when enlarged from chronic disease of its substance. At the same time that you are ascertaining the size, edges, and movability of the spleen by palpation, you appreciate its thickness, its consistence, its elasticity, and whether the surface is smooth or nodular.

An abdominal tumour may be caused by enlargement of the mesenteric or other lymphatic glands. There is no mistake more commonly made than that of attributing the big belly of children directly to enlargement of the mesenteric glands. Let it be impressed on your minds that I, with ten years

experience of the Children's Hospital, and no little experience among children since, do not remember ever to have seen a child whose large abdomen was directly due to enlargement of the mesenteric glands. So enlargement of the abdomen *directly* resulting from so-called mesenteric disease—*i.e.* tubercular disease of the mesenteric glands, must be uncommon. I have examined a great number of these children after death, and neither during life nor after death have I seen what could be called a big belly which was the direct result of disease of the mesenteric glands. When the mesenteric glands are greatly enlarged from tubercular or other disease, they lie back close to the spine. They do not come forward, they go backward. The mesentery seems to open for them, so to speak, and the enlarged glands lie close to the spine, and in front of them lie the intestines. The big belly is the result of imperfect digestion and of the weakening of the intestinal and of the abdominal walls, so that the child makes too much wind, and is unable to expel the flatus from its bowels. The disturbed digestion causes the generation of excess of flatus; the weakened intestinal walls and the weakened abdominal walls do not readily expel it. To detect enlarged mesenteric glands you have to feel for them carefully. The enlarged glands do not come near the surface like the spleen, which you cannot fail to find at once, though you may mistake the nature of the tumour. To feel a tumour formed by enlarged mesenteric glands put the patient on the back in a proper position, and then suddenly depress the walls of the abdomen. You may thus come down, pushing the flatus away, upon a hard mass lying close to the spine. Enlarged glands may sometimes be detected by grasping the two sides of the abdomen between the hands, or between the fingers of one hand; and then by bringing the fingers slowly together, you may at last feel the glands between your fingers or, if greatly enlarged, as an unmovable solid mass. By the manipulation I have described, you may feel very slightly enlarged mesenteric glands—*e.g.*, those the seat of albuminoid infiltration.

The great causes of chronic enlargement of the mesenteric glands are: albuminoid disease, tubercular disease, repeated

inflammation of special sets of glands, malignant disease, and that hypertrophy which is accompanied by excess of white corpuscles in the blood—the glandular form of leukæmia. The albuminoid never attain any great size; you just feel them roll under your finger by manipulating in the way I have described. Distended tumours formed by enlarged lymphatic glands are nodular, the nodules being large and round. This drawing shows how impossible it may be by palpation to distinguish an enlarged gland from a kidney, and how impossible it may be to distinguish enlarged glands from an enlarged pancreas. When the lymphatic glands are large enough from chronic disease to form a distinct tumour, they are fixed, and can be moved neither by respiration nor, to any extent, by palpation. We now and then find a small tumour in the right iliac fossa caused by enlarged and indurated glands. The most common cause of glandular tumour in this situation is one or more attacks of typhlitis.

The healthy pancreas can now and then be just felt in very thin persons with small lax muscles, whose lower dorsal and lumbar vertebræ are somewhat curved forward. I may have satisfied myself that I have felt it half a dozen times in my life. The pancreas crosses the aorta and the spine, and when perceptible to touch, is felt on deeply depressing the abdominal walls about a hand's breadth above the umbilicus, by then rolling the subjacent parts under the hand (the stomach and colon must both be empty). I have known the pancreas of normal size in such persons as I have described mistaken for disease; it has been taken for malignant disease of the transverse arch of the colon, and for an aneurism of the abdominal aorta. The head of the pancreas may be so large, when the seat of cancer or of tubercle (I have seen one case only of the latter) as to form a distinct abdominal tumour. The pancreas in health and in disease is fixed, *i.e.* is unchanged in position by the respiratory movements or by palpation.

✓✓ A distended gall-bladder has, there is no doubt, produced a detectable abdominal tumour able to be diagnosed, but such an occurrence is very rare. I not infrequently see cases in which it was supposed that an abdominal tumour was a distended gall-bladder, but I never saw after death a distended

gall-bladder which had been diagnosed or which had produced a detectable abdominal tumour during life. When of large size the tumour formed by a distended gall-bladder is pear-shaped; its small end is upward. It is seated just under the abdominal walls, at the outer border of the right rectus muscle. It is smooth on the surface, free from tenderness and very movable by palpation from side to side, turning on its smaller fixed upper point. Now and then distinct fluctuation has been felt in it by percussion. Sufficient distension of the gall-bladder to produce a distinct tumour is usually the result of occlusion of the cystic duct and effusion of serous fluid into the cavity of the gall-bladder. Tumours thus formed have attained the size of a child's head. A lesser degree of distension of the gall-bladder may result from occlusion of the ductus communis choledochus and the damming back of the bile into the liver and gall-bladder. A just perceptible projection at the lower border of the enlarged liver to the outer border of the right rectus muscle is not uncommon when the ductus communis choledochus is closed. In the latter class of cases there is intense jaundice; in the former there are no other symptoms than such as may arise directly from the tumour interfering with adjacent parts.

Although, systematically, they ought not to be referred to here, yet, clinically, tumours due to dilatation of the abdominal aorta and its great branches should be mentioned. It is a mistake into which almost all young practitioners fall, to suppose that every pulsatile tumour they feel in the abdomen is a dilatation of the aorta. Remember, that although by no means to be reckoned among what are called rare diseases, aneurism of the abdominal aorta is not at all common; that you will meet with a thousand cases, I was going to say, of strongly beating tumour in the abdomen, before you meet with one case of circumscribed dilatation of the aorta. So, instead of being your first, it should be your last idea, that an abdominal pulsation is due to aneurism. An abdominal aneurismal tumour has two great characteristics, and both being present, you may make the diagnosis; but either of which being absent would prevent you from saying that the tumour was an aneurism. The essential point is that the pulsation of the tumour must be expansile. Grasp

the tumour, and if it expands in all directions you may conclude that it is a true dilatation of the aorta, an aneurism. All tumours that lie in front of the aorta pulsate, and often strongly; they transmit the pulsations of the aorta to the surface, and so feel as if they in their own substance, pulsate. Then again, remember that anæmic women have a strongly pulsating aorta, that persons with aortic regurgitation have a strong pulsation in the aorta, that persons at that period of life when the coats of the arteries are degenerating have a strongly pulsating aorta; and that in certain forms of dyspepsia the aorta pulsates so strongly that patients will come to you for that special discomfort, a beating in the abdomen. Then remember, that if you suspect a tumour to be an aneurism, you feel for the expansile character of the pulsation. Failing to detect that, you pause and look for other causes rather than aneurism, you feel to explain the phenomenon. The diagnosis of aneurism can never be held as *certainly* correct till you have felt this *expansile* pulsation.

Incidentally I may mention—for we shall have to consider the subject again, when we meet with special cases—that, if the stethoscope be placed over the abdominal aorta in front, in some healthy persons, a loud systolic murmur may be heard from the pressure exerted on the vessel. The more anæmic the patient, the laxer the coats of the artery, the more certainly will a murmur be produced. Now, if you are suspecting the existence of aneurism, and hear a *bruit* at the spot, I advise you to do what I always do under these circumstances, viz., listen in the back, examine down the spine; and if the murmur heard in front is generated in the aorta—if it is from an aneurism, for example—you will hear it behind; and behind you cannot press on the aorta yourself so as to cause a murmur, or to intensify by pressure any murmur that may be caused in another way. In this way I have come to a diagnosis which otherwise I should have failed to make. In an anæmic woman there is no murmur heard in the back, though the slightest pressure in front produces a loud *bruit*. The louder the murmur in front, supposing it not to be heard in the back, the less likely is the murmur to be due to organic disease.

I shall now point out to you the general characters of tumours due to the enlargement of another organ, viz., the kidney.

As to the seat of the kidney: the twelfth rib crosses it posteriorly. Formerly physicians determined the size of the kidney by percussion: or rather I should say attempted to make out its size, for I never saw any one who percussed out the healthy kidney. I tried: but I never could succeed: and I do not believe anybody else ever did. But, by palpation, its size in all thin persons may be made pretty certainly: and, if it be enlarged, made out easily. You place one hand, as I have often shown you, at the back of the patient, under the first rib, and outside the mass of the lumbar muscles in the spinal groove. The other hand you put in front of the patient, just over the hand behind, on the right side, just under the inferior border of the liver. Having put your hands in these positions, so as to have the kidney well between them, depress the hand on the anterior wall of the abdomen, as far as possible diverting the patient's attention, and removing the tension of the muscles. Then, having depressed the hand as far as possible, using all possible means to expedite and assist its depression, having brought it down as much as possible, tilt forward the hand that is behind: and in that way the kidney is brought well under the touch of the two sets of fingers—perfectly, in the great majority of cases. ✓

Posteriorly, the region of the kidney is dull on percussion: and there is no line of resonance between the kidney-dulness and the spine. Again, there is no space between the kidney and the spinal groove muscles into which you can dip your fingers with little resistance, as there is between the spleen and the spine: and this is equally true of the healthy and of the enlarged kidney.

I told you that the kidney is moved a little by the respiratory movements. Sometimes it can be moved by the hand, and thus much more frequently than you would suppose. A movable kidney is one thing: a floating kidney is another. We very rarely see or feel a floating kidney. I have never met with one after death, though I have felt in a patient what has been supposed to be one. A floating kidney

is a kidney that has a mesentery—a fold of peritoneum attaching it very loosely to the spine. A floating kidney, therefore, can be moved about to a considerable extent—to the extent of the length of its mesentery. A movable kidney can only be passed up and down a little: it slips a little under your fingers. The cellular tissue in which it is imbedded is loose in structure. If you have ever slipped a pea out of its pod you have felt exactly the sensation that the kidney gives as it moves under the finger: it feels as if it were a smooth body in a smooth sac. It is not really so; but that is exactly the sensation. When the kidney is enlarged by disease, it is rarely movable by respiration or palpation. When chronic changes sufficient to enlarge the organ have occurred, whatever their nature, adhesions sufficient to prevent movement usually form between the capsule and adjacent parts. The loose connective tissue in which the healthy kidney is imbedded becomes condensed.

Remember that people, the lower dorsal and lumbar parts of whose spines are curved well forward, have their kidneys very prominent. Now and then, you may even see their kidneys, the organs being of natural size. This anterior curvature of spine with thin abdominal walls is not very uncommon in women. I remember a case of this kind which I gave to the students to report on, when I was examiner at the University of London. I asked for the diagnosis and treatment: and one of the examined told me that it was an ovarian tumour, and he would cut it out. It was a kidney, I believe, perfectly healthy. It might have been such a kidney, however, as this preparation shows—an hypertrophied kidney, the hypertrophy dating from an early period, being perhaps congenital.

The kidney is rounded laterally, rounded in front, rounded at its inner border, rounded at its upper border, rounded at its lower border. You can feel the lower border, and you can feel the outer border. The inner border is usually lost against the spine: and the upper border cannot be felt unless the kidney is displaced. It is important for you to remember that the kidney has no sharp edges. It is rounded on every side, and, in disease, it never loses this peculiarity. There is a woman in the hospital who has a tumour in the left side of

the abdomen which is her spleen. She has one in the right side of her abdomen which I advise you to feel. I was told that the tumour perceptible in the right renal region was the right kidney. Now, in this woman, probably a condition exists somewhat similar to that which existed in the man from whom this preparation was taken. The kidneys in this bottle belonged to a man who was seen by the late Dr. Bright. Dr. Bright thought that he had a large right kidney. You will note that the right kidney is of the size of a walnut, and the left kidney is considerably hypertrophied. The man died. I made the *post-mortem* examination, Dr. Bright being present. As soon as I had removed the kidneys, he laughed and said, 'Well, I took a piece of the liver for the kidney.' This woman now in the hospital was supposed to have a large right kidney. In her, however, the tumour is, as it was in Dr. Bright's man, a part of the right lobe of the liver. Although not very large, the tumour lies close to the right lateral wall of the abdomen—not a little way from it, as the kidney does: it touches the lateral abdominal wall. It comes outside the ascending colon: while the kidney of course would lie behind and extend a little inside it. Again, in this woman you can feel by careful manipulation, an anterior or inner acute margin. That acute margin altogether and absolutely excludes from the diagnosis enlarged kidney. In this woman, having once the cue to the real nature of the tumour, you can, by putting your fingers under the margin, trace the margin till you feel that it is continuous with the margin of the liver.

What you may mistake for the kidney, and what I have seen mistaken for it, besides this piece of the liver, are, stools in the ascending colon. Let me here, by the way, warn you against an error into which you are likely to fall, and I dare say some of you will fall, even after I thus tell you of your danger. It is that of thinking that abdominal tumours, the nature of which you do not understand, are accumulations of *fæces*. Decided tumours due to *fæcal* accumulation are infinitely rare in practice. Did I ever see an accumulation of *fæces* sufficient to cause abdominal tumour? Well, I have seen one—I may have seen two—but of no great size: and in neither was there any difficulty in determining the

✓ nature of the tumour. But I shall be within the mark, if I say that I have seen a dozen tumours that have been supposed to be faecal, and have seen something else—a cancerous mass—and the woman purged for it: big spleen, and the child purged for it till it had bloody stools. So that you will understand that your tendency to err will be in that direction: and you will bear in mind that a distinct tumour due to an accumulation of stool is rare.

Then, I have known tumours of the suprarenal capsules—a cancer there, for example—mistaken for a tumour of the kidney itself: and I do not know how you are very well to distinguish them by the touch alone. The same is true of a diseased gland.

✓✓ Again, abscess in the vicinity of the kidney may be mistaken for an enlargement of the kidney: but renal tumours never cause enlargement behind. A renal tumour is not visible in the back: it expands in front. A little greater fulness of the loin there may be, but nothing like tumour. This is an important point in reference to diagnosis. In my earlier days I fell into the mistake of supposing two or three cases to be renal, in which there were projections behind, and in which I had felt a great mass in the region of the kidney, and thought, in consequence, it was all renal when it was really outside the kidney. I fell into this mistake because it had never been impressed on my mind that renal tumours do not cause distinct posterior bulging: that tumours due to diseases of the kidney enlarge in front, whilst abscesses and other lesions, which may simulate renal tumours, often cause considerable posterior projection.

Again, in regard of the diagnosis of renal tumours, remember that the colon lies in front of the kidney—the ascending colon in front of the right, the descending colon in front of the left kidney. The proximity to the colon renders the diagnosis between nephritic colic and intestinal colic sometimes difficult. Nephritic colic will cause loss of power in the colon, and so induce constipation: thus favouring the idea that the patient has intestinal colic. Again, collections of stool in the colon may be mistaken for an enlarged kidney. A large enema will solve all doubts on this

point. In all cases of abdominal tumours, the nature and seat of which are obscure, it is right to have the bowels well emptied by a large simple enema and the bladder emptied by a catheter. Remember that a person may have a collection of hardened fæces, and yet be suffering from frequent discharges of small fluid stools.

The pelvis of the kidney often attains an enormous size from collections of fluid in it. The tumour thus formed may pass down into the iliac fossa, and up so as to be indistinguishable by percussion from the liver: but it rarely passes inwards beyond the middle line of the abdomen—never far beyond it, and never into the pelvis. But we will consider this kind of tumour hereafter.

LECTURE III

Diseases of the Spleen causing tumour in the abdomen.

Illustrative cases.

GENTLEMEN,—I have hitherto been describing those general physical characters from which I conclude that an abdominal tumour is formed by increase in size and displacement of some normal part of the abdomen, *e.g.*, liver, spleen. Now I have to speak of particular organs as causing abdominal tumour, and to bring under your notice any special cases of tumours of the abdomen which may illustrate the subject. And while doing this, I shall refer to the various diseases of the organs, but only so much as seems to me to be of practical value, and so much as I think you should positively learn in order to become yourselves practitioners of medicine, and so much as I use in every-day practice.

I commence with the spleen, because we have recently had in the wards some cases of acute and chronic enlargement of the spleen.

The spleen, you know, is a ductless organ; its function is doubtful, its structure, for our purpose as practical pathologists, but imperfectly known. We can go through every part of the kidney, we can distinguish the special diseases of each part, the diseases of its vessels, the diseases of its cells, the diseases of its membranes. Of the spleen, it is not so. Of the kidney, we can tell you to a certain degree the functions of its several parts; of the spleen, we cannot. The spleen has been extirpated in the human subject, and the patient has lived thirteen years and a half, and enjoyed health. The conclusion is that, whatever the functions of the spleen are, some other part of the body can perform these functions. It is in consequence of there being much doubt as to the functions and the structure of the spleen, that its diseases are necessarily ill-defined from each other. Pathological research

has taught us something about the functions of the spleen. There is no question that it is in some way connected with the making or the destruction of blood—of the red corpuscles, and of the white. There is one remarkable disease of the spleen in which there is a great excess of white corpuscles—leukæmia; and in this disease the excess occurs, without doubt, from disease of the spleen; and that not as a remote consequence, but directly from the disease. There seems to be a relation between the function of the spleen and the function of the lymphatic glands; for the same condition—excess of white corpuscles in the blood—exists as the result of the disease of the lymphatic glands, without there being any disease of the spleen. There seems to be a relation, too, between the functions of the spleen and of Peyer's patches, and, it may be, of other parts of the intestinal canal. And you know that there is considerable difference of opinion in regard to their functions. There is nothing distinctly known; but observation of disease lends support to the opinion that the functions of these distant parts are related to each. It is a point of considerable interest, that the spleen, the lymphatic glands, and Peyer's patches all suffer involution at the same period of life—about fifty. At that time, the spleen grows smaller, the lymphatic glands waste, and Peyer's patches smooth down and lose their peculiar structure; and that is about the period of life at which the diseases, and especially typhoid fever, in which these three parts are involved cease to be common. ✓

When considering diseases of the spleen, bear in mind the anatomical relations of the spleen to other parts. Certain diseases of the liver interfere with the return of the blood from all the radicles of the portal vein; hence ascites is a common consequence of many diseases of the liver. Diseases of the spleen cannot interfere in the least with the portal circulation; hence one reason why ascites is no character of splenic disease. And you will remember the very thick capsule of the spleen. This capsule prevents the congested spleen from relieving itself, if I may say so, by the escape of fluid into the abdomen, in the way that a congested lung often relieves itself by fluid exuding into the pleura, or a congested intestinal mucous membrane may relieve itself by

the escape of fluid, causing serous diarrhoea, or a congested peritoneum by effusion of serosity into its cavity. The vessels of the substance of the spleen cannot thus relieve themselves; and this is another reason why ascites does not follow on splenic disease.

You must bear in mind, also, the relation of the spleen to the portal circulation, and so to the liver. The blood returning from the spleen has to go through the liver; hence impediment to the passage of blood through the liver is attended with enlargement of the spleen—*i.e.* with mechanically induced congestion, and the chronic changes consequent on it. Hence, in cirrhosis of the liver, one of the symptoms is enlarged spleen. Hence, in mechanically induced congestion of the liver, and of the radicles of the portal vein—as, for example, in certain forms of heart disease, and in emphysema of the lung—enlargement of the spleen is a common consequence. The splenic blood has to pass through the liver; and anything that leads to overloading of the veins of the liver will necessarily lead to overloading of the veins of the spleen.

Then remember its relation to the stomach. I cannot tell you its exact physiological relation; but we know that it is intimately related anatomically by the vasa brevia; and that, in a certain stage of the digestive process, the spleen increases a little in size. Its mechanical relation to the stomach, too, is important, since vomiting from mechanical disturbance of the stomach is not an uncommon symptom in ✓ splenic enlargements.

✓ Again, it is related in position to the diaphragm; it lies in contact with the diaphragm. In the boy who was recently in the hospital, and who died, you may remember that we found that adhesions had formed between his diaphragm and his lung; and the inflammation had extended through the diaphragm to the peritoneum, and afforded us a good illustration of an inflammation of a serous membrane by extension, and the little tendency that these inflammations have to spread. This latter is a point of great practical importance. You puncture a man's abdomen who has some general disease, and you injure his peritoneum; and inflammation, starting from one point, spreads all over the membrane. In the other case, there is inflammation of the

part of the membrane covering the diaphragm, and it spreads no further. This boy had had pleurisy at some period. The inflammation had extended through his diaphragm; and inflammation of the serous membrane covering the diaphragm, and of that covering both spleen and liver, had taken place, resulting in adhesions. The inflammation did not, you will observe, spread over the peritoneum. I told you, in my last lecture, that adhesions were not so common between the spleen and the adjacent peritoneum as between the liver and the adjacent peritoneum. I referred, not so much to extension of inflammation through the diaphragm, for that is not very uncommon, though it is still more common in the liver, but rather to the friction-adhesions, and to adhesive inflammation of the capsule of the spleen and adjacent parts from disease of the spleen itself. These are uncommon in the spleen. We have in this drawing a good illustration of adhesion of the spleen to the peritoneum; but that is in tubercular peritonitis, where it is common enough to find a spleen united to all the adjacent parts by adhesions.

Then remember that the relation to the diaphragm is such as to give rise to a mistake occasionally. Fluid in the pleura may depress the spleen, and so cause, *it is said*, a splenic tumour. I doubt, however, whether the spleen is ever enough depressed by effusion of fluid into the pleura to cause a tumour.

Then the anatomical relation of the spleen to the adjacent colon is to be remembered. The woman, to whom we shall refer presently, had the colon overlying the lowest part of her spleen; and the spleen was, in consequence, less easily touched, especially when the colon was distended with flatus. It was said, in the notes taken during life, that the woman's spleen varied very much in size. I doubt if it did, judging from the *post-mortem* examination. I think that the spleen was more perceptible at one time than at another. I think, from the anatomical condition, that it was very little temporarily extensible. The differences in the size of the spleen, supposed to have been observed during life, were due to the organ being over-lapped at its lowest part by the colon, and so less easily felt at one time than at another. Pain in the left part of the arch and the upper part of the

descending colon, and pain from stretching of the costo-colic ligament, as well as pain from flatus in the fundus of the stomach, may be supposed to be splenic pain.

I have already referred to the anatomical relation of the spleen to the kidney, and to the possibility of an enlarged spleen being confounded with a renal tumour, or with a tumour of the suprarenal capsules.

There are two great orders of spleen-tumours, acute and chronic.

Acute enlargement of the spleen may be the result of splenitis from direct injury. This form of splenitis, however, is rare, and, when it occurs, is generally complicated with some surgical injury, which takes it out of our province, and throws the splenic enlargement into the shade. Acute enlargement of spleen is by no means uncommon; but it is always secondary to some other disease, and especially to diseases which have one peculiar and common feature. I should have formerly said, to the acute specific diseases; but we have something better than that by which to characterise them. They are all diseases marked by elevation of temperature, without local lesion to account for the elevation. For example, acute pleurisy is a febrile disease, with a local lesion to account for the elevation of temperature; a pneumonia, a febrile disease, with a local lesion to account for it; but a case of erysipelas of the head and face, where perhaps the erysipelatous inflammation is a little blush across the bridge of the nose, is a disease with elevation of temperature, without local lesion to account for it. So, likewise, are typhus fever, pyæmia, acute tuberculosis, diseases attended with elevation of temperature, without local lesion to account for it. This great group does include all the acute specific diseases; but you will see that it includes something more. It contains pyæmia, ichorrhæmia, septicæmia, and the whole of that order of cases. It includes acute deposit of tubercle, and certain cases of purpura and of scurvy even; intermittent fever comes into the same group. In a large number of these diseases, there is enlargement of the lymphatic glands of the abdomen, and a tendency to disease of Peyer's patches.

Again, in almost all these diseases there is a tendency to

hæmorrhages; in almost all there is a malignant form, where hæmorrhages occur from the nose, from the gums, from all parts of the body. In small-pox, for instance, this is sometimes seen. I have seen a case of a malignant form of that disease in which a bloody fluid issued from the eyes, from the nose, from the mouth, from the vagina, from the rectum, from the skin. In all, there is a tendency to real hæmorrhage, as well as to the escape of a solution of hæmatosin in the more malignant forms. Again, in almost all, there is excessive formation of red corpuscles and a deficiency or change of fibrine. Now, we have seen the relation of the spleen to the blood-making process; and we see that in these diseases, in which the chemical constitution of the blood is changed from an early period, it is the spleen that is damaged; and with this damage to the spleen we have alteration of fibrine and excess of red corpuscles. When acutely enlarged, the spleen is usually tender, sometimes very tender. After death, it is found dark in colour, and softer than natural.

And now let us turn to chronic tumours formed of the spleen. Enlargement and induration of the spleen result from various diseases that cause more or less continuous or remitting congestions of the organ, either by a mechanical or by a so-called vital process. Thus intermittent fever, certain forms of heart disease, and cirrhosis of the liver, are all accompanied by congestion of the spleen; all have a tendency to produce repeated congestions, long-continued congestions, and thus to induce chronic changes in its structure—induration. There is no doubt that chronic congestion of the spleen leads to thickening of the fibrous tissues, especially of the trabeculæ and of the subperitoneal fibrous tissue. Then there is albuminoid infiltration of the spleen, with which we are all familiar, increase in size of the spleen, so very common in rickets. There is the so-called ‘sago-spleen,’ said to be partial albuminoid disease. Then there are the peculiar changes (excess in cell-elements) in the structure of the spleen that occur in leukæmia.

One of the most common characteristics of chronic spleen-disease leading to induration is anæmia—hydro-oligocythæmia, if you please—a deficiency of red corpuscles, and all the consequences of this. It is to these secondary

changes in the blood that what little anasarca ever observed is chiefly due.

Secondly, we see a disposition to hæmorrhages in chronic spleen diseases; and whenever—and this is a practical point for you—whenever a patient complains of hæmorrhage here and hæmorrhage there, for which you see no direct cause, always examine the spleen. There is hæmorrhage from the gums, from the nose; and it may, in spleen-disease, even occur in the serous cavities, *e.g.*, the pleura. A great cause of the tendency to hæmorrhages, then, is the excess of white corpuscles, which may even exceed in number the red. So far as I have seen, without chronic disease of the spleen or of the lymphatic glands, if you take a drop of blood and pass it under the microscope, so as to examine the whole field, you do not get more than fifty, using the quarter-inch lens. I have seen that in pneumonia, and in some strumous persons especially; and in these I think it is due to the state of the lymphatic glands of the bronchi. But, if you find a larger number, the probability is that you have to do with some chronic disease of the spleen or glands.

Again, it is said that, in certain cases of spleen disease, you find masses of pigment in the blood, the remains of destroyed blood-corpuscles. Of that I know nothing from my own observation. Then the colour of the skin is said to be peculiar in these chronic cases of spleen-disease. Of that I have myself only seen anæmia which could be attributed to the splenic affection. The rest has been due to the natural tint of the skin, or it has been due to the conjoined hepatic disease, or to conjoined disease of the suprarenal capsules, or to some other affection. It is said, however, that there is a peculiar brownish tint of skin in some cases of splenic disease.

With reference to the question of extirpation of the spleen, I should remark, that you must make a distinction between the effects of loss of an organ and the effects of disease of that organ. It has been thought that some of these conditions could not have been due to disease of the spleen, because it could be taken away without any material effect on the system. Therefore it has been said that disease of the spleen cannot seriously affect a patient; but it is quite

clear—supposing, for a moment, the function of the spleen, in addition to other organs, to be the power of making white corpuscles—it is clear that, if you take away the spleen, it is quite possible that the other organs may supplement the spleen, and may make a full quantity of white corpuscles, and the patient suffer nothing. But suppose a disease of the spleen in which there is an excessive quantity of white corpuscles formed in it; you have the excess in the blood, with all its consequences. It is from a consideration of these two facts—viz., that the spleen has been removed without injury to life, and that the disease of the spleen leads to death—that I sanctioned in one case, and would again sanction, the removal of the spleen in cases of leukæmia.

As to the local symptoms of chronic enlargement of the spleen; there is a feeling of weight in the left hypochondriac region—a feeling of distension and uneasiness there. There is an absence of pain—actual pain; an absence of tenderness; an absence of dropsy; an absence of fever. The last point, however, admits of some question, especially in regard to leukæmia. Observations with the thermometer have not yet been made with sufficient accuracy to establish the absence of a certain amount of febrile disturbance, while there are some observations on record which point to some cases of leukæmia having been attended with irregular febrile disturbance.

I will now read you the case of Emma Hurren.

Acute Tuberculisation, Large Spleen, etc.—Emma H., æt. 29, admitted May 25th, 1868. No history of phthisis in the patient's family. Patient's health was good till she came to London, two years ago. Before that time, she lived in Ipswich for a year, and previously in Yoxford, in Suffolk, not very far from some salt-marshes; but she never had ague, nor does she know that cases of ague ever occurred there. She has been married nine years, and has had three children and three miscarriages. All her children are dead—one by accident; two by diarrhœa, aged one and five years respectively. Her habits had been temperate. She had plenty of food, and lived in an open situation, till the last two months; since that time, she has been in want, and occupied a cellar-kitchen in a court in Holborn. Two years ago, she began to suffer at times from dull aching pain in the left side of the abdomen, increased by walking. At a hospital, she was

told she had a 'lump' there, and took quinine without benefit. Nine months ago, the pain became worse, and the 'lump' bigger than it was before or has been since; and at the same time her legs and body swelled, and remained swollen for about a fortnight. During the last few weeks, she has been free from pain, but has been becoming thinner and weaker. She has no cough, but sweats much at night. She has not menstruated for the last nine months.

Present State.—June 3rd.—The patient is thin; her eyes much sunken; pallor of face considerable. She can lie easily in any position, and complains of no pain or uneasiness. There is no headache. Tongue clean; appetite good; skin hot and dry; no rash. The temperature has varied since admission, at irregular intervals, from 100·5 to 104 degrees, generally attaining its greatest elevation during the forenoon, and then sinking till night, when it will rise again. The pulse varies from 100 to 120. The respiration is about 24. There is no excess of white corpuscles in the blood. The patient has now a slight cough, without expectoration. The thorax is small and long. The right infraclavicular region is slightly duller than the left; there is no difference in respiration. Percussion and respiration are normal posteriorly. The heart is healthy. The spleen can be easily felt, reaching nearly to the crest of the ilium; its percussion dulness reaches upwards to one inch below the nipple. The anterior edge is oblique, and the notch in it is very distinct. The posterior edge is perceptible behind. The tumour moves very distinctly with respiration. It appears to vary in size from day to day, the anterior edge coming an inch and a half more forward at certain times than it does at others. It is sometimes much less distinctly felt than at others, from intestines coming in front of the lower part. The apparent variations in size bear no relation to the variations of temperature. Between the last rib and the crista ili on the right side, can be felt a body with a sharp anterior edge, apparently continuous with the anterior surface of the liver. The finger can be placed under the sharp margin. The lower border of the liver can be felt about two fingers' breadth below the margin of the thorax; it is slightly tender. The urine is quite free from albumen. The patient sleeps well.

The patient continued to lose flesh and strength; the temperature to range between 101 and 103 degrees. The pulse varied from 100 to 130. The cough became more and more frequent, hacking and loose; some moist *râles* were superadded to the other chest-signs; vomiting supervened; and she died on June 26th.

You heard it stated in the history, that the patient had had occasional pains in the left hypochondriac region. It is

always well to ascertain exactly the kind of sensation which the patients themselves vaguely term 'pain.' Only a few days ago I was in a court of law to give evidence. Dr. Walshe was at the same court; we had seen the patient together; I had attended him all through his illness; Dr. Walshe for a month or two. Now, one medical man who gave evidence swore, with the most perfect confidence, that the man suffered a great deal of pain, much to the astonishment of Dr. Walshe and myself, who were sitting by, listening to the evidence. The patient certainly had not suffered a great deal of pain in the sense in which I would use the word, or in which Dr. Walshe would have used it. He had an enormous amount of discomfort, so that he was almost always ready to tear himself in pieces, but not at all what I should call pain, nor what I would teach you to call pain. The swelling of the body, mentioned in the account of this case, was probably the result of disturbed digestion *plus* 'the lump in the belly.' Remember what I told you in a former lecture; how patients with abdominal tumours frequently do not suffer distension from the tumour till they have excessive accumulation of flatus, and then, in consequence of the presence of the tumour, an amount of flatus which would not by itself cause uneasiness, gives rise to very considerable discomfort. This woman's body swelled, probably on account of flatus, but she could have borne that amount of flatus if she had not had a big spleen. At the same time her legs swelled. Now, this swelling of her legs was due to a certain amount of fluid in the cellular tissue; and that was the result, not of any mechanical effect of her spleen-disease, such as may result from a diseased liver or a diseased heart, but of her anæmia, which, especially when conjoined with a certain amount of emaciation, will give rise to some degree of swelling of the legs. The watery serosity more readily than healthy serosity escapes from the capillaries; and, when a person is emaciated, the vessels are less well supported. In old books, the symptoms of diseases of other organs are often confounded with the symptoms of spleen-disease. For example, derangement of the circulation in consequence of diseased liver has often been supposed to be due to spleen-disease. It was noted in the account that the respirations were frequent. Now, spleen-disease has no

tendency to alter the respiration, unless it be by interfering with the descent of the diaphragm by its large size, and so making the patient breathe more frequently. The apparent variations in size were probably due to variation in the distended colon. The colon does not lie in front of the spleen, it does in front of the kidney; but when the spleen is considerably enlarged, its lower part may be, as it was in this woman, overlapped by the distended colon. The woman had cough which continued to increase. Now, cough is no symptom of spleen-disease, nor is rapid emaciation such as is noted in this case. Remember that, in the time she was in the hospital, no marked emaciation would occur from simple spleen disease. The fluctuation that was felt was probably aerial fluctuation, and not the fluctuation of fluid.

When this woman came into the hospital the first question was, What was the tumour that we felt? We had no doubt that it was the spleen, for the reasons which I have given you: first, its situation; secondly, the character of its edge; thirdly, its mobility. We felt the posterior edge as well as the anterior; and, if we required further evidence, it was exceedingly superficial, nothing lay between it and the margin of the thorax.

The second question was, 'Were the symptoms due to splenic enlargement?' This was a point of great importance in prognosis and in treatment. No, they were not due to it. At any rate, when I saw the woman, no one could entertain the slightest doubt that they were due to something else. In the first place, the spleen was perfectly indolent. There was no pain or tenderness. Now, had this been an acute enlargement, or splenitis, to lead to fever, there would have been tenderness. In all cases in which the spleen is actually enlarged from febrile disturbance when there may be a form of inflammation, there is tenderness. In typhoid fever, in scarlet fever, in measles, there is a certain amount of tenderness. Even when the patients are semi-comatose, they often shrink when you touch it. Secondly, febrile disturbance was present. Now febrile disturbance is not a consequence of spleen-disease. In some febrile diseases, as I have told you, the spleen is enlarged, but it is moderately, compared to this case; never attaining the size it had in this case;

and, again, the fever in such cases precedes the splenic enlargement; in this case, the febrile disturbance was very considerable, but in no relation to the enlargement of her spleen. We had evidence that the condition of her spleen had existed for two years, and we know that chronic enlargements of the spleen do not have, as consequence, fever of such degree as was here present, and so we knew that her illness must be due to something else than her spleen-disease. To exclude leukæmia the blood was examined and no excess of white corpuscles was found.

I am not going to discuss the case at any length, but I may briefly say that we supposed that an acute deposit of tubercle was going on, because of the duration of the disease, and the absence of any of the acute specific diseases, the absence of pyæmia, the absence of any other affections, together with the constant elevation of all temperature, varying from day to day and at different parts of the day; and this led us to the conclusion that it was an acute deposit of tubercle, affecting especially, we thought, her lungs, because of her increasing cough, and because she had a certain amount of physical signs.

And now I will read you an account of the *post-mortem* examination thirty hours after death. The diaphragm was greatly arched; the spleen more under the chest-walls than it was felt to be during life; the lower end covered by the transverse arch of the colon, which was distended with flatus; the costo-colic ligament was very broad. The liver lay very obliquely, so that the left lobe extended very little beyond the middle line; the body felt in the right lumbar region was part of the right lobe of the liver itself, lying vertically and extending much below and in front of the kidney, so that it would not have been possible to feel the latter; the intestines and peritoneum were redder than natural. Both lungs were studded throughout with grey granulations; there were evidences of recent pneumonia, and much mucopurulent matter in the bronchial tubes. The kidneys were healthy. The spleen weighed 17 oz.; it was $8\frac{1}{2}$ inches in length, and $4\frac{1}{2}$ inches in breadth; the trabeculae were more visible than they should be; the Malpighian bodies less so; the tissue generally was much firmer and tougher than

natural. The liver was not enlarged, but its cut surface was granular, the margin of the granules being very vascular, the granules pale and soft. The spleen lay high under the ribs, for the diaphragm was greatly arched after death, because the patient died at the termination of an expiration. This is a point not always sufficiently thought of in considering the *post-mortem* position of organs. The patient dies at the termination of the deepest expiration he ever made in his life, and the diaphragm is greatly arched in consequence, and the organs lie higher up under the ribs than they were felt to do during life. There was scarcely any fluid in the abdominal cavity. I mentioned to you the possibility of a certain amount of fluctuation being caused by a distended colon containing fluid. The condition of her lungs was sufficient to enable us to say what was the cause of death. The spleen presented the characters that we commonly find in those which have been the subject of chronic congestion and of the changes consequent on congestion. It is possible, very possible, that her living in the neighbourhood of a marsh may have had something to do with the spleen disease. The white patch on the anterior margin of the spleen was at the spot where it had rubbed up and down during respiration. We felt it to do so, and as it pressed against the chest it led to thickening of the capsule. The microscopic characters were just those of a spleen that had been long congested, containing a large number of fibro-plastic corpuscles in the process of development into tissue. It was as if the cellular tissue of the organ were becoming much increased in quantity.

Those are the principal points in connection with this case. I should mention to you the case of the boy who was lately in the hospital, and who died of pericarditis. We found that his spleen contained a considerable excess of blood. It was much enlarged, but never descended below the margin of the thorax. But, in addition to the congestion, which was probably due to the state of the heart, we found two spots on the spleen, of which I should like to read you an account.

‘There were two crimson spots at the surface of the spleen. One was about $\frac{1}{8}$ of an inch in breadth, and a $\frac{1}{4}$

of an inch in depth, extending into the substance of the spleen, the broadest part being at the surface. The other was somewhat larger, somewhat paler; and the tissue around was very dark. They were both wedge-shaped, both almost structureless and granular. The one looked like a recent clot of blood of that peculiar form; and the other was a little puckered at the surface, and the tissue around rather congested.'

Now these spots were the result of the circulation in the blood of minute particles of fibrine; in fact, they were due to embolism, and to coagulation of blood around the particle of fibrine, which was exceedingly small; and, being arrested here, blood accumulated in the vessels around, and was extravasated. These spots are not uncommon in the disease from which this boy suffered—acute rheumatism, with endocarditis; and in these plates you will see representations of the same thing in the kidney. Particles of fibrine are carried from the valves of the heart, are arrested in the capillary circulation of the liver or kidney, and so give rise to these wedge-shaped fibrinous-looking masses. Rayer termed the condition 'rheumatic nephritis.' We might just as well term the condition in the spleen 'rheumatic splenitis.' Rokitansky has termed it 'capillary phlebitis'; but now there is no question that these wedge-shaped masses are due to embolism.

I should have mentioned to you that, in cases of acute tuberculosis, the spleen is enlarged; and in children it is often at the same time the seat of a deposit of tubercle, but in the adult it is the exception to find tubercle in the spleen. You know that the organs are most prone to deposit of tubercle which are in a state of active work; and you know in what an active condition in the child are the spleen, lymphatic glands, and Peyer's patches, all of which are then especially liable to deposit of tubercle in early life.

Cancer of the spleen is exceedingly rare; you would think first of everything else. But it is the seat, now and then, of a deposit of cancer, which assumes usually the nodular form; but it is very rare, and I have never seen it except in conjunction with deposits in other organs. There was a woman in the hospital lately whom I supposed to have cancer of the spleen. You all saw her. She lay in my female ward; her

name was Beaumont, her age 49. She had scirrhus cancer of the right breast, and a cancerous mass in the anterior wall of her rectum. Her liver was rather large and hard, perhaps infiltrated with cancer. She was sallow, and offered a good specimen of the appearance of the subjects of cancerous cachexia. In this woman a large tumour occupied the left hypochondrium, and passed up under the ribs. It was hard, with a sharp edge which met the margin of the thorax at a right angle. It was plainly the spleen. Its dulness extended up to the level of the nipple, and the lower edge was on a level with the umbilicus. It was situated just beneath the parietes, moved up and down with the movements of respiration, and could be pushed a little from side to side by the hands behind and in front of it. Its surface was irregular, and in the anterior border several distinct nodules could be felt. It was not tender. The woman left the hospital, and, after a few weeks, died in the country.

LECTURE IV

Enlargements of the Liver.—Illustrative Case •

GENTLEMEN,—I considered at the last lecture tumours of the abdomen formed by the enlarged spleen. I do not mean that I finished the subject, for I intend, from time to time, to bring under your notice other cases of splenic disease causing tumour in the abdomen.

To-day I shall briefly consider the subject of liver-tumours. I mean by that, tumours of the abdomen dependent on enlargement of the liver. First, I wish to recall to your mind the anatomical relations of the liver to the diaphragm, to the pleura, to the lungs, and to the pericardium. For practical purposes you are not likely to forget its relation to the lungs and pleura, but I am quite sure that you are likely to overlook its relation to the heart and pericardium, for I did so myself till experience taught me to remember it. Remember the close relation in position of the liver to the stomach. The importance of remembering this is illustrated by the case I shall relate to you presently. Recollect also the relation of the liver to the kidney, to the supra-renal capsule, to the colon, and of the left lobe of the liver to the spleen.

Displacement of the spleen, sufficient to cause tumour in the abdomen, I told you was, to say the least, infrequent; displacements of the liver, sufficient to cause tumour in the abdomen, is common; displacement of the liver downwards is very frequently caused by effusion into the pleura; very frequently by dilated heart; frequently by emphysema of the lung; less frequently by effusion into the pericardium; and rarely by new formation between the liver and the diaphragm or in the lung itself.

Great dilatation of the right side of the heart always

causes some displacement of the liver, and often displacement sufficient to cause true abdominal tumour. Displacement of the liver never causes ascites or jaundice. You know that the liver is displaced, especially by noticing the rounded upper surface; you see, passing across the abdomen, just below the margin of the thorax and below the ensiform cartilage, something that is rounded, and then find by percussion that it is the upper surface of the liver. Fluid in the right pleura, sufficient in quantity to displace the liver downwards, is accompanied by such loss of power or paralysis of the same side of the diaphragm that when the patient breathes there is little or no ascent or descent of the liver. In these cases, also, the other evidences of effusion into the pleura are present. When a dilated heart depresses the liver the pulsation of the right ventricle is felt below the ensiform cartilage, and you see, unless the patient is very fat, the rounded upper border of the left, and of a small portion of the right lobe of the liver. In such a case the liver may move up and down in respiration. Emphysema of the lung is a common cause of a certain amount of displacement of the liver; but, in this case, percussion enables you to determine the cause.

The upper rounded surface of the depressed liver is well seen in a man now in my wards in the hospital, and to whose case I shall presently refer. As I attach importance to your appreciating by sight, during life, this upper rounded surface of the liver, I have frequently pointed it out to you in this man. So much for the relation of the liver to the diaphragm, to the pleura, to the pericardium, to the lung, and to the heart.

The relation in position between the liver and the stomach is also very important. Disease may extend from the liver to the stomach, and so give rise to some peculiar symptoms. A man in the hospital lately, to whose case I shall presently refer, had cancer of the liver; the disease, as we shall see, extended to the stomach. Cancer of the stomach may spread to the liver, though the converse is more frequent, *i.e.* cancer more often passes by extension from the liver to the stomach than from the stomach to the liver. Abscess of the liver may burst into the stomach; hydatid tumours of the liver

may burst into the stomach; chronic ulcers of the stomach may eat into the liver. Then remember the relation in position of the liver to the colon. The colon sometimes lies in front of the liver, and renders a diagnosis difficult which would otherwise be easy. You may think there is a tumour originating below the liver, when really it is the liver itself, if you forget that the colon sometimes passes in front over the right lobe. Again, the colon or the dilated stomach may yield resonance enough to cause enlargement of the liver to be overlooked. Certain common abnormalities of form of the liver should be borne in mind when attempting to make a diagnosis of abdominal tumours. Now and then the left lobe of the liver is very small, and the right lobe lies very low, the organ being perfectly healthy. You may make a great mistake if you do not remember this; you may examine a patient complaining of some dyspeptic symptoms, and mistake this right lobe for an enlargement, and conclude that he has a diseased liver. Again, variations in the position of the liver in different persons should be borne in mind. In some persons it lies very vertically, especially in persons with a small thorax, and in women who lace tightly. On the other hand, in some persons with a large thorax, and in some persons with incipient emphysema of the lung, a small portion only of the liver may be felt, because, the diaphragm lying horizontally, the liver lies horizontally also. I saw, only a few days ago, a case which illustrates these remarks. There was abdominal tumour, and the question was what was its origin. The patient was a lady, and had therefore worn stays. Her liver had been compressed and lay vertically. The margin of the thorax had pressed upon the liver and had caused, about an inch above its margin, a thickening of the capsule, and a diminution in the thickness of the liver at the spot. Subsequently the lady had become the subject of cancer of the liver, which had led to considerable enlargement, and the organ extended downwards to the crest of the ilium. The transverse linear depression had been carried downwards as the organ increased in size. In passing the hand upwards over the enlarged liver one came to the transverse linear depression I have just described, and the first idea which arose in the mind was that there was a tumour totally uncon-

nected with the liver, the part below the depression being taken for a tumour unconnected with the liver, and considerable doubt had been expressed concerning the nature of the ailment. You will remember this transverse linear depression on the liver, the result of compression, and the consequent apparent separation of the lower part of the right lobe.

Now and then the liver lies very much to the right. It did so in the woman on whose case I lectured last Monday. It appeared to have been pushed there by the distended colon, the distended stomach, and the large spleen. The consequence was that the right lobe could be felt as far as the crest of the ilium; and as it was thin, overlapped, and lay in contact with the right kidney, it was mistaken for a renal tumour. I have told you already how we knew that it was not; viz., by the sharp edge and the nearness to the lateral abdominal parietes, and its ascent and descent with the rise and fall of the diaphragm in respiration.

Having determined that a given tumour is hepatic, we desire to know the disease causing the enlargement. Chronic impediment to the systemic venous circulation causes anasarca. An impediment to the passage of blood through the heart or lungs has general anasarca as a mechanical consequence. Now, the liver has no direct relation to the systemic circulation; no blood from the lower extremities, no blood from the kidneys, passes through the liver. But the blood from the lower extremities, and from the kidneys, passes through the vena cava, which lies in the notch at the back of the liver: that is its only mechanical relation to the systemic venous circulation. But, as a matter of fact, oedema of the lower extremities, of all the parts which empty their blood into the inferior vena cava, does occur in certain cases of disease of the liver. How is it induced in these cases? Liver disease produces oedema of the lower half of the body in two ways. First, causing great distension of the belly, either directly by its size or indirectly by the ascites it induces. Great distension of the belly, whatever its cause, is necessarily accompanied by compression of the anterior wall of the vena cava, as it lies on the posterior surface of the abdomen, and thus a certain amount of impediment to the passage of the blood through the vessel is produced. What-

ever force pushes forward the anterior wall of the abdomen, will, to some extent, compress everything lying on the inner surface of the posterior wall. The liver never compresses the vena cava simply by its weight. It is the distension of the belly by the liver itself, or by the great disturbance by digestion, and consequent flatus in the intestines; or, more commonly still, by fluid in the peritoneum, which is accompanied by compression of the vena cava. Very fat people with large abdomens often have œdema of the lower extremities from this cause.

Secondly, hepatic disease may induce œdema at the lower half of the body by directly narrowing the vena cava in the notch at the posterior part of the organ. For example, cancerous growths in the liver are occasionally developed in such a situation that they push the anterior wall of the vena cava back into the vessel, and so narrow its calibre. In cirrhosis of the liver, too, it is not uncommon to find one or more nodules projecting back into the vena cava. In such a case, if after death the finger be passed up the vena cava where it lies in the notch behind the liver, these projections into the vessel may be felt. It is in these ways that the vena cava is compressed, and its channel narrowed. The mere weight of the liver, as you will see, if you think of it for a moment, can have no physical effect in compressing the vena cava, which is placed in a notch in the posterior part of the organ.

It is important to remember that this narrowing of the vena cava produces an effect, not only on the veins of the lower extremities, but also on all the parts from which the blood is returned to the inferior vena cava. Thus an impediment to the flow of the blood through the vena cava is necessarily an impediment to the return of blood from the kidneys, and hence you see a mechanical cause for the relation between hepatic and some other abdominal tumours (pregnancy) and albuminuria, a cause which is superadded to, and enhances the effect of, any constitutional condition which may affect alike both organs. Remember always, in estimating the occurrence of anasarca, or œdema of the lower extremities, the relation between renal disease and any other cause of dropsy. A patient, that is to say, having Bright's disease, in its nature tending to produce anasarca, though in

extent too trifling to give rise to it, if he had any disease superadded calculated also to produce anasarca, then the two shall produce such a degree of anasarca as either one alone would be totally inefficient to produce. Let a man with an impediment to the return of blood through the vena cava, insufficient to cause œdema of the lower extremities, become the subject of a renal disease also insufficient to produce anasarca, and the two conjoined shall produce considerable effusion of serosity into the cellular tissue of the lower half of the body. A man has hepatic disease impeding, in a very trifling degree, the flow of blood through the liver; no effusion into the cavity of the peritoneum is produced; he becomes the subject of Bright's disease, and ascites is the result of the two diseases, whilst neither would have alone produced it. So remember what I would call the intensifying character of Bright's disease in the production of local œdemas.

Now, what is the influence of hepatic disease on the portal circulation in producing symptoms? Remember that the hæmorrhoidal veins, the intestinal veins, the veins from the stomach, from the spleen, from the peritoneum, all empty their blood into the portal vein, and that the blood of the portal vein passes through the liver; consequently, any and all of the diseases of the liver which interfere with the flow of blood through the liver have, as consequences, disturbance of the digestive organs,—congestions, that is to say, of those parts; and, in a very large number of cases, one or more of the following symptoms:—Hæmorrhoids, with or without a flow of blood from them. When a patient consults you for hæmorrhoids, don't forget for a moment their relation to hepatic disease: you may kill the patient if you do not remember it. Diarrhœa is another symptom of impediment to the flow of blood through the liver. A patient may owe his health, or even his life, to his repeated attacks of diarrhœa. He has hæmorrhoids because the blood from his rectum has to pass through his liver. Congestion of his portal vein follows, and the hæmorrhoidal veins pour out a quantity of blood and relieve it, just as a man having congestion of the brain and meninges suffers an epistaxis, which relieves him. Again, a man has congestion of the radicles of his portal vein, and has a diarrhœa which wards off the bad

effects of the hepatic disease, relieves the overfilled portal radicles. Tie his hæmorrhoidal veins, stop his hæmorrhoidal flux, and he becomes the subject of all the ill effects of congestion of the portal vein. Stop his diarrhœa, and he has ascites, perhaps hæmatemesis. We see instances of these—every day, I was going to say. Certainly I never pass a week without applying a knowledge of these simple facts to practice. Then hæmatemesis is another symptom. In rarer cases, the impediment to the flow through the liver, leading to overloading of the veins of the stomach with blood, a great escape takes place—a capillary hæmorrhage, commonly to the great relief of the patient, though it may be, if excessive, to his death. Again, enlargement of the spleen results from diseases of the liver, which impede the passage of blood through the portal vein. All the splenic blood has to go through the liver, and any impediment to the flow through the liver leads to congestion of the spleen. Again, ascites occurs from the overfilling of the radicles of the portal vein, which receive the blood from the peritoneum. Effusion of serum takes place from the overfilled capillaries; and serosity in the peritoneum is ascites.

Now, it is important to bear in mind that the occurrence of any one of these hæmorrhages or effusions may prevent the production of the others by relieving the congestion, which is the common cause of all. The diarrhœa may keep the patient free from hæmorrhoids, free from ascites, free from hæmatemesis, free from enlargement of the spleen. His portal vein is effectually relieved by the escape of fluid from the intestinal mucous membrane—that is, from the intestinal radicles of the portal vein.

There is one point in the anatomy of these parts to which I perhaps ought to have referred before. You will remember that there are a great number of anastomoses between the portal vein itself and the superior vena cava; and in some persons these are so free that they escape all mechanically-produced troubles resulting from impediment to the flow of blood through the liver. In the vast majority of persons, however, these anastomoses are totally inefficient for the establishment of a collateral circulation. They can afford only an infinitesimally slight relief to the over-distended

portal vein. So much, then, for the relation between the systemic circulation, the portal circulation, and certain diseases of the liver, *i.e.* diseases of the liver which impede the flow of blood through the portal vein, *i.e.* the trunk or its terminal twigs.

I told you that in determining the nature of the disease of the liver which produces a tumour, we kept in view the influence of the disease on the circulation through the liver, and on the secretion from the liver. We have considered the former—*i.e.* evidences of interference with the flow of blood through the liver, and now come to the latter—the secretion from the liver.

Bile is said by some persons to be first formed in the blood, but this point does not practically matter to us. The question is still too much *sub judice* for us to draw any practical conclusions from it. It is enough for us to know that any mechanical impediment to the escape of bile from the liver causes jaundice; leads to the presence of bile in quantity in the blood; that jaundice may occur without such mechanical impediments; and that whatever the cause of this accumulation of bile in the blood, certain nervous phenomena may follow. I say this because it has been supposed that it is only one disease that leads to the peculiar nervous phenomena—namely, what has been called acute yellow atrophy of the liver. There is no doubt that these phenomena, delirium, etc., may occur, whatever the cause of jaundice, and whether it be acute or chronic. Now, nervous phenomena in jaundice are due to one or two causes—viz., 1, to blood-poisoning, *i.e.* the presence in the blood of bile, or of some of its elements, or of some of the products of its decomposition; and 2, to inflammation of the membranes of the brain which occur in a certain proportion of fatal cases of jaundice. It is the meningitis of the convex surface of the brain which suffers in those cases. I have described to you some of the consequences of impediment to the flow of blood through the liver, and impediment to the escape of bile from the liver—as only some of the diseases of the liver have the effect of damming back the blood in the portal veins, or of interfering with the escape of bile; we may practically divide hepatic diseases which cause tumour into two great classes—

those which produce ascites and jaundice, and those which do not. Those conditions which produce ascites must impede the flow of blood through the liver; and those which impede the flow of blood may also impede mechanically the escape of bile from the liver. There are, however, a certain set of cases which do produce jaundice, without impeding the flow of blood through the liver. These are the two great groups; so that, when you come to the bedside of the patient and feel a big liver—*i.e.* a tumour which you conclude, from its physical condition, to be the liver—the first questions you answer are, 'Has the patient any fluid in the cavity of his belly? has he any jaundice? has he ever had jaundice?' Then you inquire about diarrhoea, hæmorrhoids, hæmatemesis, œdema of the lower extremities, and enlargement of the spleen; but, at the bedside, first determine the presence or absence of ascites and of jaundice. I think that the examination which a practical man would make would be something like this. He would ask, 'How long have you been ill?' and then, passing the hand over the abdomen, and feeling a big liver, he would percuss the upper border, notice the position and character of the lower border and the consistence of the liver; and then look at the patient, and see if he has jaundice, and examine him to ascertain if he has ascites; and then proceed to the other symptoms.

And now to apply what I have said to the case before us. It is a case of a common disease—*viz.*, cancer of the liver.

Thomas H., aged 35, shoemaker, was admitted June 5th, 1868. The only point worthy of note in the patient's family history is, that his mother died at about forty-five, of an 'internal hæmorrhage,' after bringing up blood for three days. Till lately, the patient had always enjoyed good health. His habits had been temperate. During the last few years he has lived poorly, and worked in a close crowded workshop. There is no history of syphilis. He is married, and has two healthy children. During the last eight or nine years, he has suffered slightly from hæmorrhoids. He dates the commencement of his present illness four months ago, when, after a rather heavy meal, he experienced great fulness and discomfort at his stomach (epigastric region). He became thinner, and felt weaker, and was sent by some friends to Eastbourne. While there, a fortnight before admission, he noticed that his skin

was becoming yellow; and, about the same time, he began to bring up his food from one to three hours after taking it, and he has since continued to do so occasionally. A day or two after the commencement of the sickness, he brought up a quantity of matter 'like coffee-grounds.' From the time the jaundice was first noted, it never disappeared or diminished in degree.

Present State.—The patient is much emaciated, his face equally with his trunk. There is an universal lemon-yellow tint of skin and of the conjunctivæ. The face is slightly dusky. He lies easily in any position, and complains of no pain. The abdomen is not prominent; the sides very slightly bulging; the extreme flanks dull. Imperfect fluctuation is detectable. The umbilicus is slightly tender, reddened, and indurated, the hardness extending round it about two-thirds of an inch. This condition of the umbilicus did not cause any discomfort; the patient did not know of its existence. The liver-dulness commences in the nipple-line at the sixth rib, four fingers' breadth below the nipple. The margin can be felt about three fingers' breadth below the margin of the thorax, not sharp; but both the margin and the upper surface feel extremely hard. There is general fulness and hardness at the epigastrium. To the left of the ensiform cartilage, a hard, prominent nodule, of about the size of a marble, can be felt on the liver. No tumour can be detected elsewhere in the abdomen; but just above Poupart's ligament, in the right groin, is a hard round gland, of the size of a small cob-nut. The urine exhibits abundant evidence of bile.

June 6th.—This morning, the patient brought up about half a pint of thick 'coffee-ground' matter.

June 15th.—Since last report, the patient's condition has not altered. Occasionally he vomits his food, and once or twice small quantities of the coffee-ground matter. The vomiting is preceded by nausea, but not accompanied by retching. He is taking a mixture of nitro-hydrochloric acid and spirits of chloroform, which, he thinks, is doing him good.

June 26th.—The nodules of the liver are larger than they were. A slight depression can be felt in the one at the epigastrium. The fluid in the abdomen is increased. There is no oedema of the lower extremities. Emaciation is extreme; colour unchanged. He complains of a burning pain at the epigastrium.

June 28th.—Since last report, the patient has been gradually sinking, vomiting occasionally, but not constantly; and he died this morning at 10 A.M.

From the date of admission till the day of death, the temperature ranged from 98 to 98.6 degrees. The pulse never exceeded 64 till a few days before death. The respirations were 14 to 18.

Post-Mortem Examination, thirty hours after death.—The surface of the body appears more intensely yellow than during life. All the tissues of the body are also stained yellow. The left lung is somewhat cedematous. On the surface of both lungs are numerous greyish-white patches, in size from a pin's head to a split pea, solid, with deep red margins. At the base of the right lung is a circumscribed patch, probably malignant. The liver projects less downwards than it was felt during life. The surface is covered with nodules, evidently malignant, from the size of a pin's head to that of a nut. The prominence which could be felt at the epigastrium is now seen to consist of several nodular masses. The upper border of the liver is adherent to the diaphragm, and the adhesions are infiltrated with cancerous matter. The weight of the liver is 61 ozs. and it measures 10 inches by seven. The right lobe is small. The little nodules on the margin have depressions in their centres. The substance of the liver is dark green, and is studded throughout with cancerous nodules. The induration about the umbilicus is due to malignant disease. The peritoneum is infiltrated with nodules of cancer, especially near the liver, and in the omentum, which is adherent to the parietes. The stomach is small; its coats are thickened. At the pyloric end, the wall is infiltrated with cancerous matter, and ulcerated. The diseased part is $2\frac{1}{2}$ inches in extent, but does not reach to the pylorus itself, which is quite healthy. The vermiform appendix is solid, from infiltration with cancerous matter. Between the rectum and the base of the bladder, about 5 inches from the anus, is a hard mass, of the size of a Maltese orange, involving the walls of both viscera, but encroaching to only a slight extent on the cavity of either. The kidneys are healthy, but yellow. The brain is healthy; the white matter scarcely, if at all, stained.

This man was of an age (35) when cancer of the liver is by no means uncommon. Although there is no distinct family history of the disease, it is very possible, from the mother's symptoms, that she had cancer of the stomach. Though his symptoms had not lasted very long, you must remember that patients always date their illness in these chronic cases much later than it really commenced. This man dated his illness from the first discomfort he experienced, but probably the disease had been present for months before. The jaundice in this case was permanent. It is so in all cases of cancer of the liver. Once established, it never goes away; for it results from compression of some of the hepatic ducts, or of the cystic duct, by the growth of a

tumour; and the growth usually increases. The coffee-ground vomiting, you know, is a sign of hæmatemesis. The blood, oozing from a large number of capillaries, is acted on by the gastric juice as it does so, and is converted into that black matter. Only do not confound bilious vomiting, dark green bile, with 'coffee-ground' black matter. Sometimes, even when you see it, you will have a difficulty in determining which it is. Sometimes you may use chemical reagents which may assist you. And one other point is useful: if you dilute the bile, however dark it is, you commonly bring out the greenish colour; while altered blood will still retain its black tint. It is noted that the patient suffered no pain. Cancer of the liver is one form of cancer that is unaccompanied by pain, unless the peritoneum becomes involved, as it usually does towards the end of the case, and then the pain may be severe.

The condition of the umbilicus is a point to which I would direct your especial attention. I have noticed, in several cases of cancer of the liver, that one of the earliest indications of the exact nature of the disease was obtained from an examination of the umbilicus. This is a fact of great practical importance; and, when you have a case which you suspect to be hepatic cancer, or about which you are doubtful, you had better examine the umbilicus, for there you may get the key to the whole mystery.

In this case, the liver lay rather low for cancer, in which the dulness usually extends considerably upwards. The cause of this, I believe, is the adhesion of the liver to the diaphragm, and the paralysis of the latter in consequence of the growth into it of cancerous nodules. Whenever you feel on the surface of the liver distinct nodules, as we did in this case, you may be quite sure that you have to do with either cancer of the liver, cirrhosis of the liver of an unusual form, syphilitic disease of the liver, or hydatid disease of the liver. Hydatids of the liver, I think, you might have excluded in this case. When more than one hydatid nodule is to be felt, one or more would certainly form large rounded tumours. If the nodules that were perceptible to touch were larger than a pea, I think you may come positively to the conclusion that the disease was not cirrhosis; that it was either

cancer or syphilitic disease. But then syphilitic diseases of the liver in the adult are infinitely rarely, all but never, attended with peritonitis, with jaundice, or with ascites, conditions common in cancer. But a growth in the liver increasing with the rapidity with which, as the subsequent notes show, this growth did, could be nothing but cancer. But let me caution you about a fallacy. The patient was growing thinner, and in such a case it might be that the liver was more easily felt. Here there was no doubt, however, of the actual increase in size. Again, a depression felt on the centre of a nodule on the liver, such as was felt in this case, is absolutely diagnostic of cancer. The anasarca which occurred towards the end of the man's life was probably in part due to the fluid in the abdomen; in part, to the narrowing of the vena cava; in part, to changes in the blood, rendering it more watery; in part, to the emaciation. There was a condition of blood favourable to its escape from the vessels, a considerable amount of emaciation, and therefore less support of the capillaries, conditions which might alone lead to a small amount of œdema; and if there be a slight compression of the vena cava, would be sufficient to determine a decided amount of effusion of serosity with the cellular tissue. At the *post-mortem* examination, we found the liver to lie higher than it was felt during life, just as we found the spleen in the case which I mentioned to you last week. The patient dies at the end of an expiration, the diaphragm accordingly is greatly arched, and the organs in contact with the diaphragm lie high up under the margin of the thorax. There was a considerable cancerous growth in the wall of the stomach, which might alone have given rise to the hæmatemesis. There were no symptoms during life of pyloric constriction, and after death we found the pylorus free from disease. Now when cancer occurs in the stomach primarily, beginning as a separate centre, it occurs, as a rule, at the pylorus, or at the fundus, or at the cardiac orifice. In this man, it was not at the pylorus, and it had doubtless passed by extension from the liver. Some inflammation had occurred over a cancerous mass in the liver; adhesion had formed between it and the stomach, and the coats of the latter were infiltrated with cancer.

Cancer of the liver, I may say, producing abdominal tumour, occurs under two forms—infiltrating cancer, of which we have a specimen in this preparation, and cancerous nodules. Now and then cancer nodules in the liver are so small as to give rise to no symptoms. In the infiltrated form, there is less tendency to inflammation of the peritoneum than in the others, in which there is great tenderness, and often friction to be heard. You may find in the liver scirrhus, encephaloid, and colloid cancer. Colloid is rare; perfect specimens of scirrhus, and perfect specimens of encephaloid, are rare. The usual form is what has been called a scirrho-encephaloid, not very soft and not very hard. The occurrence of colloid cancer in the liver has been doubted. We have an excellent specimen in University College Museum. The diagnosis in this case rested—1st, On the size of the liver; the nodules on its surface to be felt during life; the increase in the size of the nodules while the patient was under observation; the depression on the surface of one of the nodules. 2nd, On the cancerous condition of the umbilicus. 3rd, On the gland in the groin.

As mechanical consequences of the cancer, there were—1. Ascites; 2. Jaundice. As points bearing on the diagnosis—Tenderness of the surface of the liver; hæmatemesis.

You see from this case how large an amount of cancer may be rapidly formed without any elevation of temperature, without any increased frequency of pulse. There is another case in the hospital to which I wish to direct your attention but it is too long to go into to-day. I may say, however, that the man's primary disease is a dilated heart—primary, that is to say, in relation to his liver. The dilated heart has depressed the liver, and rendered visible through the wall of the abdomen the rounded upper surface, which, when seen, is so characteristic of depression downwards of the liver. He has, in addition, a certain amount of enlargement of his liver, from congestion, to which I shall have to refer on another occasion.

LECTURE V

*Abdominal Enchondroma*¹

GENTLEMEN,—In the preceding lectures, I have described enlargements of the liver, the spleen, and the kidney, as causing abdominal tumours. To-day I shall speak of a tumour in the abdomen, not due to the enlargement of an organ.

This photograph was taken from a woman lately in the Hospital; and the preparation on the table, removed from the woman's abdomen after death, is the tumour which caused the appearance represented in the photograph. I shall first read an abstract of the case, and then tell you what the tumour was supposed to be during life, the grounds for that opinion, and how far the opinion was verified after death.

M. O., aged about 36, widow, was admitted on July 8th. The patient had good health until the commencement of growth of the tumour. She was married at nineteen, and had seven children. Her husband died three years ago. The catamenia had always been regular. Six and a half years since, a few days after the birth of a stillborn child, the patient was seized with violent gnawing pain in the right hip, especially severe when walking or standing. The pain lasted three or four weeks, and then left the hip and settled in the right knee. There was neither redness nor swelling about the joints. The pain in the knee left her two or three months before her last confinement (four years ago); but afterwards it returned worse than ever, and continued to the present time unchanged in character and seat. The child born four years ago is healthy and still living. While recovering from this confinement, she observed a lump in her side. It was situated about midway between the cartilages of the ribs and the crista ilii, and in the nipple line, or a little outside it. Its size, when first

¹ *British Medical Journal*, January 1870.

noticed, was about that of a cricket-ball. It was intensely hard, but not tender. She heard the surgeon who attended her remark that it was probably a bony tumour. It was quite fixed. For about twelve months it did not increase in size; nor had she any pain, except in the knee. After this, the tumour began to increase. Neither at this nor at any subsequent time did she observe anything wrong with her urine; it has never been thick or bloody.

About two years since, the tumour commenced to grow very rapidly, and at the same time it became softer. Twelve months ago, her thigh became drawn up towards the abdomen, and she was not after that time able to straighten it. She continued to feel pain in the knee; but the tumour was never very painful. For some months before admission, she occasionally vomited her food.

When seen after admission, the patient was lying quietly in bed, and complained of no pain. She could lie on her back, or on either side. Her complexion was pallid, but not sallow; the expression cheerful. The face and body were fairly well nourished. The tongue was clean. The bowels required medicine to make them act. A large tumour was seen to occupy the entire right side of the abdomen, extending not more than three or four inches to the left of the median line. It was so firmly fixed that it could not be moved, except with the whole body.

The photograph indicates the shape and size of the tumour. On a level with the nipple, near the upper border, were three small prominences, hard, resistant, and slightly movable. Immediately under these, the mass was exceedingly hard. The great rounded prominence was elastic, and was semi-fluctuating; as was also the prominence of which the umbilicus was the centre. At the inner border of the latter, to its left, were two or three small hard lumps. The portion of the tumour in the lower part of the abdomen was also evidently a large cyst. The intestine was over the left side of the tumour. Behind, on the right side of the spine, there was another swelling, as large as the two fists, firm, but elastic, and evidently of the same character as the softer portion of the tumour in front, of which the projection behind was evidently part. It could be traced behind to overlap the transverse processes of the lumbar vertebræ, and the posterior part of the iliac crest near the spine. The tumour descended into the pelvis. By a vaginal examination, a mass was found, occupying chiefly the right side of the pelvis, of the size of the foetal head, smooth and elastic, clearly containing fluid. The uterus was healthy, pushed up and to the right. On the left border, just above Poupart's ligament, was a small tumour, of about the size of a bantam's egg, movable, and apparently separate, or loosely connected with the rest.

The right thigh was flexed upon the abdomen, and could not be straightened. There was complete paralysis of the branches of the anterior and external cutaneous nerves, motor and sensory.

The urine was acid, of specific gravity 1017, with the faintest trace of albumen. A deposit of pus fell, but not more than might be accounted for by a leucorrhœa from which the patient suffered.

July 29th.—A fine exploring trocar was passed into the part of the tumour next the umbilicus. A red tenacious fluid escaped, of about the consistence of the white of an egg. Under the microscope, it was found to contain great quantities of red blood-corpuscles and a few larger cells. Some minute whitish flakes, which floated in it, were found to consist of large granular and fatty cells, about 1-2000th to 1-800th of an inch in size, for the most part rounded, but in some rare instances with outrunners.

The patient continued in much the same condition—becoming thinner, the pain in the leg and œdema increasing—till August 25th, when one of the cysts was tapped, under the direction of Dr. Ringer, and about two pints of fluid removed, precisely similar to that just described. Considerable relief followed, and no ill effects resulted from the operation.

On August 27th, another cyst was tapped, to the right of the umbilicus; but only an ounce of very tenacious fluid could be obtained.

On August 29th, she was tapped again, still more to the right: and six ounces of similar fluid were removed.

Next day (August 30th) there was considerable tenderness around the puncture; and on August 31st the pulse was 148, wiry; temperature 105·4 deg. The tongue was furred; the tenderness was increased. During the next four or five days, the signs of peritonitis gradually subsided, and the temperature fell nearly to the normal.

On September 5th, however, the right leg (in which the œdema, partially reduced by the tapping, had again returned) became the seat of erysipelas. The skin became red, and blebs formed at different places. The temperature rose to 102 deg., and remained at that height for several days.

During this time the patient had been much troubled with vomiting, more at some times than others. She emaciated rapidly. The character of the tumour remained unaltered, but its size increased; and the pressure of the pelvic portion on the veins caused extreme œdema of the external genital organs and occasional retention of urine. To relieve the last-named symptoms, it was in contemplation to tap the pelvic part of the tumour through the vagina. The idea, however, was abandoned, in consequence

of the prostrate condition of the patient, who steadily sank, and died on September 15th.

Post-Mortem Examination by Dr. Bastian, forty-eight hours after death.—The heart and lungs, liver and spleen, were all healthy. The right kidney was displaced upwards, not involved in the new growth, and fatty. The left kidney was healthy. The abdominal cavity was distended with a large, somewhat rounded, growth, partly cystic in character. The cæcum was superficially adherent, but no other part of the intestinal canal was connected with the morbid growth. After careful dissection, it was found that the main portion of the growth was of a soft, enchondromatous character, apparently having its origin at or near the right iliac synchondrosis. The growth was closely adherent to, and indeed continuous with, the lumbar vertebræ, and also the edge and much of the surface of the right iliac bone. The latter was much thinned at some places, and at one small spot was actually perforated. The uterus and ovaries were perfectly free and healthy; merely displaced to the left side of the pelvis. On section, the tumour was found to have a central portion, somewhat hemispherical in shape, eight and a half inches in diameter. This was for the most part solid, though there were smallish cysts scattered through its substance, mostly containing a thick sanguineous fluid. Many large cysts—some containing half a pint of thick pultaceous fluid—were situated at the periphery of the growth: some of them were opened during its removal. The weight of the solid portion of the tumour was fifteen pounds.

When the woman came under observation, the first question to be answered was this—Is the tumour formed by an enlarged viscus? I have told you that the first points to be investigated, the first questions to be answered, when you meet with an abdominal tumour are, ‘Is it an enlarged viscus, enlarged from disease or otherwise; or is it a misplaced viscus?’ This could be no misplaced viscus. There could be no question that it was a large body not natural to the abdomen. To answer the question whether it is an enlarged viscus or not, I told you that we note especially the situation of the tumour. This tumour was seated in the right side of the abdomen, extended some inches across the middle line to the left, was in contact with the right lateral abdominal parietes, descended into the pelvis, ascended into the right hypochondriac region, and formed in the right lumbar region a prominence of considerable size between the last rib and

the crest of the ilium. That was the situation of the tumour, the size of the tumour, the extent of the tumour. (Fig. 1.)

Now these few facts, which I have drawn out from the case, were ample evidence that the tumour was not formed by diseased enlargement of the right lobe of the liver, by

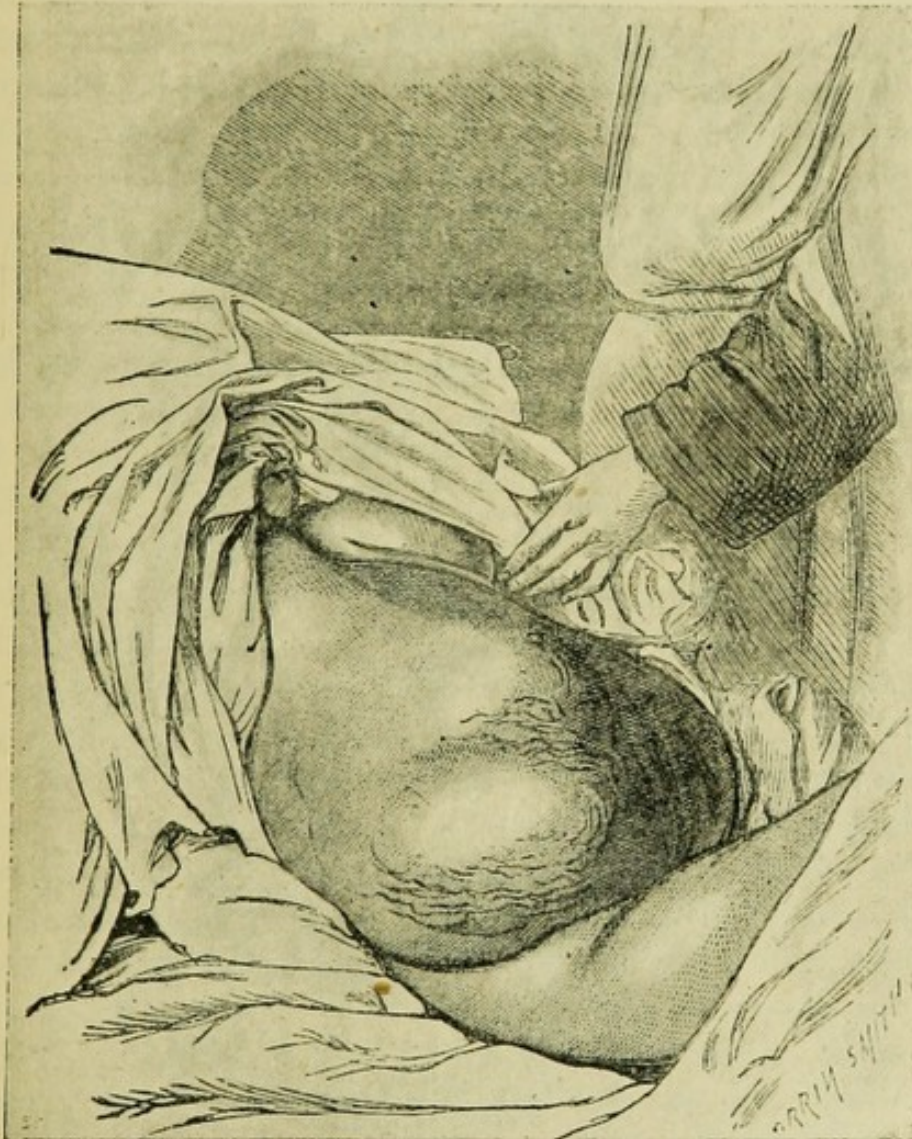
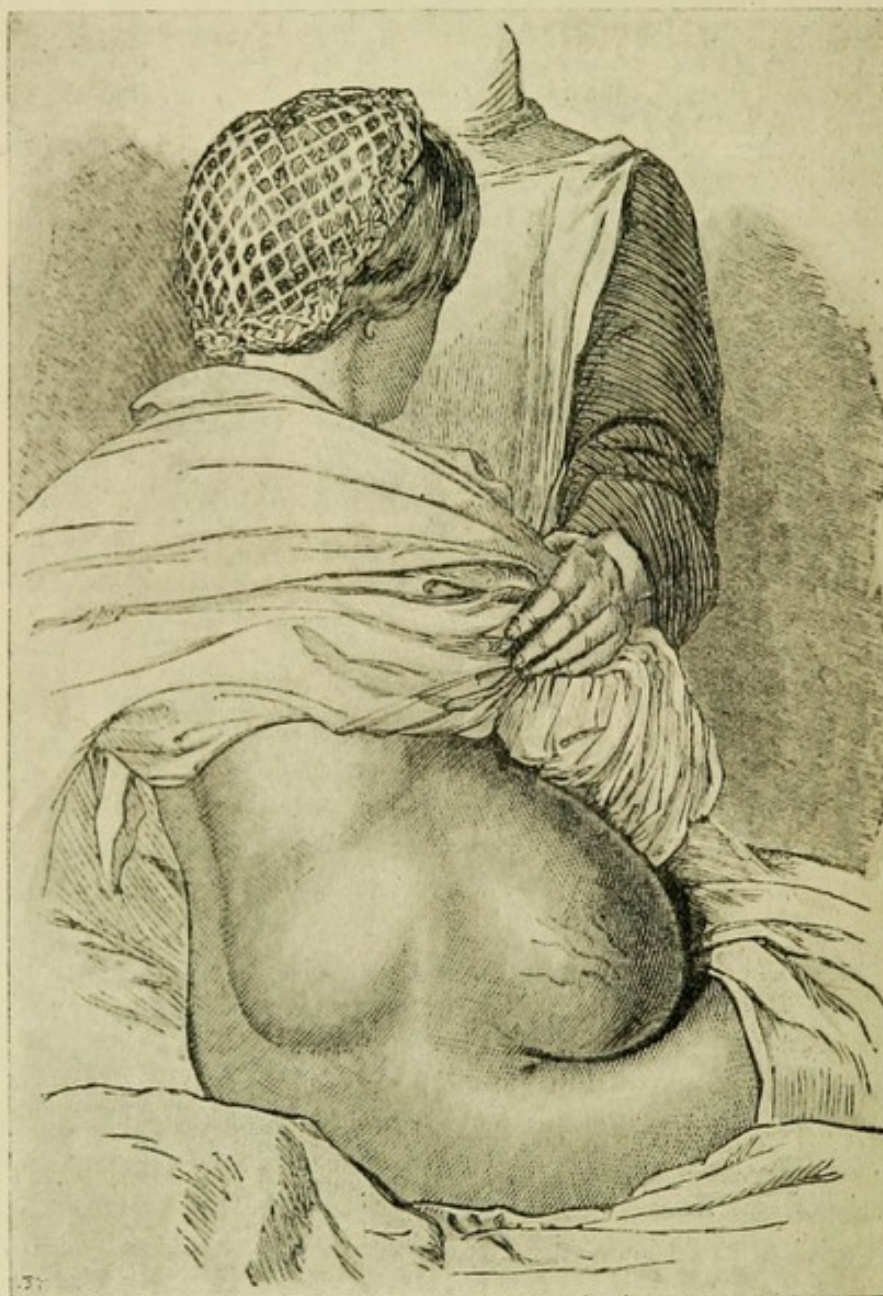


Fig. 1, from a photograph, shows the anterior aspect of the abdomen.

disease of the kidney, by the ovary, or by the mesenteric glands. No matter what the disease affecting the parts I have mentioned might have been, neither could have caused such a marked prominence in the back as is represented in the photograph (Fig. 2), and neither liver, kidney, nor mesenteric glands could have descended, as this tumour did,

into the pelvis. On making an examination *per vaginam*, we found that it passed down on the right of the pelvis, and pushed the uterus to the side. Again, a diseased mass originating in the ovary or in the mesenteric glands, would, ere



[Fig. 2 shows the posterior projection.

it had attained so large a size as this, have spread more to the left. The tumour in this woman scarcely passed the middle line. Note then in regard to this tumour these two facts—
1. The projection backwards, between the crest of the ilium and the last rib: 2. The small extent to which it passed beyond

the middle line in front. We thus excluded, without reference to the history, simply from the present condition, all enlargements of the liver, the kidney, the ovary, the mesenteric glands, in fact all possible abdominal viscera, as the cause of the tumour. The intestine was in front of the mass—of the left side of it, at any rate. This fact, conjoined with the history which I have read to you, the account of where it began, together with the above reasons for excluding abdominal organs as its cause, indicated that the tumour was post-peritoneal in origin, that it arose behind the peritoneum. Having come to the conclusion that it was post-peritoneal and a new growth, we then had to ask ourselves what was its nature. At the time when she came under observation, it was clear that the tumour was partly solid, and partly cystic; solid and cysts made up the mass. We knew it was solid by the touch and feel; we knew it to be cystic by the fluctuation and the elasticity, which were unmistakable evidence of the fluid, even without the exploring trocar. Now, whilst there was evidence enough in the touch to tell us that part was solid, the history of the case rendered it probable, in the very highest degree, that it was completely solid in its origin. The surgeon who first found it said he thought it was a 'bony tumour.' Nothing in the history could be better proof than was this remark, that the tumour was solid at its outset. It seemed probable, from the situation at the time when it was first discovered, which was very well described by the woman, that it was connected with the transverse processes or with some parts of the lumbar vertebræ, between the crest of the ilium and the last rib. So also it was probable that at that time it was very fixed, in close connection with the lumbar vertebræ. Again, the pain that was felt was not in the growth, but at the knee. It was the pain felt at the extremity of the nerves that were pressed by it. You know how frequently, in damage to the course of those nerves, the pain is felt in the knee. The loss of sensibility, and the contraction of the limb, all pointed to a degree of pressure that was little likely to be exercised by anything but a solid growth. A cystic tumour in its origin would not have produced any such pressure as that, at any rate not in the abdomen. It might have done so in a limited space, but not

in the abdomen. When we first felt the tumour, it was, partly at least, formed of cysts; but these, the history and symptoms indicated, were formed subsequently to a solid growth. At first the tumour increased slowly in size; then it grew rapidly, and it is probable that during that rapid growth the cysts were developed.

The conclusion to which we came, then, you may remember, soon after the woman's admission, was, that the tumour was solid—probably springing from bone, or at any rate in close connection with bone, growing in all directions, pressing on the nerves of the lower extremity; at first increasing slowly, and then rapidly, with the formation of cysts.

Now, an enchondromatous tumour may have these characters. Enchondromatous tumours spring from bone, from cartilage, from fibrous tissue. These are precisely the parts with which the tumour appeared to be connected. The lumbar vertebræ and the bones of the pelvis are common enough starting-points of the enchondromatous growths. An enchondromatous tumour is a cartilaginous tumour; it is formed of cartilage, as the name signifies. It is surrounded by a fibrous sheath, sending in processes of fibrous tissue, which run across the tumour. On these fibrous septa, the vessels are especially situated. The cartilage is not always like adult cartilage; it is commonly like foetal cartilage, and even a very rudimentary form of foetal cartilage. The consistence of an enchondromatous tumour varies a good deal. It may be quite solid, if resembling adult cartilage; or soft, if like foetal cartilage; and sometimes it has a jelly-like consistence only. At their origin, cartilaginous tumours are usually solid and fixed, just as this was. When large, they are elastic, and formed in part of cysts—a combination, as some have supposed, of cyst (?) and enchondroma—while others have supposed that the fluid has another origin. The cysts usually contain a fluid, such as we got from tapping one of these cysts; that is to say, a fluid of the consistence of white of egg, tenacious, and containing in it cells such as are represented in these drawings, and such as you find in cartilage. You find these floating in the fluid. Some persons have supposed that this fluid is formed by the softening down of pre-existing cartilage—by a retrograde metamorphosis. But the facts

given by Virchow and others to prove this origin of the fluid are by no means conclusive to my mind. As far as I have read, the evidence on this point consists of mere assertions—that is, as to the mode in which the cysts are formed. You find some solid cartilage, some gelatinous cartilage, some that is softer still, and you find fluid; therefore each of these softer conditions, it has been asserted, is a mere stage of the more solid. I am inclined to the opinion that, in some cases at least, the fluid is formed from the first, and is formed at once fluid, and not by solid softening down. The cartilage-cells are in it, but they are quite free; and, instead of the connective matter between the cells being a solid matrix, it is a fluid.

I will tell you two arguments deducible from this case which are against the view that the fluid was formed by a softening down of the matrix. There was no oleine, or next to none, in the fluid removed. The second argument is that this fluid reformed. It was not cartilage that formed in the cyst and softened down, but fluid identical with that removed by tapping reformed. As the internal surface of the cyst was then capable of forming a fluid similar to that which we removed, is it not probable that the primary fluid was formed in the same way? It is very unlikely that the lining membrane of the cyst should form a fluid identical with that which was formed by the mere softening down of a pre-existing solid. These seem to me strong arguments. It is, however, a matter of no moment to us, as clinical physicians, how the fluid is formed. I merely mentioned the point, because the case in some particulars is an uncommon one, and the subject one of considerable pathological interest.

We came to the conclusion during life, then, that it was an enchondromatous tumour, with cysts in it, such as are often found in enchondromata. Several other views as to its nature were, however, possible. We thought of a collection of hydatid cysts; but, on considering the matter, the character of the solid part and the solid origin of the tumour excluded the idea. It was, indeed, a mere thought passing through one's mind. The extreme degree of pressure that led to the paralysis and to the contraction of the limb should have excluded the idea of hydatid cysts.

Again, there was the question, Was it malignant? I showed that photograph yesterday to a gentleman—a good pathologist—and that was the first question he asked me: ‘Is it malignant disease?’ No, it was not. We excluded that idea for these reasons: the history, the duration of the disease, the solid nature of it at its origin, and the little the patient’s health suffered for so long a time, were all opposed to the idea. When the patient came under observation, she was in pretty good health, except for the great bulk of the growth. She was fairly well-nourished, and had none of the complexion of cancer which she would have had if this had been a rapidly growing cancer. Again, the consistence, the elasticity, and the mode of growth, were all opposed to the idea that it was malignant disease; and, on the introduction of the trocar, the character of the fluid removed seemed to render it improbable, almost impossible, that it could be malignant. At the same time, we could not say that a portion of the tumour was not malignant. A combination of enchondroma with malignant disease is not uncommon, so that there might have been, superadded to the original enchondroma, a certain amount of cancer. We could not say positively that there was not, although we had no evidence that there was, and some facts, *e.g.* the aspect of the patient—against it.

In the treatment of the case, there was nothing more to do than to alleviate the suffering of the patient. The end was certain. How long it might be delayed, no one could say.

She died, you will notice, from an attack of erysipelas of the leg. That is a very common mode of termination in all chronic non-malignant diseases. I do not mean by erysipelas, but an acute attack of some sort. Some have a little pleurisy, some die from pneumonia, some from peritonitis, from a trifling inflammation, a catarrh, a bronchitis. It is rare for patients to die directly from a chronic disease. The rule is for a slight acute illness to carry them off.

There are a few special points in the case to which I desire to direct your attention.

Remember the pain felt first in the hip, then in the knee, but most severely in the knee. Let that impress upon you how tumours in this situation are not uncommonly attended

by pain in the knee; and, therefore, having a patient complaining to you of this pain, do not be content with a careful examination of the hip, but look to the abdomen to see if you can find anything there to account for it. You see that six years and a half before she came under observation, after the birth of a still-born child, the patient was seized with pain in the right hip and pain in the right knee. Probably the tumour began about that time, and perhaps made a little advance then, but it was only two years and a half afterwards that she became aware that she had a tumour, and then it was the size of a cricket-ball. A patient often has a tumour in his abdomen of which he is not cognisant till the growth has attained a considerable size, and then the patient will tell you that the tumour has grown rapidly; that it has only been of so long standing. He will say it has only been there six months, or a year, which means that it is only for that time that he has known of it, though the tumour may have been there for an indefinite time.

She had suffered occasionally from vomiting her food. This is a common symptom of abdominal tumours. It may be produced mechanically by the tumour pressing on the stomach. It was probably produced in that way here. Or, the tumour may cause vomiting by leading to peritonitis. In some cases, the vomiting probably takes place after a sudden increase of size of the tumour in the direction of the stomach. Then, after a little time, the stomach gets accustomed to the increased pressure, and the vomiting stops. Remember, then, these two causes of vomiting in abdominal tumours—leaving out affections interfering with the function of the kidney—pressure and peritonitis. Another point: the woman, it is said, had a faint trace of albumen in her urine, and when it stood, a little pus fell; but the woman was the subject of leucorrhœa, and was very likely to have a little leucorrhœal fluid mixed with the urine. The leucorrhœal fluid contains pus and albumen, and thus you will find a trace of albumen in the urine, just as a man with a gleet, with a little cystitis, with a little pyelitis, will have pus in the urine, and with the pus will be a trace of albumen. In these cases, remember that the quantity of albumen is in proportion to the quantity of pus. It sometimes requires considerable care to prevent the

admixture of purulent matters with the urine, especially in women. The œdema of the leg was entirely due to the pressure on the veins in the pelvis—on the iliac vein, not on the vena cava, or, of course, both legs would have suffered, whereas it was only the right that became œdematous. When the tapping took place, the size of the tumour was lessened and the œdema diminished. The tapping was attended with a little peritonitis, a very common consequence of tapping cysts in the abdomen. The woman's kidneys, you see, were healthy. If she had been the subject of albuminuria and had had such a peritonitis, the chances would have been vastly against her recovery from it. The tapping however afforded only a temporary relief, for the fluid returned. The size of the tumour is a matter of considerable interest. So far as I know, it is one of the largest enchondromatous abdominal tumours on record, if not the largest. The solid part, after letting out the fluid, weighed fifteen pounds.

And now, gentlemen, we will look at the tumour and also at some of these drawings and preparations of enchondromatous growths.

ON THE USE OF THE NEUTRAL SULPHITES
IN THE TREATMENT OF DISEASES
ATTENDED BY THE DEVELOPMENT OF
PARASITIC PLANTS,
ESPECIALLY THE SARCINA VENTRICULI

Delivered at University College Hospital on June 25th, 1851

CLINICAL LECTURE ON THE USE OF THE NEUTRAL
SULPHITES, IN THE TREATMENT OF DISEASES
ATTENDED BY THE DEVELOPMENT OF PARA-
SITIC PLANTS, ESPECIALLY THE *SARCINA*
*VENTRICULI*¹

THE great object, Gentlemen, in the treatment of diseases, is the removal of that lesion of structure, or that condition of the system on which the *symptoms depend*; but should it happen, either from our inability to detect the primary lesion,—the disease properly so called—or to remove it when detected, that we are compelled to alleviate suffering only, then we treat symptoms. Frequently, however, by the relief we afford to symptoms, we do more than alleviate distress; we prolong life, or haply even place the patient in such a condition that nature accomplishes what our remedies fail to effect, and what she would be incapable of effecting were not the symptoms referred to removed.

To-day I propose to bring before you a case which illustrates the fact, that the relief of a symptom may exercise a markedly favourable influence on what is, perhaps, an incurable organic disease,—and I seize this occasion the rather, because it affords me also an opportunity of illustrating the advantages that the science of medicine has derived from the progress of the collateral sciences, and of proving to you that the microscope is not a mere toy for the non-practical man, but ‘one aid the more’ in enabling us to determine the disease under which the patient labours, and so of treating him the more successfully, and that a knowledge of chemistry may serve, in *some cases*, to direct us in our choice of remedies. No better example than the case

¹ *Medical Times and Gazette*, August 23rd, 1851.

of James Martin could be offered of the advantages to be derived from the use of the microscope in rendering diagnosis more precise, and of the benefits chemistry may confer on therapeutics.

You have all repeatedly seen the man referred to in ward 4. You may remember his appearance when he entered the hospital—emaciated, with a worn and suffering-indicating countenance, generally sitting up in bed, with his knees drawn up to his abdomen, his head and shoulders bent forwards, his arms clasped round his raised legs, or lying on his face with a pillow forced by his clenched hands on the region of the umbilicus; bitterly complaining of the misery he suffered, of a dreadful burning sensation, a fire seated in the region of his stomach, and, every now and then, of ‘flashing pains,’ as of flames darting upwards, of constant evacuation of enormous quantities of flatus, and of one or two attacks of vomiting daily. For more than twenty years this man had been thus suffering. You may see him now in the ward offering a striking contrast to his former self. His face has in a great measure lost its pain-worn aspect, the eructation of flatus has ceased, and when on his bed he reclines as men in health. He is gaining flesh.

James Martin, now aged 66, the son of healthy parents, who died at the ages of 82 and 80 respectively, enlisted into the Artillery when 18 years of age, served in the Peninsula, was at Waterloo, and subsequently went with his regiment to North America, where he remained six years. He was discharged in 1824. After leaving the army, he worked in the Thames Tunnel, and was there when the water broke in. He was supposed to be dead when first removed, and for a year he attended as an out-patient at Guy’s Hospital. In 1829 he entered the Police Force, in which he continued for seven years. After leaving it, he entered the Queen of Spain’s service, and returned with the Legion in 1837. Though formerly of intemperate habits, he avers that for many years he has been very sober. With the exception of an attack of ague while in America, his health up to the time of the accident in the Thames Tunnel was excellent. From that event he dates the commencement of his present ailment. While in the police he suffered from intense pain at the epigastrium, and used often to retire into back streets, and there lean with his abdomen firmly fixed against an iron post. At first, the vomiting occurred once or

twice a week; afterwards, every day. It was at this time, he says, that he first vomited blood. From the time of the accident he has been subject to constipation, and long tried all sorts of quack medicines; and, to relieve the pain in the abdomen, took a teaspoonful of carbonate of soda twice a day. While in Spain he endured many hardships, but was not so much a martyr to his old complaint; and, for the first six months after his return, he enjoyed comparative health.

In the early part of 1838 he entered University College Hospital, and, by his own account, has been unable to work since. He has been at different times in- and out-patient at this and other hospitals, and, on one occasion, was an inmate of the parish infirmary. He was an out-patient when I was appointed to this Hospital, but too ill to attend himself. His wife came for his medicines. It was in consequence of the description she gave of the vomited matters (*viz.*, that they frothed over the vessel in which they were received like yeast) that I examined them microscopically. I then found the bodies described by Mr. Goodsir, in 1842, as *sarcina ventriculi*. When I first visited Martin, a year ago, this was his condition,—he was in bed and had not been up for more than a few minutes for several days. He said that, in consequence of pain in his abdomen, he slept but little. He was free from headache, and his complexion was clear, his skin was cool and there was no trace of oedema of the extremities. Neither the liver nor spleen was enlarged. There was decided tenderness at the epigastrium, and so far as this permitted manipulation, no tumour could be detected. He stated (and his countenance bore witness to the truth of his assertion) that he then was, and had been, suffering acutely about the umbilicus, above, below, and on either side of that spot; that the pain was ‘smarting and burning like a blister;’ that if he sat up, he felt as though he had ‘a cord around his bowels, tying them back to his spine;’ that he was in the habit of sitting in bed, with his knees drawn up towards his abdomen, his head and shoulders bent forwards, his arms resting on his knees; and that, while in this position, he would often get his wife to place some hard body—at one time a bundle of clothes, and at another, ‘the bellows’—between his knees and abdomen, the pressure of which seemed to give some relief during the paroxysms of pain. The pulse was only 72, the respirations 18 in the minute; his appetite was indifferent. His bowels had long been most obstinate, never acting without medicine, and that of the most powerful kind, croton oil, etc. He remarked, that whenever the vomiting diminished the pain increased, *so that he desired the vomiting to continue*. While an out-patient I tried for his relief nitrate of silver, hydrocyanic acid, creosote, morphia, henbane, large

doses of carbonate of soda, etc. I thought the morphia lessened the frequency of the vomiting, but, although it produced drowsiness, it seemed to increase the pain. With difficulty I persuaded him to come into the hospital, and, so satisfied was his wife that death would speedily terminate his sufferings, that after he had been in a few days, she earnestly entreated me to discharge him lest he should die here.

He was admitted into the hospital as an in-patient March the 15th, 1851, and on the 16th the following notes of the case were taken:—Mind and special senses normal; *arcus senilis* strongly marked; can walk with facility, but feels weak; expression worn, is extremely thin, skin cool, mucous membrane pale, no sallowness of complexion, no anasarca, legs drawn up towards his abdomen and his arms around his knees, shoulders bent forwards, his head resting on the elevated inferior extremities; tongue large, flabby, indented at the edges, and thinly furred; no dysphagia, appetite generally good; had for his dinner yesterday two potatoes and some broth; dined between twelve and one o'clock; at five o'clock P.M. vomited, ejected the whole of the matters now in the basin in about five minutes; the quantity of ejected matter reserved is two quarts.

Characters of the Vomited Matter.—Floating on the surface is a layer, from a quarter to half an inch thick, of a brownish colour, closely resembling yeast in general characters, entangling in its substance quantities of air-bubbles. *Microscopical Elements.*—Striated muscular fibres; *sarcinæ ventriculi* in great numbers, of a dark yellowish colour; *torulæ* very numerous, undistinguishable from the yeast-plant in size and shape; fat-globules and starch.

After eating his dinner felt comfortable and satisfied for two hours, then experienced considerable thirst; about four o'clock P.M. took a cupful of cold fluid. He thinks that cold fluids excite vomiting, hot prevent it; that cold fluids cause pain, hot fluids relieve it; from the time of drinking the fluid he felt severe burning twisting pain over nearly the whole abdomen; the burning pain was most severe about the epigastric region, the twisting pain about the umbilicus; to use his own expression, 'he felt in a burning flame;' much flatus was discharged before vomiting, chiefly by belching, each eructation of flatus was attended with severe sense of burning up the centre of the sternum, called by the patient 'a flashing pain;' after vomiting, the pain and burning were considerably relieved; still the pain continued during the whole night, increasing in severity at intervals. Five grains of extract of colocynth, with two drops of croton oil, were taken last night; these operated about eight o'clock A.M., producing three stools; the action of the bowels relieved a sensation of tightness. He had this morning for his breakfast, about eight o'clock A.M., a cup of

tea and a little bread and butter. At twelve o'clock complained of a nipping pain between the umbilicus and pubes, a burning pain from the epigastrium upwards over the right mammary and sternal region; no vomiting to-day; complains of tightness and fulness of abdomen. Abdomen convex; convexity less marked at epigastric and hypochondriac regions than below the umbilicus; interval between recti muscles distinct; tenderness in epigastric and umbilical regions; slight tenderness also in hypogastric; more in hypochondriac and umbilical regions. Some sense of resistance on pressure over recti muscles; resonance generally normal; no tumour to be detected in any part of abdomen; there is, however, greater fulness in the vicinity of the pylorus than elsewhere (from tenderness and distension the manipulation was less deep than subsequently); passes his urine freely; pulse 60, regular; lungs¹ and heart generally healthy, only the second sound at the base is murmurish. The stool contains a large amount of yellow amorphous matter, sarcinæ, and triple phosphates; its reaction alkaline.

The daily reports from this time tell of his continued suffering; of the frequency with which he was observed in what is termed, in some of the reports, his old position.

On April the 22nd the following report was made:—

At $\frac{1}{2}$ past 11 P.M. the twisting and burning pain came on, and he brought up about 80 ounces of fluid at two efforts; then drank about half a pint of gruel, after which he continued eructating flatus and vomiting a little fluid till 3 A.M.; the fluid last ejected was about 40 ozs. in quantity. He describes the pain which preceded the vomiting to have been of a bursting kind. He says he felt 'as if being torn to pieces.' He complains that during the night he suffered from headache and vertigo. The abdomen is now very tender; he complains of a bitter taste in his mouth, which he says is always full of water. Bowels acted last night; pulse 76. The vomited matter was strongly acid, and resembled in all particulars that above described, and the stools were as before alkaline, and contained sarcinæ.

On May the 29th the report states:—Much freer from pain; all sense of 'flashing burning' gone; lies ordinarily with legs extended, as if free from pain. His present state offers a striking contrast to his condition on admission. Bowels acted to-day. No stool the preceding three days.

As the hardness of the abdomen disappeared and the distension diminished, it was pretty clearly made out that the stomach was dilated, the stomach-note being audible to the umbilicus; and

¹ The case has been much abridged as regards the organs not bearing directly on the disease.

an ill-defined solid body was imperfectly felt about two inches to the right of the middle line, midway between the umbilicus and cartilages of the ribs.

His present state you have all witnessed ; he suffers little pain ; is freed from the eructation of flatus ; his position is unconstrained ; his abdomen is much less full than on admission ; he is gaining flesh ; the worn expression is comparatively trifling, and he sleeps well. The alteration in his general carriage and position strikes his wife and friends most forcibly. It is, he affirms, many years since he enjoyed the same state of comfort. I must briefly advert to the condition of his urine. It is acid when first passed, but becomes alkaline rather more quickly than healthy urine should. One of my excellent clinical clerks, Mr. Simpson, has been kind enough to examine it daily for some time, and he notices no difference in the degree of acidity, as manifested by litmus paper, whether Martin is vomiting or not. An analysis of the matter vomited before the man's admission to the Hospital was kindly made by Professor Graham. It contained a large excess of free hydrochloric acid, a little acetic acid, alcohol, and sugar ; the gas disengaged was carbonic acid. It was identical, in all essential particulars, Mr. Graham says, with the matter vomited by a man suffering from *sarcina ventriculi*, under the care of Dr. Bence Jones, in St. George's Hospital.

Now, did the primary disease in the case of Martin, consist in the presence of the *sarcina* in the stomach, or was the *sarcina* (as Schlossberger from his cases concludes it invariably is) merely an epiphenomenon ?

Let us see what lesions of the stomach experience has shown to be coincident with the occurrence of *sarcinæ*. It appears from cases on record, and especially from the observations of Dr. Todd, recorded in the *Medical Gazette* of May 2nd, that if the food is retarded in its passage through the stomach, *sarcinæ* are developed there, and that thickening of the pylorus, narrowing of the pyloric orifice, and dilatation of the stomach, are the lesions most frequently found after death in such cases. Now, the pylorus is, in its healthy state, closed by muscular action. The muscular fibres by which this closure is effected, are, I need not say, of the kind termed plain, *i.e.* they belong to the class of involuntary muscles.

Constriction of the pylorus may be organic or spasmodic. The pylorus is the seat of ulceration ; the ulcer heals ;

contraction of the cicatrix follows; and hence stricture of the pyloric opening. The submucous tissue is the seat of the exudation of lymph, simple fibrillating or contractile lymph, or of fibro-plastic blastema; permanent stricture of the pylorus is the result; or the same tissue is the seat of cancerous infiltration, and coarctation of the same part follows. An error in diet is committed; digestion is imperfectly performed; and the pylorus, healthy in structure, refuses to allow the offending substance to pass into the duodenum. It is a well-known law of the animal economy, that increased demand leads to increased supply; that when a part is called on to perform extra duty, not only is the nutritive material carried to that part in quantity sufficient to compensate for waste, but in still larger quantities; and hence hypertrophy of the over-exercised structure ensues. This is especially true of the muscular system, and peculiarly so of the involuntary portion of that system.

Again, experience proves that when, from any cause, coarctation of the orifice leading from a hollow viscus takes place, the viscus itself frequently undergoes dilatation: whatever, therefore, interferes with the free exit of food through the pylorus may lead to dilatation of the stomach.

Taking all these facts into consideration, it seems to me that the very presence of the *sarcinæ*, and of the fermenting intensely acid fluid may possibly, nay, probably (by keeping the muscular fibres that close the pylorus in constant action) be, in some cases, the cause of the organic coarctation of the pylorus and the consequent dilatation of the stomach, which are, as I tell you, so frequently the lesions found after death, where these vegetables have been detected in the vomited matter during life. But supposing that organic disease exists anterior to the development of the *torulæ*, and the *sarcinæ*, and to the secretion of the acid fermenting fluid, yet must we allow that the irritation produced by their presence would be likely to excite spasmodic closure of the pylorus, and so add, at least, to the difficulty with which the contents of the stomach would find their way through the organically coarctated orifice; while the rapid disengagement of gas could not fail to increase the dilatation of the stomach, already over-distended by the ingesta.

Thus, then, whether the primary disease be (as, in some cases I am disposed to regard it to be) the secretion of a fluid favourable to the development of the germs of *sarcinæ*, or whether the development of these bodies be the result of the retardation of the food in a dilated stomach, the consequence of a mechanical impediment to its transit through the pylorus (as in many cases I have no doubt *it is*); whether the vegetable organisms, and the fermentation which accompanies their development, be the cause or the consequence of the pyloric stricture, still must it be desirable to check their development and prevent their growth, as a means, if not of curing, at least of retarding, the progress of the organic disease.

But, allowing for a moment that our expectations could not reach thus far, yet would it still be a gain to prevent the distension caused by the disengagement of the carbonic acid, the product of fermentation, to check the formation of that flatus, the very eructation of which is a constant source of distress to the patient. 'The three symptoms,' says Dr. Walshe, speaking of cancer of the stomach, 'which most torture patients, and for which they most earnestly implore relief, are flatus, vomiting, and constipation.' But to return to Martin, and to the bearing of his case on the question here stated. Some years since he suffered from mechanical violence; subsequently he had hæmatemesis, suffered from pain in the abdomen, and vomited his food often many hours after eating it; and presumptive evidence is afforded, by percussion and palpation, of dilatation of the stomach, and thickening of the pylorus. When he first came under our observations, the symptoms, according to his own and his wife's account, had continued almost unabated in intensity for many years, and certainly they were as severe three years since as they were on his admission.

The long continuance of the stomach disease (more than twenty years) without marked alteration of the symptoms, without the implication of other organs, and without any indication of the existence of constitutionally malignant disease, as well as the fact that vomiting of blood was among the earliest of the symptoms, renders the existence of scirrhus of the pylorus improbable, while it renders the existence of

ulceration in the vicinity of the pylorus, cicatrisation of the ulcer, and subsequent thickening of the submucous tissue, extremely probable; but still, remember, only probable. Taking, however, this view of the primary stomach disease, I cannot but think that this organic coarctation of the pyloric orifice and dilatation of the stomach, have both been increased by the presence of the *sarcinæ*, the *torulæ*, the acid fluid that, under the stimulus of their presence, is poured into the stomach in such large quantities, and the gas which accompanies their formation. I have read to you some notes, which illustrate the improvement that has taken place in our patient's condition since his admission into the hospital. Let us now see by what means that improvement has been effected.

It appears from the notes, that, from his first admission till the 5th of April, he vomited almost daily an enormous amount of thick fluid in a state of fermentation, loaded with *sarcinæ* and *torulæ*. To give you an idea of the quantity, I may mention that, on March 10th, he vomited two quarts; the 17th, one quart; on the 19th, a pint and a half; on the 20th, one quart; on the 22nd, five pints; on the 24th, two pints and a half; the drugs given during the same three weeks were hydrocyanic acid, acetate of morphia, and sulphate of zinc. You will remember he had previously taken creosote, nitrate of silver, etc.

At this time, guided by the knowledge that *sarcina* is a vegetable, and having had my attention called by my friend Mr. Marshall to the fact that Professor Graham had suggested the employment of sulphurous acid in cholera, when that disease was supposed to depend on the presence of a fungus in the intestinal canal, as the agent the most destructive to vegetable life, and, at the same time, one that might be evolved in the human stomach with impunity, I administered the sulphite of potash.

The power exercised by the remedy has been most remarkable and most unequivocal.

Before taking it, he was vomiting daily from 40 to 100 ounces of the fluid, loaded with *sarcinæ* and *torulæ*; thus, on the 1st of April, he vomited 100 ounces; on the evening of the 2nd, between 70 and 80 ounces, although he had the

same morning, from the action of sulphate of zinc, vomited 50 ounces. No vomiting on the 3rd. On the 4th, in consequence of the extreme pain and sense of distension, sulphate of zinc was again administered.

On the 5th, he took $\mathfrak{z}$ ss. of the sulphite of potash early in the morning; in the evening he vomited. On the 6th the dose was increased to 1 drachm. On the 7th he vomited 12 ozs. only of acid fluid, on the surface of which there was but a small amount of scum; it contained perfect *sarcinæ*, but no *torulæ*.

From the 8th, he took 3 drachms of the sulphite daily, each dose being given in $\mathfrak{z}$ iss. of water. At 6 A.M. on the 9th, he ejected from the stomach 4 ozs.; the *sarcinæ* were now decidedly less numerous, and there were no *torulæ*. In the evening of the 6th he again vomited, but the vomited matters were now free from *sarcinæ* and *torulæ*, and there was appearance of fermentation. Between the 9th and the 18th he vomited three times, but on neither occasion did the vomited matter contain *sarcinæ* or *torulæ*.

On the evening of the 18th the sulphite of potash was omitted; and on the 19th, he vomited a fluid in a state of fermentation, containing *torulæ*, but no *sarcinæ*. On the 20th he vomited 9 ozs. of a similar fluid; these specimens were not examined by myself, but by a most trustworthy observer, Mr. Morris, the physician's assistant. On the 21st, there was a thick scum on the surface of the vomited matters, which contained *sarcinæ* and *torulæ* in abundance.

The sulphite of potash, in drachm doses, was again administered; no further vomiting occurred till the 27th, when, without any apparent cause, it recommenced; the vomited matters had their old yeast-like odour and appearance, and were loaded with *sarcinæ* and *torulæ*; I now, you will remember, began to suspect that the man could not be taking his medicine regularly, but on inquiry, no omission could be discovered. The medicine itself was then tested, and it was found that it gave off no odour of sulphurous acid, on the addition of a stronger acid. At the dispensary we learned that a fresh stock of the drug had come in on the 20th. On submitting a specimen to Mr. Graham, he found that it contained no trace of sulphurous acid. At his suggestion I

prescribed from this time the sulphite of soda, a more stable salt, and one less liable to be decomposed either in the preparation or by keeping.

On the 29th of April, half a drachm of it was given at bedtime, and on the 31st, half a drachm three times a day. On the 1st of May, our patient vomited 40 ozs. of a fluid covered with a thick scum; the vomited matters were not examined microscopically.

On the 2nd, the dose was increased to a drachm. There was no vomiting till the 5th; the vomited matters were in quantity about 50 ozs.; had no scum floating on their surface, and were entirely free from sarcinæ and torulæ.

On account of the burning pain which he occasionally complained of, I now ordered him a mixture containing sodæ carb., two scruples in each dose. On the 13th, 15th, and 16th, he again vomited sarcinæ in quantity, and as I suspected that the soda prevented the evolution of sulphurous acid, I omitted the carbonate. From the 16th of May to the 19th of June, the vomited matter contained no trace of sarcinæ or torulæ, and had lost its yeasty odour and appearance. *During these thirty-three days he ejected about 230 ozs. of acid fluid; during the first eight days of his residence here he ejected 380 ozs.*

On the 19th of June, the vomited matters contained a considerable amount of sarcinæ. On inquiry at the dispensary, I found that the stock of sulphite was nearly exhausted, and on testing it by the addition of a stronger acid, it gave off but little odour of sulphurous acid.

I increased the dose to ℥iv., and on the next day to ℥iss.

From the 19th of June to the 1st of July he vomited once only, and the ejected matter contained no sarcinæ.

About the end of June my attention was directed, by Mr. Thompson, of Croydon, to a case of chronic vomiting, with phosphatic urine (evidently a case of sarcinæ ventriculi), reported by Dr. G. Bird to have been cured by strychnia.

On the 30th of June the sulphite was omitted, and I gave strychnia in small doses, but the vomiting returned directly; the ejecta were found to contain sarcinæ and torulæ. On the evening of the 3rd the sulphite was again given; the vomiting ceased till the 10th, and then the vomited matter

contained neither *sarcinæ* nor *torulæ*, and were free from the yeast-like head. On the 14th vomiting occurred, but no *sarcinæ*. As I was desirous of examining the *sarcinæ* for the purpose of inquiring into their mode of development, I directed the sulphite to be omitted on the 16th. On the 17th, 18th, and 19th he vomited large quantity of the yeast-like fluid, containing abundance of *sarcinæ*. On the 20th he resumed the use of the sulphite; and although yesterday, *i.e.* the 24th, he vomited about 3 ozs. of acid fluid, still it was transparent, and had no trace of *sarcinæ* or *torulæ*. Now, gentlemen, I wish particularly to impress upon you the marked improvement that followed in this case from the cessation of the production of the *sarcinæ*. Coetaneous with their disappearance, the sense of distension and the terrific eructation of flatus ceased; the burning pain at the epigastrium abated considerably, and the pains which our patient described as 'flashing, darting upward' vanished. By preventing the formation of the *sarcinæ* and stopping the fermentation, we have, then, greatly relieved his sufferings; and allowing the existence of organic stricture of the pylorus, we have placed the patient in a much more favourable position than before, for we have removed a cause of constant spasmodic constriction of the pylorus, and have placed the stomach in a much more favourable position to resume its normal dimensions, by preventing the formation of the gas, one cause of its constant distension; and it is obvious, that if a dilated, hollow, muscular viscus is to resume its normal size, it must never be allowed to become distended.

But perhaps it may be supposed that diet has assisted in removing the *sarcinæ*. Not at all, for Martin has had a pretty liberal supply of arrowroot, beef-tea, mutton, milk, bread, and potatoes, with two ounces of rum daily. Sometimes the meat has been replaced by fish; sometimes the diet has been chiefly animal, sometimes vegetable; but this has exerted, apparently, no influence on the formation of the *sarcinæ*. It is true the *sarcinæ* return directly the sulphite of soda is omitted; but then, even though it were necessary for this man to continue its use for life, it would be no great trouble. Sulphite of soda is not more disagreeable to the palate than carbonate of soda, and is less injurious, and

many take the latter daily. But I hope, by steadily pursuing its use, by the employment of unfermented bread as to-day suggested by Mr. Graham, and by some other alterations in his diet and drugs, that ultimately he may be able to omit the sulphite entirely.

I cannot conclude without observing, that considerable benefit may be anticipated from the employment of sulphurous acid in all diseases attended with the development of parasitic plants. I would mention *porrigo* especially. That it would prevent the growth of the epiphyte there is little doubt, and, should it do so and yet not cure the disease, the question of the relation between the two would be settled.

In some forms of dyspepsia, attended with the discharge of large quantities of flatus, I think I have seen considerable benefit from its use.

On the whole, I am satisfied that in the sulphite of soda we have a valuable addition to our *materia medica*.

Let me advise such of you as may be tempted to employ the neutral sulphites, to test before administering them. An omission of this precaution may lead to disappointment.

Now, Gentlemen, I think that I have proved my point with reference to the value of the microscope and chemistry. Without the microscope the vegetable bodies could not have been detected, and without the aid of the chemist the remedy for them would have been unknown; and I think also that I have shown you that more than temporary benefit sometimes accrues to the patient from the treatment of individual symptoms.

ON SARCINÆ IN THE VENTRICLES
OF THE BRAIN

1853

ON THE NATURE AND EXTENT OF THE VENTRICLES

OF THE BRAIN

1838

ON SARCINÆ IN THE VENTRICLES OF THE BRAIN

ON A CASE IN WHICH THE SARCINA VENTRICULI, SEU SARCINA GOODSIRII, WAS FOUND IN THE FLUID REMOVED FROM THE VENTRICLES OF THE HUMAN BRAIN¹

THE sarcina Goodsirii has been found, not only in adults and children, but also in the lower animals—viz., the rabbit, the dog, and the tortoise.² It is by no means rarely present in the matters ejected from the human stomach and intestinal canal. In 1847, Heller³ described a case in which he discovered sarcinæ in the urine; subsequently, Dr. Mackay⁴ detected them in the same fluid; and Virchow⁵ and Zenker⁶ in the lungs.

To all of the last-mentioned cases it has been objected that the sarcinæ were in reality derived from the stomach; that when found in the urine, some of the vomited matters had been accidentally mixed with that fluid after its escape from the bladder; that when found in the lungs, the sarcinæ had passed into them during the act of vomiting. The more recent observations of Heller⁷ have, however, placed beyond a doubt the fact that sarcinæ Goodsirii are occasionally present in the urine when that fluid is free from admixture with vomited matters. In one of the two cases last described by

¹ From the *Brit. and For. Med. Chir. Review* for Oct. 1853 (vol. xii.).

² Canstatt's *Jahresbericht*, Dritter Band, 1850.

³ Eine eigenthümliches Harn sediment. *Archiv für physiologische und pathologische Chemie und Mikroskopie*, Heft. 4.

⁴ *Lectures on Clinical Medicine*, Dr. J. H. Bennett, July 1851.

⁵ Schlossberger, Die Sarcina, Württemberg, *Corresp. Bl.* N. 26.

⁶ Henle's *Zeitschrift*, Band iii.

⁷ *Archiv für physiologische und pathologische Chemie*, etc., 1852, p. 30.

Heller, the urine was examined for many weeks in succession, and a sediment in it of an inch in depth sometimes observed, composed of sarcinæ only, or of sarcinæ mingled with a little carbonate of lime. The following case lends support to the foregoing evidence in favour of the opinion that the stomach and intestinal canal are not the exclusive seats of the development of the sarcinæ Goodsirii.

A boy, aged 4 years, was admitted under my care into the Hospital for Sick Children, June 24th, 1852; he died July 3rd. The child was of fair complexion, well-made, and moderately stout. His illness was reported to have commenced on June 17th, with pain in the head. On the 19th, he was taken as an out-patient to King's College Hospital; subsequently to that time he never complained of headache, only he seemed heavy. He did not keep to his bed till the 23rd. After his admission into the Children's Hospital, the most prominent symptoms were—drowsiness, talking in sleep, an irregularly diffused scarlet rash on the skin, redness and swelling of the tonsils, with a white patch on the left tonsil, frequent pulse, sordes about the teeth, and some dryness and brownness of the tongue. On the 30th my notes say, 'Almost incessant grinding of the teeth; seems quite sensible when awake; asks for the cold wash to be applied to his head.'

Between the 30th of June and the day of the child's death—i.e. July 3rd—restlessness, strabismus, inequality of the pupils, and redness of the conjunctivæ. General convulsions commenced about 4 A.M. on the 3rd, and continued till the child's death at $\frac{1}{2}$ -past 4 A.M.

The examination of the body was commenced ten and a half hours after death. The vessels of the dura mater were more filled with blood than is usual. Numerous semi-transparent grey granulations were seated *on the arachnoid lining of the dura mater*, and a few similar granulations on the visceral arachnoid. The arachnoid itself was dry; the cerebral convolutions were flattened; the minute vessels of the pia mater, on the surface of the convolutions, were abnormally injected with blood. In the grey matter of the cerebrum were about fifteen masses of yellow tubercle—the largest was oval, $\frac{1}{2}$ an inch by a $\frac{1}{4}$ of an inch—the smallest about the size of a very large pin's head. The pia mater, dipping between

the convolutions, was studded with grey granulations. Four ounces of colourless serosity were removed from the lateral ventricles—that which first escaped on opening the ventricles was transparent, that which flowed towards the last was turbid. The fornix and septum lucidum were white and of a creamy consistence. Viewed from within, the floor of the third ventricle was highly vascular. The membranes covering the base of the brain were opaque, tough, and loaded with serosity. The fluid removed from the ventricles was alkaline and albuminous; after standing twenty-four hours the deposit of albumen constituted one-seventh of the fluid tested. In the cerebellum were several masses of yellow tubercle.

Microscopical characters of the softened Fornix and Septum Lucidum.—There were no granular corpuscles, no free fat-granules, detected in the softened septum lucidum and fornix. In the cerebral substance, adjacent to some of the tubercles, were numerous large granular corpuscles.

The peritoneum was studded with grey granulations; it was abnormally vascular. Grey granulations studded the pleuræ, pericardium, and lungs; the bronchial glands were stuffed with tubercle; the liver was dotted throughout with small transparent grey granulations; yellow tubercles in considerable number were found in the spleen and kidneys; there was an ulcer on either tonsil. The small and large intestines were extensively ulcerated; the edge and floor of some of the ulcers were covered with tubercles.

July 5th, 11 A.M.—The fluid removed from the lateral ventricles of the brain was examined more particularly than it had previously been. After its removal from the cerebrum, on the 3rd, the fluid had been kept in an open glass vessel, in a large, light, and airy room. It turned turmeric paper brown; its odour was sickly, brain-like, not ammoniacal; it seemed as if just beginning to decompose. There was a little sediment at the bottom of the vessel.

A drop of the fluid containing some of the sediment was examined with a magnifying power of about 200 diameters; the following objects, *and those ONLY*, were contained in it:

1. A considerable number of spherical bodies $\frac{1}{4000}$ th of an inch in diameter, of a pale yellowish colour (blood-discs altered in form?)

2. Square bodies, each side of which measured $\frac{1}{2000}$ th of an inch ; some were a little larger than this, others a little smaller. The surface of each body was divided by cross lines into four equally-sized compartments, and each of these quarters was again divided into four. In some of the bodies the lines producing the secondary quarterings were well, in others imperfectly, marked ; while in others these secondary quarters were themselves divided by cross lines into four parts. The angles of the bodies were somewhat rounded. Some of these bodies were, my notes state, 'as well-formed sarcinæ as I have ever seen.'
3. Oval bodies about $\frac{1}{5000}$ th of an inch in breadth, and $\frac{1}{2500}$ th of an inch in length, distinctly divided by a transverse line into two equal parts ; each of these two parts being very obscurely divided by a longitudinal line into two other parts.

The fluid removed from the pericardium had been standing side by side with that removed from the ventricles of the brain ; it was alkaline, but contained no sarcinæ.

It was a question when this child came under observation, whether or not he was suffering from scarlatina. The fatal termination was evidently the result of acute tuberculosis. Unfortunately, the fluid from the ventricles of the brain was not examined for forty-eight hours after its removal from the body. The questions, therefore, arise—Were the sarcinæ in this case developed after the fluid in which they were found was taken from the cerebral ventricles ? Were they developed after death, but within the body ? Did they exist in the ventricles while the child was yet living ? To whichever of these questions the answer be in the affirmative, the occurrence of sarcinæ under the conditions mentioned is remarkable.

The fluid in which sarcinæ have been found in the stomach has been on all occasions acid ; and in the same situation vinous fermentation has been their constant concomitant. So invariably have the *torulæ cerevisiæ*, and sarcinæ Goodsirii been found together, that Simon of Halle¹ has maintained

¹ Ueber die Entwicklung der Sarcine aus dem Hefenpilze. Virchow's *Archiv*, Band ii.

that the latter are merely an advanced stage of the development of the yeast plant. In the case I have detailed, as in one of Heller's cases, the fluid in which the sarcinæ formed was alkaline, and in it there was no trace of torulæ, and no evolution of gas. Supposing the sarcinæ to have been developed in the fluid after its removal from the body, then this case stands alone, inasmuch as they have never heretofore been known to form, except in the interior of the bodies of animals.

As to the stages in the development of the sarcinæ, they would appear from this case to be briefly these. A simple cell is divided into two parts by a transverse line; each of the two cells thus formed is again divided into two by a longitudinal line; each quarter of the primary cell subsequently experiences the same changes as the primary cell itself. This description of the mode of development of the sarcinæ differs *in toto* from that given by Simon of Halle,¹ who states that the primary cell increases its size and changes its form by the formation of nuclei in the interior; and as widely from that given by Pockel,² who affirms that the increase in size of the primary cell is partly the effect of endogenous cell-formation, and partly of gemmation, while comparatively it differs but little from that given by Frerichs.³

¹ *Loc. cit.*

² Nonnulla de Sarcina Goodsirii, Würzburg.

³ Ueber Sarcina Ventriculi. Häser's *Archiv*, Band x.

A BRIEF ANALYSIS OF THE APPEARANCES
OBSERVED IN THE BODIES OF SEVEN-
TEEN PERSONS 'BURNT TO DEATH'

1891

A NEW SYSTEM OF THE ALPHABET
OR
A METHOD OF WRITING IN THE ROMAN OR
ITALIAN CHARACTER, WHICH IS
EASILY LEARNED, AND WHICH
IS THE MOST PERFECT AND
USEFUL OF ANY THAT IS
NOW IN USE.

1721

A BRIEF ANALYSIS OF THE APPEARANCES
OBSERVED IN THE BODIES OF SEVEN-
TEEN PERSONS 'BURNT TO DEATH' ¹

IN looking through my papers I came on notes of the appearances found after death in each of the fourteen persons who lost their lives in a house in the vicinity of Holborn destroyed by fire. As there were several points of interest in these cases, which at the time the notes were taken were not well known to the profession, in order to satisfy myself as to their accuracy and frequency I obtained permission, by the kindness of Mr. Wakley (then coroner), to examine the bodies of three people destroyed in a public-house burned down in Great Portland Street. I therefore have notes of the appearances found after death in the bodies of seventeen persons discovered dead in houses destroyed by fire. As some persons suspected that the fourteen persons in the house near to Holborn had died from the inhalation of arsenuretted hydrogen (there was close by and affected by the fire a collection of minerals), I killed some rabbits with arsenuretted hydrogen, some with carbonic acid, and some with carbonic oxide, all kindly prepared for the purpose under the direction of the gentleman who was then professor of chemistry at University College. In those killed by inhaling arsenuretted hydrogen, the blood itself, the liver and other viscera, the colour of which is chiefly due to the blood in their vessels, were dark greyish-black. The greater part of the blood was of the consistence of treacle, but in it were some hard blackish clots. It was clear from the colour of the blood and of the viscera that arsenuretted hydrogen had nothing to do with the death of any of the seventeen persons referred to. In the rabbits

¹ A paper published in the *Lancet*, August 22, 1891.

killed by inhaling CO_2 the blood and the viscera were purple; in those killed by the inhalation of CO they were brilliant red.

The several points of interest and importance to which I have referred may be arranged under the following heads:—

1. The rigidly flexed position of the limbs. The force required to straighten the flexed limbs, and the force with which after being straightened the limbs regained their rigidly flexed position, proved that the rigidity and flexure were due to shortening of the muscles. The shortening was the result of the muscles being cooked after death. We see the shortening and rigidity of muscles in joints cooked for the table. But, although the permanent flexion and rigidity were due to cooking, it may be that convulsive movements due to asphyxia had in some cases assisted in first flexing the limbs; had, however, the limbs not been cooked the rigor mortis would have ceased in the usual time, and the limbs then have been easily straightened.

2. The bright red colour of the blood and of all the viscera and mucous membranes present in the majority of the cases was without doubt due to death having resulted from the inhalation of carbonic oxide.

3. The fluid state of the blood. In two only of the seventeen cases were a few small clots present. The persons in whose bodies clots were found, it will be seen, lost their lives, partly at least, from mechanical injuries, the falling in of the walls, etc. The fluid state of the blood was apparently the consequence of the high temperature of the locality in which the persons were when they died from the inhalation of carbonic oxide, etc., and the very high temperature to which the bodies were subjected directly after death. When animals are killed by inhaling carbonic oxide at an ordinary temperature, the blood is coagulated very firmly.

4. The presence of fluid blood in the cavity of the arachnoid.¹ The fluidity of the blood and the high temperature of the spot in which the body lay were apparently the cause

¹ After birth, blood (so far as my own experience teaches) is rarely transuded into the cavity of the arachnoid. The causes of its transudation that I have noted are the changes in the blood due to malignant acute specific disease—*e.g.*, malignant scarlet fever and malignant typhus, in which diseases, as in the cases of 'burnt to death,' the blood is often fluid. In some cases of poisoning, blood has been found in the cavity of the arach-

of the transudation of blood into the cavity of the arachnoid in the cases recorded in this paper.

5. The protrusion of the tongue between the teeth, the firm clenching of the jaws on the tongue (so firm in two cases that blood escaped from the tongue) and the escape of frothy mucus from the mouth and nostrils. These symptoms seem to have been due to a convulsive expiratory effort, the consequences of asphyxia. The carbonic oxide made the blood bright red, but it took the place of the oxygen of the air, and the red corpuscles no longer carried oxygen in a form able to be applied to oxygenate the tissues.¹ The clenching of the jaw was permanent in the majority of the cases from the masseters being cooked after death. In one or two cases, when the body was first seen, the jaws were clenched from cadaveric rigidity, but in these cases it quickly passed away.

6. The presence of carbonaceous matter in the mucus contained in the larynx, the trachea, and in the majority of cases in the bronchi. The presence of carbonaceous matter in the air-passages was manifestly due to the inhalation during life of smoke.

7. The thymus in many cases was noted to be unusually large, having regard to the age of the subject. The cause of this I did not see grounds for stating.

8. The urinary bladder was always contracted.

It has often been a judicial question whether a person was burned to death, or was murdered and the body afterwards burned by the murderer to shield himself from the legal consequences of his crime. In answering the question thus raised the presence of clots in the blood or the fluidity of the blood would have to be noted; if the blood and viscera were of a brilliant red colour, then it would be clear that the subject had died from inhaling carbonic oxide. The presence of carbonaceous matter in the mucus in the air-tubes would prove that the person must have inhaled the smoke, and therefore could not have been dead when placed on the fire.

noid, as I found this in a case of oxalic-acid poisoning. Intra-cranial aneurisms, bursting, extravasate blood into the cavity—*i.e.* in these cases blood is extravasated, not merely transuded.

¹ Thus carbon monoxide, by combining with the hæmoglobin of the red corpuscles, and so preventing the corpuscles from acting as oxygen carriers, produces asphyxia through deficiency of oxygen. Foster, Bk. II. p. 609.

The protrusion of the tongue has been supposed to prove (see case of Countess Goerlitz) that the person was strangled before being burned. The cases recorded in this paper show that for such purpose the sign is worthless.

CASE I.—W. S——, æt. 10. This boy had on a cotton nightshirt and trousers. The hair of the scalp was covered with dirt and dust, but was only slightly singed. The shirt was partially destroyed by fire over the right arm, the right side of the body, and the back. There were several small abrasions on various parts of the back and lower extremities. Beneath the trousers there was some vesication, and around the vesicles the cutis was rosy red.

1. The arms were strongly flexed and rigid; they formed a right angle at the elbow. The fingers of the left hand were flexed on the palm of the hand, embracing the thumb. The fingers of the right hand were moderately flexed; the thumb was extended. Considerable force was necessary to overcome the rigidity of the elbow and of the fingers, and when the extending force was relaxed the forearm and fingers regained the flexed position.

2. The blood was remarkably bright red, and that before it was exposed to the air. The liver, spleen, pancreas, and kidneys were bright red. The mucous membranes of the pharynx, tonsils, uvula, and soft palate, of the larynx, trachea, and bronchi were bright red.

3. The blood was fluid; there was no trace of a clot in any part of the body. The heart was of normal consistence; both sides contained a moderate amount of fluid blood.

4. A considerable quantity of bright red fluid blood was found in the cavity of the arachnoid. There was no extravasated blood in the pia mater.

5. The jaws were firmly clenched, but the tongue protruded between the teeth. Frothy mucus had escaped from the mouth and nostrils, and a little blood from the tongue. There was a normal quantity of reddish fluid in the pericardium; very little in either pleura.

6. The bronchi contained a considerable quantity of mucus, dark-brownish in colour, evidently due to the inhalation of smoke.

7. The thymus gland was very large for the child's age.

The dirt and dust on the boy's scalp, and the abrasions of the skin on the back and lower extremities, rendered it highly probable that he had been injured by the dusty rubbish of the falling walls before he was burnt, while the vesications with rosy red margins beneath the trousers clearly proved that these parts had been burned before life

was extinct. The colour of the blood, liver, etc., and the carbonaceous matter in the mucus in the air-passage were incontestable evidence that CO and smoke were inhaled before death. The protrusion of the tongue, the clenching of the jaws, and the frothy mucus issuing from the mouth and nostrils were evidently the consequences of rapidly advancing asphyxia.

CASE II.—Alfred S——, æt. 15. This lad had on a shirt much burnt about the right side and posteriorly. The hair on the scalp was burnt off the left side; the whole body was burnt and charred posteriorly; the thorax and abdomen were anteriorly uninjured. The left arm was very extensively charred, but the hand and wrist were almost uninjured, except at places where there was vesication, as if from scalding water. The lower extremities, both anteriorly and posteriorly, were very much burnt.

1. The right forearm was flexed on the arm. The hand was partly contracted. The left forearm was flexed at right angles with the upper arm; the hand relaxed. The lower extremities were flexed at the knees, and both feet powerfully extended.

2. The substance of the heart was bright red. Kidneys bright red; spleen very pale.

The stomach and diaphragm were both ruptured.

3. The blood was fluid throughout the body; no clots in any vessel.

4. There was no blood in the arachnoid.

5. The tongue protruded from the mouth, and was compressed between the teeth. The jaws were firmly clenched.

6. The pharynx, larynx, trachea, and bronchi were all full of black mucus.

8. The urinary bladder was contracted.

Death in this case was caused by the inhalation of CO, by the obstruction of the air-tubes by mucus loaded with carbonaceous matter, and by the shock to the nervous system by the mechanical injury which produced the rupture of the stomach and diaphragm. The vesications on the left hand and wrist show that their cause—*i.e.* the local application of heated solids, air or water, was applied before the complete extinction of life.

CASE III.—Richard S——, æt. 52. The nightdress was almost destroyed.

1. The burns on the body were chiefly on the right side, and on that side the upper and lower extremities were quite rigid,

while the left were flaccid. There were a very few burns on the surface, with red halo around them.

2. The lungs were congested, and, like the other viscera, darker than the same organs in the majority of the bodies examined.

3. The blood in the right and left sides of the heart and in the vessels was generally fluid.

4. The cavity of the arachnoid contained a quantity of fluid blood; the red points on the cut surface of the brain were abnormally numerous.

5. The tongue was protruding from the mouth, and was compressed between the teeth.

6. In the trachea and bronchi was much mucus coloured by carbonaceous matter. The laryngeal mucous membrane was studded by some spots of extravasated blood.

Judging from the state of the laryngeal mucous membrane and the colour of the blood and viscera, this man died from asphyxia, the consequence of the inhalation of CO_2 . It is evident from the halo around some of the burns that they were inflicted before life was extinct. The rigidity of the right limbs was manifestly due to their having been cooked after death, the flaccidity of the left limbs to their not having been exposed to any great heat.

CASE IV.—Jesse S——, æt. 2.

1. The whole of the body was very much charred.

2 and 3. The substance of the heart was healthy, but of a peculiar pinkish hue. The blood in the right side of the heart and throughout the body was fluid and very red. The viscera and mucous membranes generally were bright pink.

4. There was a large quantity of bright red blood in the cavity of the arachnoid. The vessels on the surface of the brain was injected with bright red blood.

5. The tongue was protruding from the mouth, and was compressed between the teeth. The jaws were so firmly clenched that some blood was escaping from the tongue. Frothy mucus was escaping from the mouth and nostrils.

6. The pharynx, larynx, and bronchi contained mucus with carbonaceous matter diffused through it.

8. The urinary bladder was contracted.

Asphyxia from the inhalation of CO was the cause of this child's death.

CASE V.—Wm. H——, æt. 20. This man had on a night-shirt which was not in the least burned. It had been torn

off the chest: the right arm of the shirt was stained with blood. The trousers were pulled on as high as the knees; they were torn over the left knee. They were neither burnt nor scorched, but were very wet. The hair of the head was matted with blood, but not singed. There were three large lacerated wounds on the forehead and face. The right hand and the right and left arms were burned superficially at places. The lower jaw was fixed by the rigidity of its muscles (from cooking) about half an inch from the upper jaw; the tongue, however, did not protrude. At the back of the tongue, close in front of the epiglottis, were some small stony substances resembling mortar. In the pharynx, by the side of the glottis, was one pebble. The trachea and bronchi contained much bloody mucus.

2. The right side of the heart was gorged with dark fluid blood. The left ventricle was contracted; there were no clots on either side of the heart. The liver, spleen, and kidneys were all much congested with dark venous blood.

8. The bladder was contracted.

This man was manifestly killed by the mechanical injuries inflicted on him by a falling wall. The hard mortar and pebble in the pharynx had caused blood to escape and enter the trachea and bronchi.

It was evident, from the colour of the blood and the distension of the right side of the heart, that some defective aeration of the blood had resulted from the obstruction to the entrance of air into the lungs, by the bloody mucus in the air-passages. The rigidity of the jaws was due to the temperature to which the part was exposed after death. The fluid condition of the blood resulted from the heat of the air in which the body was at the time of death, and directly after death.

CASE VI.—Mrs. H——, æt. 50. This body was much charred, burnt, and blackened all over.

1. Both arms were flexed and rigid; both hands were clenched. The left leg was flexed; the foot extended to the utmost. The right foot was drawn backwards, so that the toes touched the heel. The right leg below the knee was so much charred about its middle that it fell off in the removal of the body.

2. The liver, spleen, and kidneys were very pale.

3. Both sides of the heart were empty; they looked as if they had been cooked. The lungs were shrivelled up, as if from the action of fire. In either pleura were 2 ozs. of red-coloured fluid.

4. There was no fluid in the cavity of the arachnoid.
5. The lips were slightly apart, and the tongue was protruding.

The notes of the case of this woman are too scanty to prove more than that her body was burned after her death; the protruding tongue renders it probable that asphyxia was the cause of death. I have no note of the contents of larynx, etc.

CASE VII.—Robert M. P——, æt. 28. The face was so much charred as not to be recognisable.

1. Both arms and both hands were flexed and rigid; both arms were deeply destroyed by fire.

2. The liver and other viscera and all the mucous membranes were bright red.

3. The left side of the skull was charred, and between the charred bones and the dura mater was a clot of cooked blood the consistence of putty, and half an inch thick. The heart was hypertrophied; the right side was greatly dilated, and contained a few minute firm clots, and a large quantity of fluid blood; with the exception of these minute clots, and the mass of cooked blood between the dura mater and the charred calvaria, the blood throughout the body was fluid.

4. There was no fluid in the cavity of the arachnoid; the brain was pink and hard, evidently from being cooked.

5. The tongue protruded from the mouth, and was clenched between the teeth.

6. In the larynx and the trachea there was mucus, very dark from mixture of carbonaceous matter, *i.e.* smoke.

The colour of the viscera leads to the conclusion that the man died from asphyxia, resulting from the inhalation of CO. This man was the subject of severe, long-standing disease of the heart. His death was probably hastened by the blow he had experienced on the head; that this blow was inflicted before death is shown by the clot of blood between the charred skull and the dura mater.

CASE VIII.—William S——, æt. 12. This boy had on a shirt and a calico blouse; both were burned posteriorly, but not at all in front. This difference was due to the heated state of the material on which the body lay.

1. There was no rigidity of the upper limbs. The legs were much burned, the left rather more than the right; both legs were flexed and rigid. The left foot was extended and rigid, evidently burned after death.

2. The lungs and the spleen were bright red in colour, the liver and kidneys were pale.

3. There were no clots in the heart or blood-vessels; the superior longitudinal sinus and the torcular herophili contained some red fluid blood. The right side of the heart was relaxed, the left contracted; both sides contained fluid blood, but no clots.

4. The arachnoid cavity contained a small quantity of red fluid blood.

6. The larynx, trachea, and bronchi contained mucus of a blackish colour, evidently due to inhaled smoke.

7. The thymus gland was rather large.

CASE IX.—Mrs. S——. The body and the extremities were extensively charred, the left side less so than the right. The face, especially on the right side, was so much destroyed that the features were recognised with difficulty. It was evident that some parts of the skin were burned before life was extinct, or just after, for around the burns on the posterior aspect of the left thigh there was a halo of redness, and the cuticle was raised from the cutis by serosity. The line of demarcation between the burnt and normal parts was at places well defined.

1. The right arm and the fingers of both hands were firmly flexed. When the rigidity was overcome, no sooner was the extending force removed than the flexion returned.

2. The mucous membrane of the pharynx was bright red, and sprinkled with crimson spots; the epiglottis was bright red, and its under surface was singularly spotted with red spots, varying in size from a point to a line. There was a little thickening of the aryteno-epiglottidean folds, evidently from effusion of serosity. The liver, spleen, and kidneys were pale.

3. The blood throughout the blood-vessels was fluid.

4. There was a large amount of bright red fluid blood in the cavity of the arachnoid. The vessels of the pia mater were perhaps a trifle fuller than natural.

5. The tongue protruded beyond the lower teeth. The woman's upper teeth projected considerably beyond the lower.

6. The mucous membrane of the larynx and trachea resembled the under surface of the epiglottis, and was covered with mucus, thickly streaked with carbonaceous matter.

7. The remains of the thymus gland were unusually large.

8. The bladder was contracted.

The state of the mucous membrane of the pharynx, epiglottis, larynx, and trachea was probably due to the heated air breathed when the smoke entered the air-passages.

CASE X.—Mary S——, æt. 5. The child had on a chemise and nightdress. Both were very much burned, especially posteriorly. The trunk and the upper part of both thighs were uniformly charred. The hair was burned off both temples.

1. The right arm was semiflexed, the knees slightly raised.
2. and 3. There was bright red fluid blood, and no trace of any clot throughout the vascular system. There was a large quantity of bright red fluid blood in the right auricle. The vessels of the cerebrum and cerebellum were injected throughout with bright red blood. The liver was rather pale, the spleen very pale, and the kidneys natural.
4. There was a quantity of bright red fluid blood in the cavity of the arachnoid.
5. The tongue was protruding and compressed between the teeth. Blood was escaping from the tongue, and frothy mucus from the mouth and nostrils.
6. The trachea contained some mucus of a blackish colour, evidently from the admixture of inhaled smoke.

CASE XI.—Harvey S.—, æt. 14. His day shirt and all the clothes on the body were very little burnt, rather more behind than in front.

1. The lower extremities, and especially the feet, were rather rigid.
2. On section, the lungs were bright red. The liver and spleen were pale. The mucous membrane of the larynx and the trachea was bright red.
3. The blood throughout was fluid. There were three drachms in the right side of the heart, and two drachms in the left.
4. On the convex surface of the brain in the arachnoid cavity was a large quantity of bright red fluid blood.
5. The lower jaw was relaxed, and the tongue was within the mouth.
6. The bronchi were filled with mucus loaded with black carbonaceous matter.
7. The thymus gland was large. The pleura contained a small quantity of reddish fluid; the pericardium two or three drachms.
8. The bladder was contracted.

Here, as in several other cases, the posterior part of the shirt was more burned than the front part. This, of course, arose from heat of the part on which the dying or dead body lay.

CASE XII.—William H——, æt. 53. On the body were a shirt and flannel waistcoat. The tails of the shirt were but little burned, the flannel waistcoat not at all.

1. The right and left arms and forearms were a good deal charred. Lower limbs not burned. The right arm and hand were strongly flexed and rigid; the left were semiflexed.

2. The substance of the heart was of a bright red colour. The colour of the other viscera was not recorded in the notes.

3. The blood in the heart and vessels examined was fluid.

4. The presence or absence of blood in the arachnoid was not recorded.

5. The body was on its back, and the mouth was open. A day or two before the body was examined the mouth was closed and the tongue was protruding. The jaws, therefore, were closed from cadaveric rigidity, and were not, as in the majority of the cases, clenched from the effects of cooking.

CASE XIII.—John H—, æt. 14. The clothing was a shirt and socks. These were very slightly burned in front, but more posteriorly. The body was covered with dust and dirt. The cutis and subcutaneous tissues were deeply burned. The burned parts had no well-defined margin or red halo around them. On the inner aspect of the right leg on three spots the cutis had been destroyed without any signs of having been burned. The cutis had also been knocked off the face and shoulders. A patch of extravasated blood was found on removing the scalp, about the size of a five-shilling-piece.

2. The lungs were of a bright red colour; in the right breast was an opening leading to a cavity in the pleura; the spleen and liver were pale.

4. There was no blood in the cavity of the arachnoid.

5. The jaws were clenched, but the tongue was within the mouth.

6. The trachea contained a quantity of blackish mucus; the discolouration was due to carbonaceous matter.

7. The thymus gland was unusually large.

8. The bladder was contracted.

This lad was probably killed by the falling walls of the house. The dust and dirt covering the body, the injuries to the skin, and the patch of extravasated blood under the scalp, all point to this.

CASE XIV.—Joseph P—, æt. 48. The right side of the trunk posteriorly and the right foot, calf, and part of the thigh were deeply charred.

1. The arms and fingers were flexed, and the legs semi-flexed. Considerable force was necessary to straighten them. They could not be straightened without the skin being cracked. The cuticle

could be separated to a considerable extent from the extremities, the nails being detached with the cuticle.

2. The muscles, the liver, and all the internal organs were bright red.

3. The blood was fluid in all parts of the body, and very bright red in colour. There was more than an ounce and a half in the right auricle and ventricle, and about half an ounce in the left side of the heart.

4. A considerable quantity of fluid was present in the cavity of the arachnoid, but no blood.

5. The tongue was protruded and clenched between the teeth.

6. The mucous membrane of the pharynx, larynx, and trachea was bright red. Some carbonaceous matter was present in the mucus in the larynx, and trachea. The bronchial tubes contained a quantity of mucus; but there was no appearance of carbonaceous matter in the mucus lower down than the trachea.

8. The urinary bladder was contracted.

The appearances above might prove that the cause of death was carbonate oxide asphyxia, and that the body was burned directly after the cessation of life.

CASE XV.—Maria S——, æt. 15. The child had on a frock and a small chemise. The frock was burnt around its lower margin; the chemise was uninjured beneath the clothing. The child was burnt posteriorly. A large triangular wound on the forehead extended down to the pericranium; the scalp was somewhat detached above the face.

1. Both the legs and the left arm were quite rigid.

2. The liver was bright red from its blood; the spleen palish pink. The lungs resembled the other organs in their bright red colour, due to the blood in them.

3. Both sides of the heart contained some bright red fluid blood.

4. Some bright red fluid blood was contained in the cavity of the arachnoid.

5. The tongue protruded a considerable distance beyond the teeth; the teeth compressed the tongue.

6. The trachea and bronchi contained a small quantity of dark mucus (smoke dried).

7. The thymus gland was large.

8. The urinary bladder was contracted, but contained one drachm of urine.

CASE XVI.—Harriet S——, æt. 15. The night-dress was burnt in a few spots in front, rather more at the back.

1. The right hand was across the body, the left flexed and raised towards the face; both slightly contracted. There was a superficial wound just above the left ear, from which a quantity of blood, sufficient to mat the hair on the left side of the head, had escaped. The face and forehead, like other parts of the body, were denuded of cuticle, and had a half-cooked appearance, evidently from exposure to heat after death, there being no vesication and no sign of reaction at the edges of the burns.

2. The liver was of a bright red colour, as were all the mucous membranes; the spleen was pale.¹

3. The blood was fluid; the right ventricle contained more than the left.

4. There was a quantity of bright red fluid blood in the cavity of the arachnoid, and a little reddish fluid in either pleura.

5. The tongue was protruded, and compressed between the teeth; frothy mucus was issuing from the mouth and nostrils.

6. The trachea and bronchi contained a large amount of mucus of a dark brownish or black colour, as if from inhaling smoke.

7. The thymus gland was large for the age of the subject.

This girl evidently died directly from asphyxia, the consequence of inhaling CO.

CASE XVII.—Sarah J——, aged twenty-five. The trunk was extensively charred; no red halo at the edges of any of the burnt parts. The skull was fractured in three or four places. The bones of the skull were charred at places.

1. On both sides the hands were bent on the forearm, the forearm on the upper arm; the fingers of both hands were clenched on the palms. The flexion and rigidity were extreme.

2. The lungs had a somewhat rosy tint; the kidneys were very florid; the liver and spleen not brighter than natural.

3. There were coagula on both sides of the heart.

4. There was a quantity of fluid, but no blood, in the cavity of the arachnoid.

5. The teeth were charred, but the tongue protruded a little between them.

6. The bronchi contained a large quantity of black mucus—blackened by the smoke inhaled.

7. The bladder was contracted.

¹ 'Among the appearances observed in animals destroyed by CO, Ssabinski has pointed out a bloodless condition of the spleen. This organ had a rose-red colour, but when a section of it was made scarcely any blood flowed from it.' Taylor, *Principles and Practice of Medical Jurisprudence*, second edition, vol. ii. p. 110.

The death of this woman was the result of mechanical injuries (fractured skull, etc.) inflicted by the falling walls.

The absence of any redness at the margins of the parts that showed signs of having been burned, leads to the conclusion that the burns on the body were inflicted after death. The death having resulted from mechanical injury, the body probably was exposed at once to a temperature high enough to prevent the blood coagulating.

P.S.—Dr. Ringer and some others, whose names have escaped my memory, assisted in taking the foregoing notes.

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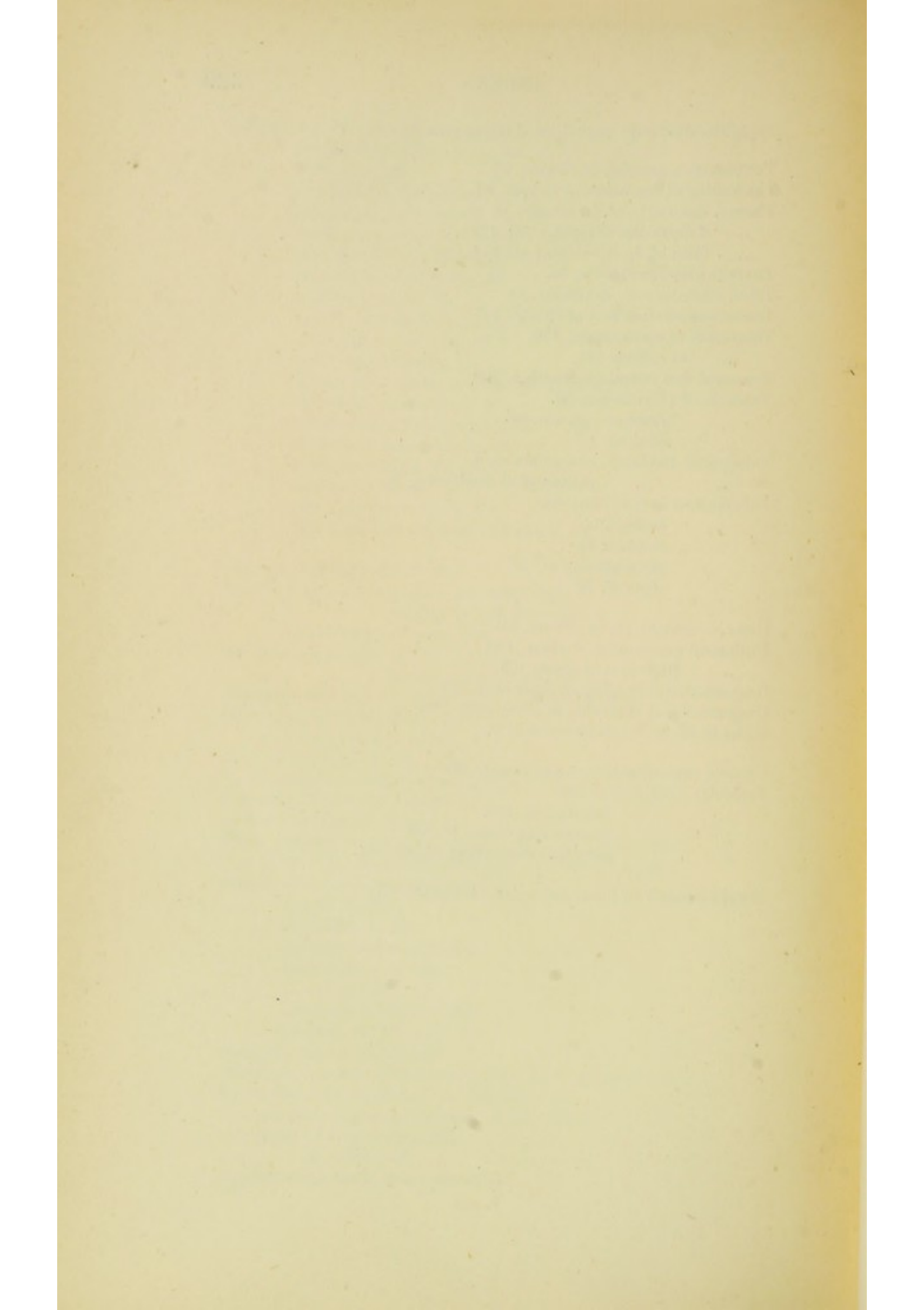
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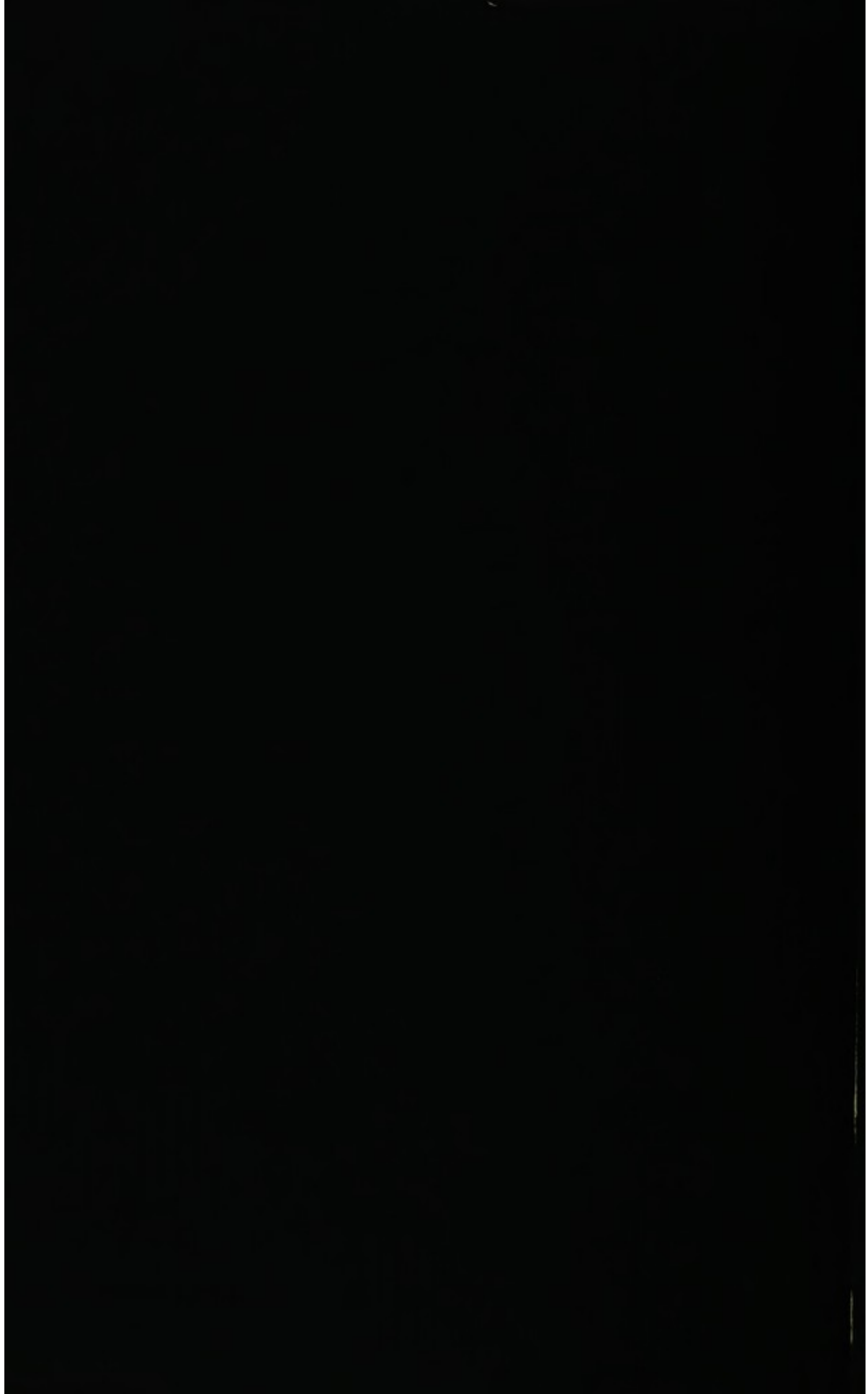
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