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NOTES ON PATHOLOGY.

NOTES ON PATHOLOGY

A HANDBOOK

FOR

THE POST-MORTEM ROOM

BY THE LATE

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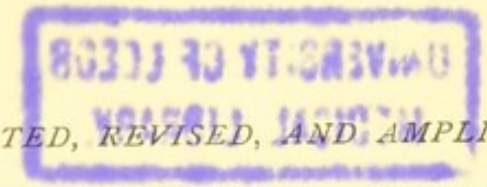

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MDCCCXCII

EDITORS' PREFACE.

THE story of the origin of this little book is well told by Dr. Goodhart in the Introduction. The Notes, as delivered by the late Dr. Carrington, were first published in the pages of the *Guy's Hospital Gazette* during the years 1887 and 1888; the eagerness with which they were then read by students induced the Gazette Committee to believe they would be of more use if published in book form.

While endeavouring to keep to the original as far as possible, the Editors have felt it advisable, here and there, to make some alterations in the wording and arrangement of the Notes, and in places to insert entirely fresh matter.

A debt of deep gratitude is due to Mrs. Carrington (the late Dr. Carrington's mother) for her kindness in permitting the Notes to be published first in the Gazette and now in their present form, and also to Dr. Goodhart, who has not only written the Introduction, but has very kindly looked over the proof-sheets, and given many very valuable suggestions.

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April, 1892.

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INTRODUCTION.

It may perhaps be said that the study of medicine at the present day carries along its working votaries chiefly in two lines: there are those who make a constant practice of frequenting the *post-mortem* room, and who check their clinical work, so far as is possible, by a careful comparison of the signs and symptoms observed during life with the appearances and conditions shown to exist after death; and there are those who approach, and obtain their knowledge of, disease far more by the projection, so to speak, of their physiological teaching upon the canvas of disease—a process which may, possibly, be adequate to the elaboration of a scheme of pathology of a sort, without any intervention of morbid anatomy.

It is needless to say that both methods of study should, and in the present day to some extent must, be combined; both are necessary for any complete study of disease. The physiological line, where it tends, as it may very readily do, to undue culture of an imaginative pathology, is checked by the revelations of the dead-house; while the rude facts of disease as seen there are revivified by a familiarity with the problems

that present themselves in the physiological laboratory.

From any point of view, however, it would seem that this little book may be of service to the student of medicine, for, while it presents itself as a handbook for those who make a practice of being much in the *post-mortem* room, and affords concise and carefully prepared synopses of the demonstrable facts of disease, it advisedly wants and demands, as its lamented author would have said himself had he been alive to write its introduction, the exercise of as full a measure of thought upon the *physiology* of degeneration and dissolution as the circumstances of the student admit of.

I say this because I should be loath—the Editors who have done their work so faithfully and well would be so too—to send the book forth with an imprimatur upon it that should dedicate it solely, or indeed chiefly, to the necessities of examinations. That it will be useful for such is its intention, but its chief purpose is to present a skeleton map to be filled in by experience as it is acquired.

The book originated in this way. It has now for some years been the custom of the Demonstrators of Morbid Anatomy at Guy's Hospital, as it is no doubt with those who teach pathology at other institutions, to supplement the making of the autopsy, which necessarily to some extent runs counter to any continuous exposition, by other demonstrations, in which, while still giving prominence to the actual exhibition of diseased structures, each organ in turn is taken in

review, and treated in somewhat ampler detail, by the aid of the microscope and museum specimens. The late Dr. Carrington was in the habit of giving an outline of his course of demonstrations of this kind to his class, and the present volume is composed of these. Thus, starting with tubercle in general, it passes on by natural transition to pulmonary tuberculosis, and then to pulmonary diseases of other kinds. Thereafter follow in order chapters on the heart, the liver, the kidney, the stomach and intestines, and the brain.

A work of this kind obviously does not aim at being exhaustive ; it does aim at grouping the description of the various common lesions of the several organs in compendious form, and thus at being a concise and trustworthy handbook to the *post-mortem* room. As such, after going carefully through it, I think it will prove useful. And I may add that if it does this, and in any way facilitates and encourages the study of morbid anatomy, it will have done something towards the spread of a branch of knowledge which, without any disparagement of the importance of other sources of information, is, I believe, more inspiring, more instinct with the gifts of a sober self-confidence and of comfort in the many exigencies of the practice of medicine, than any.

Much is required of the student of medicine at the present day, and he is ofttimes bewildered by the multitude of his studies. It is much to be feared that as years go on matters will not improve in this respect, and therefore it may be well to say, whatever else is

given the go-by, as much time as is possible should be set apart, while the material is at hand, for acquiring a knowledge of morbid anatomy. The field of clinical observation is always with us, but, student life over, the opportunity of making any extensive additions to our knowledge of disease as it shows itself after death is gone, and poor indeed, to my thinking, is he who is not well furnished in this respect. A knowledge of morbid anatomy will not, alas ! always enable us to cure disease, but it always puts us on the road for an accurate diagnosis, and an accurate diagnosis is an indispensable preliminary to any rational, and in the long run successful, treatment.

JAMES F. GOODHART.

CHAPTER I.

TUBERCLE.

DEFINITION by Ziegler:—"A non-vascular growth which never exceeds a certain size (millet seed), and which at some stages of its development undergoes caseation."

N.B.—It is quite true that large caseous tubercular masses are met with, but these are produced by the aggregation of the smaller masses.

Tubercle belongs to a group of pathological results which are known as Granulomata.

These are—

I. Tubercle.
II. Lupus Tubercle.
III. Gumma.
IV. Leprosy.
V. Actinomycosis.

Anatomical Structure of Tubercle.

In the centre there is often a blood-vessel, but this is not newly formed; the tubercle grows frequently round a pre-existent vessel.

It consists of a small round nodule, which contains the following elements:—

1. At the periphery there are **connective-tissue cells**, the nuclei of which are undergoing proliferation.

2. The main substance of the tubercle consists of **nucleated cells**, indistinguishable from leucocytes.

3. At the centre these cells will be found to have undergone **fatty degeneration**; some will be small and angular, but at the extreme centre they will be broken down into a structureless debris.

4. Large cells resembling epithelium with large nuclei will be met with (so-called **epithelioid cells**).

5. **Giant Cells.**—These are masses of protoplasm from ten to forty times the size of white blood corpuscles. They contain many nuclei; the periphery, according to Hamilton, is of firmer consistence than the centre, and gives off delicate threads of connective tissue, in the meshes of which the above-described cells are contained.

These giant cells are not peculiar to tubercle; they are found in granulation tissue and in sarcomata; probably the best specimens are met with in myeloid sarcomata.

6. **Stroma.**—Many observers describe a stroma for tubercle exactly resembling connective tissue of adenoid tissue, *i.e.* a stroma consisting of delicate fibres, at the point of intersection of which are found nuclei.

N.B.—The presence of this is denied by others (Cornil and Ranvier), and the appearances are said to be due to changes produced by hardening reagents. The importance of this point is that it makes the structure of tubercle identical with that of adenoid tissue, and raises the question whether tubercle may not be a lymphomatous growth.

Finally.—It is now stated, and universally believed,

that the essential element of tubercle is the **bacillus tuberculosis.**

The **bacillus tuberculosis** is a rod-shaped body found in the cells, and particularly in the giant cells and around them, singly or in clusters; it is found chiefly in recent caseating tubercles, but sometimes in old ones.

N.B.—Some authorities have found this bacillus in museum specimens of supra-renal capsules put up for Addison's disease.

The length of the bacillus is about one quarter the diameter of a red blood corpuscle (the diameter of the red blood corpuscle is $\frac{1}{3200}$th of an inch). 1–12800 = inch

This bacillus produces spores in its interior; it can be cultivated in ox-serum at a temperature of 86° to 105·8° F. It will not grow well in parts freely movable, and only grows very slowly.

[Methods of Staining.

To demonstrate the bacilli in sputum the following will be found convenient in practice :—

The sputum is spread in a thin layer on a cover-glass and dried by being passed three or four times through the flame of a spirit lamp. Then a few drops of Dr. Heneage Gibbes's double-stain are poured into a watch-glass and slightly warmed by being held for a few minutes above the flame. The cover-glass is then placed on the stain and allowed to remain for four or five minutes, then washed in methylated spirit until no more colour comes away, dried either in the air or over the spirit lamp, and mounted in Canada balsam.

The stain is made as follows (*vide Lancet*, May 5th, 1883):—

Take of—

Rosaniline hydrochloride . .	2 grammes.
Methyl blue	1 gramme.

Rub them up in a glass mortar.

Then dissolve—

Aniline oil 3 c.c. in rectified spirit .	15 c.c.
Slowly add distilled water	15 c.c.

Keep in a stoppered bottle.

To demonstrate the bacilli in tissues:—

Sections should be cut and stained with the above fluid for three or four hours; then remove them direct into methylated spirit, and there leave until the excess of stain be washed off, when they must be placed in clean spirit, and afterwards in absolute alcohol for five minutes; they are then cleared with oil of cloves and mounted in Canada balsam.—Eds.]

Origin of Giant Cells.

a. By overgrowth of a single cell (Hamilton).
b. By aggregation of numerous cells.
c. By coalescence of leucocytes within capillaries.
d. By buds of protoplasm from the walls of capillaries, which should normally form fresh capillaries.

Forms of Tubercle.

a. Grey miliary tubercle. The previous description applies to this form.
b. Yellow tubercle. Yellow caseous masses of

variable size, softening and breaking down in the centre, and due to fatty degeneration of the grey tubercle.

c. There is another form in which the grey tubercle, instead of softening, becomes of cartilaginous hardness and undergoes a fibroid change; at a later stage it becomes pigmented.

Origin of Tubercle.

1. Is it a new growth ?
2. Is it a product of inflammation ?
3. Is it a product of infection ?
4. Is it due to a specific cause ?

Taking these points separately :—

1. Is it a New Growth ?

a. It has the structure of lymphatic tissue, unless Cornil and Ranvier are correct in denying the presence of stroma.

b. It especially affects lymphoid structures ; *e.g.*, solitary follicles, spleen, Peyer's patches, lymphatic glands. Dr. Klein has shown that lymphatic tissue is met with in the lung at the point at which the bronchioles expand into air-cells. Now this is just the point where tubercle of lung begins in phthisis.

c. It behaves exactly like a malignant lymphoma in its tendency to become generalised ; *i.e.*, a chronic primary tubercular deposit may at any time set up secondary deposits. But, although the above is undoubtedly true, it must be admitted that tubercle occurs independently of lymphoid tissue—*e.g.*, in liver and connective tissue of organs generally.

2. Is it a Product of Inflammation?

First, we must take the state of the lungs, and if we determine the nature of tubercle there, the same will apply throughout the body.

Virchow and Niemeyer say that all tubercle is inflammatory, and that the caseous deposits met with in phthisis are really pneumonic products which have undergone fatty degeneration and have broken down; and, further, if grey miliary tubercle be found in connection with such caseous products, it is secondary to them (*vide* inoculation experiments described later on). In favour of this view we have the facts that—

a. In ordinary phthisis the caseous products consist of degenerated epithelium, and the structure of tubercle given above is not to be made out. It may very well be true that in these cases the tubercle structure is destroyed as rapidly as it is formed; certainly if not found in the lung, it is frequently found in other organs—*e.g.*, intestinal canal, glands, spleen, etc.

b. *Very rarely* we find caseous degeneration of the lung limited to one side.

Against this view we will commence by taking a case of tuberculosis of the lung, and compare the behaviour of phthisis with it. Every one admits that acute miliary tuberculosis is *the type* of tubercular disease, so that if we take this as our type, we can compare the tuberculosis of phthisis with it.

Acute Miliary Tuberculosis has the following characteristics:—

a. It consists in the scattering, more or less

closely, throughout the lung of grey miliary tubercles as described above.

b. Tubercles at the upper part of the lung are always older than those at the lower part, are frequently found yellow and caseous, and softening in the centre.

c. There is a wide production of miliary tubercles throughout the body.

Against the inflammatory theory we have the facts that—

a. Pneumonia is a disease of the base, and phthisis of the apex, and so far resembles miliary tuberculosis.

b. Acute pneumonia does not caseate or break down into cavities.

c. In phthisis if we do not find the tubercular growth in the lung, we shall in some other organ.

But it must be admitted that even in acute miliary tuberculosis there is much pneumonia, and this pneumonia behaves in exactly the same way as the nodules having a typical structure of tubercle—*i.e.*, in caseating, breaking down, and forming cavities; further, secondary nodules having exactly the same structure as those in the alveoli of the lungs have been found in the liver. Therefore it seems fair to describe the pneumonia products as "tubercle" equally with that possessing the typical structure. If we do not do so, we exclude miliary tuberculosis, in which in a large number of cases the products are entirely epithelial, and most cases of phthisis; and yet these cases behave in the following particulars as acute miliary tuberculosis :—

a. In commencing at the apex.
b. In caseating and crepitating.
c. In being accompanied by undoubted miliary tubercles in other organs.

In fact, one may probably say that there is no anatomical structure limited to tubercle. As described by Fagge, "tubercle is due to an irritant of small size and low intensity; if the irritant lodge in the alveolus of a lung, the products are epithelial; if it should lodge in connective tissue, the products consist of granulation tissue; so that there is nothing specific in the anatomical character, but there is in the irritant cause." After the bacillus was discovered, Fagge regarded this as the irritant of low intensity.

3. Is it a Product of Infection

Inoculation Experiments.—Villemin, Cohnheim, and Wilson Fox introduced pieces of caseous matter beneath the skin, or into the peritoneal cavity, of animals. The result was—

a. Nodule at point of inoculation.
b. Secondary nodules, exactly resembling miliary tubercle, in lungs, liver, spleen, and serous membranes.

But they soon found that caseous material was not essential; that pieces of growth and putrid muscle, or even setons, would produce the same effect—in other words that any irritant might set up tuberculosis. From this a theory of tuberculosis was deduced: that the initial stage of phthisis or tubercular ulceration was a common catarrh, that this led to a swelling of the neighbouring glands, that these underwent caseation, and the caseous products set up tuberculosis.

Against this view it is certain—

a. That in the human subject dried-up abscesses and caseous growths will not set up tuberculosis.

b. One observer, the late Wilson Fox, has admitted that he did not take care to isolate the animals (guinea-pigs) experimented upon from those undoubtedly tubercular, and here of course would be a probable source of infection by the bacilli.

If a small nodule of caseous matter be placed in the outer chamber of the eye of a rabbit, it at first disappears, and three weeks after an eruption of miliary tubercles takes place on the iris (Cohnheim).

4. Is it Due to a Specific Cause?

Tubercle Bacillus.—This has been already described, but some further facts are important.

a. It can be cultivated in sterilised ox-serum at a temperature of 86° to 105·8° F.

b. Such cultivations may be injected into animals, and when so injected will set up acute miliary tuberculosis.

[The most recent experiments tend to show that only when tubercle is introduced is tubercle produced.—Eds.]

c. There is probably some tissue proclivity or general predisposition necessary for their growth.

Some such reason as this seems necessary to account for the exemption from contagion; then again, when

tubercle is hereditary in a family, it often does not break out for many years.

The above suggestions will explain this; probably it does no harm until the tissue be in some way injured.

[We are justified in believing that the bacillus tuberculosis is the cause of all tubercular processes. Its presence, at least in the early stages, rather than any especial anatomical structure, must be the essential characteristic of tubercle.—EDS.]

[According to this theory, tuberculosis may be acquired by *inoculation*, *inhalation*, or *swallowing*.

a. **Inoculation.**—Comparatively rare, and usually causes a local lesion only.

b. **Inhalation.**—By far the most common source of infection. The bacilli, or rather their spores, may be found in air-borne dust, especially in rooms occupied by phthisical persons. The daily sputa of a single patient may contain twenty million bacilli, which will retain their virulence for months (Bollinger).

c. **Swallowing.**—The following may be received into the alimentary canal:—

(i.) Milk from a tuberculous cow.

Tuberculosis of the udder, in the form of softening nodules, is not uncommon among milch cows; again, absence of complication of the udder does not ensure safety; there is experimental evidence to prove that bacilli may be present in the milk from tuberculous cows in which the udders are unaffected, and also that tuberculosis may be imparted to young animals by the use of such milk. (N.B.—The high mortality of young children

from tubercular peritonitis, "tabes mesenterica," and tuberculous intestine is very striking.)

(ii.) The flesh of animals which have suffered during life from tuberculosis. The cooking to which meat is subjected is not sufficient to destroy bacilli that may be present in the deeper parts.—EDS.]

Distribution of Tubercle.

The **Distribution of Tubercle** is of some importance, both clinically, and as showing the influence of tissue proclivity.

As a rule, in one individual it takes a certain distribution.

a. The commonest is ordinary phthisis, with ulceration of the larynx and intestines; now, as showing tissue proclivity, acute pneumonia has the same tendency.

b. Serous membrane tubercle :—

Peritoneum. Pleuræ. Pericardium.

Not arachnoid membrane; and, curiously, this is not now known as a serous membrane.

The importance of this form is that it for the most part remains so limited, and is often recoverable from.

c. Genito-urinary tubercle (often primary).

Kidney.	Urethra.
Ureter.	Fallopian tube.
Bladder.	Ovary.
Testis.	Uterus.

d. Tubercular disease of adrenals (Addison's disease), often associated with caries of spine. Solitary yellow tubercle of the brain and tubercle of supra-renals have been found together, and glioma of the same two organs.

e. Tubercle of lymph glands and spleen. The analogy is Hodgkin's disease.

f. Tuberculosis of joints comparable with pyæmia.

All these forms of distribution are of clinical importance, and they all show that different tracts have associated proclivity for disease.

Whatever may be the nature of tubercle (inflammatory or specific), there is no doubt of the following :—

That both yellow and grey tubercles are met with in the same lungs, or that either form may be exclusively met with.

When all the tubercle is of the grey miliary form, we have acute miliary tuberculosis, the lung of which has already been described.

Pulmonary Phthisis.

The lung of pulmonary phthisis varies much in appearance in relation to the duration of the disease; the oldest mischief is almost always at the apex (more often, perhaps, of the left lung) in adults, but in children this localisation is not so definite; here the mischief often creeps in at the root of the lung.

A common occurrence is to find caseous masses of very variable extent at the apex, more or less riddled by cavities; lower down, caseous material not yet broken down; and, still lower, grey miliary tubercle. We may say almost without contradiction that in this case we invariably find the opposite lung diseased; it

may be that there is only a deposit of grey tubercle at the apex, or there may be caseation and cavitation to a variable extent.

According to the chronicity of the disease the tubercular products vary. If the advance of the disease has been rapid, we find the products all yellow and softened, if chronic more or less fibroid change, so that the lung is infiltrated in all directions by fibroid deposit and caseous or calcareous masses, surrounded by zones of *fibroid phthisis*, and no doubt includes most of the cases so called. Here again, the oldest disease is at the apex, and undoubted tuberculosis will be found in other parts of the body (intestine most commonly).

In the rapidly progressive case, tubercle quickly undergoes fatty degeneration, caseates, and breaks down; in the other case the tubercle undergoes a fibroid change.

Vomicæ are of variable size, from the smallest possible up to the size of a whole lobe, and when recent have soft, irregular ragged walls composed of yellow caseous material; sooner or later, if the patient survive, they become quiescent and undergo cure; the walls become smooth, they become surrounded by a zone of fibrous tissue, and internally they are found to be traversed by trabeculæ bands, which are either remnants of the original connective tissue of the lung or obliterated branches of the pulmonary artery.

It is very important to note that these branches are frequently the seat of small aneurisms, which are present in the majority of chronic cases, and which are the almost invariable cause of the fatal hæmoptysis which so frequently cuts off a case of chronic phthisis.

Besides cavities formed by degeneration, some of them are found to be due to dilated tubes; these would

be due to the same causes as those to be discussed under chronic pneumonia.

Chronic Fibroid Phthisis is, no doubt, in the great majority of cases, a true tubercular disease, but the question is raised whether it may not in some cases be the result of an ordinary acute pneumonia.

Now, there is no doubt that acute pneumonia in the vast majority of cases ends acutely, and does not become chronic (Dr. Wilks says *never*). But under the heading of **chronic interstitial pneumonia**, two classes must be described :—

a. (Wilks.) Cases of old pleurisy in which we find the pleura greatly thickened, including that between the lobes. The inter-lobular connective tissue is thickened and the whole lung more or less contracted and destroyed by infiltration of fibroid tissue. This is limited to one lung; there is no tubercle found in these cases.

b. Besides these causes, there are a few undoubted cases in which, after an attack of acute pneumonia, the bases of one or both lungs have been found contracted and destroyed by infiltration of fibrous tissue, and with thickened pleuræ. Now, to these cases the term "chronic interstitial pneumonia" applies, and they constitute a small proportion of the cases of clinical "*fibroid phthisis.*"

Pneumokoniosis.—*Synonyms* : Anthracosis, or miner's phthisis ; mill-grinder's phthisis ; steel-grinder's phthisis (grinders of pens and pointed instruments).

A name given to chronic disease of the lung due to inhalation of various forms of dust.

The pathology of this disease is probably dual.

1. There is a form which gives rise to bronchitis, and, following upon this, a dilatation of the walls of the bronchial tubes, then thickening and finally overgrowth of the connective tissue of the lung; in other words, a *chronic interstitial pneumonia.*

2. But there is no doubt there is a second class of cases in which the phthisis is truly tubercular. In these cases—

a. The disease attacks one lung before the other.
b. It commences at the apex, and breaks down into cavities.
c. It is accompanied by tubercle, either in the lungs or elsewhere.

Miner's Phthisis may be of either form, and is due to the irritation caused by minute fragments of coal-dust. The lungs become intensely black, and the seat of the pigment is in the inter-alveolar connective tissue, the peribronchial and inter-lobular tissues, and, finally, the bronchial glands.

[The mortality of workers in "dust occupations" depends not so much on the source of the dust, whether vegetable or mineral, but upon the character of the particles which compose the dust. The heaviest mortality is met with in those occupations in which the dust is composed of hard, angular, sharp particles, which, becoming impacted in the mucous membrane, are not easily displaced, and eventually set up the changes described above. Dust composed of soft and rounded particles does not cause nearly so much mischief. The comparative mortality of males in certain dust-inhaling occupations from phthisis and diseases of the respiratory organs is shown in the following table,

taken from Dr. Ogle's supplement to the forty-fifth Annual Report of the Registrar-General, in 1885 :—

—	Phthisis.	Diseases of Respiratory Organs.	Phthisis and Diseases of Respiratory Organs.
Coal-miner . . .	126	202	328
Carpenter, Joiner . .	204	133	337
Baker, Confectioner .	212	186	398
Mason, Bricklayer, Builder	252	201	453
Wool Manufacturer . .	257	205	462
Cotton Manufacturer .	272	271	543
Quarryman . . .	308	274	582
Cutler	371	389	760
File-maker . . .	433	350	783
Earthenware Manufacturer . . .	473	645	1,118
Cornish Tin-miner . .	690	458	1,148
All Males (England and Wales)	220	182	402
Fishermen . . .	108	90	198

Dr. Ogle explains the comparative immunity of coal-miners by the microscopical character of the coal-dust, the particles of which are particularly free from sharp angles and points; he is also inclined to attribute to coal-dust a special property hindering the development of tuberculosis.

The Cornish tin-miners come at the bottom of the list; they work in a heated and vitiated atmosphere, as coal-miners do, but the dust they inhale is a sharp, angular, and very irritant stone-dust, instead of the comparatively innocuous coal-dust.—Eds.]

Causation of Phthisis.

1. **Hereditary taint.**
2. **Exposure to cold and wet.**—By Buchanan's investi-

gations as to the condition of soil predisposing to phthisis, it is undoubtedly proved that it is more prevalent where the soil is composed of clay, or where the *surface water* is of necessity in excess. When districts presenting these conditions have been drained, phthisis becomes far less prevalent.

3. **Mal-hygiene**—*i.e.*, overcrowding, bad air, and insufficiency of food. An important point in connection with this is that in Iceland phthisis is unknown, and yet these conditions are fulfilled there to an extreme degree. The fatty nature of the food there is supposed to be the preventing cause (?). But as regards *this* country at least there is no doubt that these conditions operate in the production of phthisis. The much-diminished mortality from phthisis amongst soldiers, since their barracks have been ventilated, is a proof of this.

4. **Noxious trades** (*cf.* grinders' phthisis).

5. **Alcohol** (Dr. Moxon).—Not proven, except that no doubt the chronic drunkard is more liable to disease.

6. **Syphilis.**—The evidence in favour of the causation is not conclusive. Cases of chronic phthisis with much fibroid growth have been found, associated with gummata in the liver, testis, and other organs, which last are of course absolute proof of syphilis; it is supposed that the phthisis has been due to syphilis, and has had its fibroid character stamped upon it by that disease. Nodules like gummata have been described in the lung; but histologically they are of course indistinguishable from tubercle (except when tubercle bacilli can be demonstrated), and further they have always been found in the upper part of the lung. The best proof is that now and then such cases get rapidly well under anti-syphilitic remedies (Dr. Moxon).

7. **Diabetes.**—No doubt ordinary tubercular phthisis may occur in diabetes, as in any other disease. But it is rare to meet with anything like tubercle in this disease. In a series of cases tabulated by Dr. Fagge, seventeen out of forty cases of diabetes died of phthisis. In twelve out of the seventeen no tubercle was found in the body. The probability is that the so-called **diabetic phthisis** is pneumonic and non-tubercular in most cases. The disease is characterised by rounded patches at the apex or base of the lung (very frequently the latter), which are the size of a hazel nut or larger, and in which a cavity rapidly forms containing a central slough. In other words, the disease resembles the boils and carbuncles common in diabetes. Dr. Goodhart's name of **necrotic pneumonia** is probably more applicable than phthisis. Still, however, it must be remembered that true tubercular phthisis and tubercular deposit in the intestines and elsewhere may be met with in diabetes.

[Dr. Purdy in his monograph on diabetes states that in his experience **tubercular phthisis** attacks the majority of patients in whom diabetes has existed beyond two or three years.—Eds.]

Prognosis of Tubercle.

There is not the slightest doubt that tubercle sometimes undergoes cure. Scars and caseous nodules, surrounded by fibrous tissue and calcareous nodules, are constantly being met with at the apices of the lungs in patients who have died from other diseases, these nodules being the result of ancient tubercle.

Bristowe believes that even small miliary tuberculosis undergoes retrogression. He describes the lung in such cases as seamed throughout with minute stellate cica-

trices, and the pulmonary tissue around spongy and coarse from consequent emphysema. This statement may be compared with the fibroid transformation of tubercle described above (*q.v.*).

Tubercle as affecting Organs other than the Lungs.

Intestine.—Tubercle of the intestine occurs as a complication in about half the cases of phthisis pulmonum.

It affects both small and large intestine, one or both, most commonly the cæcum.

NOTE.—Typhoid affects usually *small* intestine.
Dysentery affects usually *large* intestine.

It commences as a deposit of grey tubercle in the solitary glands and Peyer's patches; sometimes, however, it starts in the sub-mucous tissue independently. The grey tubercle speedily becomes yellow and caseous; the mucous membrane covering the tubercle sloughs off, and a little shallow ulcer results; then these ulcers run together and form sinuous ulcers; subsequently, by the continued formation of tubercle and its caseation, large tracts of ulceration are produced. The typical tubercular ulcer of the intestines is generally—

a. Irregular, circular, and running round the transverse axis of the bowel.

b. The edges are thickened and nodular, and not undermined.

c. In the base of the ulcer, on the peritoneal surface, small miliary tubercles may be seen.

These ulcers rarely perforate, and do not often bleed.

The ulceration leads to inflammation of the peritoneal coat; and, further than that, more or less extensive deposits of tubercle, which rapidly become yellow and caseous, take place in the peritoneum and mesentery;

further, the mesenteric glands become yellow and caseous. The result of this is—

a. The coils become adherent to one another.
b. The mesentery, thickened and shortened, binds them down to the vertebral column.
c. The omentum becomes puckered up into a hard, transverse mass, usually about the umbilicus.
d. Ulceration extends between adjacent adherent coils, so that the original lumen is lost.
e. Lastly, there is very little or no ascitic fluid.

This disease is one form of *tubercular peritonitis* (tabes mesenterica). In adults it is secondary to phthisis or some other tubercular disease; in children it may be primary.

Serous Membrane Tubercle.—Serous membrane tubercle affects peritoneum, pleura, and pericardium (not pia mater or arachnoid). It may be limited to the abdomen, and then gives rise to a second form of tubercular peritonitis. The tubercle is of the miliary variety. The serous membranes are found studded with more or less numerous grey tubercles, which, however, also become yellow and caseous.

Of course miliary tuberculosis of serous membranes is frequently found as a complication of other tubercular diseases, such as phthisis, or as part of a general eruption of tubercle (acute tuberculosis); but there is a form which only affects these serous membranes, which is primary, and which is curable.

This form of tuberculosis is accompanied by much fluid effusion, so that serous cavities become distended with clear yellow serum, often with flakes of lymph floating in it.

Tubercular Laryngitis and Tracheitis.—After the intes-

tine, the larynx is probably most often affected. It is very rarely primary, generally occurring at the end of a case of pulmonary phthisis.

There are two forms :—

a. **Miliary Tuberculosis,** in which there is a deposit of grey granulations in the mucous membrane of the larynx, and possibly the other air passages, with much swelling and injection of the air passages.

N.B.—The epiglottis being often affected, there is much distress and dysphagia.

b. In the second form an ulcer starts at the back of the arytaenoid cartilage, and may spread laterally and in depth, and lead to necrosis of the cartilages and their expectoration.

Genito-urinary Tubercle.—Tubercle may affect the kidney in two ways :—

a. As part of a general tuberculosis.
b. As a primary disease.

As part of a general tuberculosis it has no special importance; it is only a part of a grave and fatal disease; but as a primary disease it is of the utmost importance.

Besides the kidney, the whole of the genital and urinary tracts may be involved :—ureters, bladder, urethra, vas deferens, testis, fallopian tubes, uterus. This form of tuberculosis does not of necessity start in the kidneys. It may commence in the testis or the kidney, or both kidney and testis may be affected simultaneously.

The disease commences generally at the apices of the

Malpighian pyramids, or at their bases, between the pyramids and cortex. There is no doubt that at first there is a deposit of grey miliary tubercle; this, however, is rarely seen, for tubercle of the kidney very rapidly caseates.

What is generally seen in an early stage is a deposit of round caseous nodules, of variable size, in the position named; these nodules, of various sizes, by aggregation increase in bulk, soften in the centre, and form vomicæ. But coincidently with this the pelvis of the kidney undergoes tuberculosis; the tubercles become deposited in the mucous membrane, and frequently, perhaps generally, the tubercular disease leads to obliteration of the lumen of the ureters, and, following upon this, we get dilatation of the pelvis and calices, and flattening out of the Malpighian pyramids; and the final result is that we get a large, often very large, organ, with dilated pelvis and calices, and riddled in all directions by cavities containing caseous debris.

It is of special importance to note that tuberculosis of the kidney may be a primary disease.

Spirit specimens of renal tuberculosis cannot be distinguished by the naked eye from growths in the kidney—*e.g.*, sarcoma.

Tubercle of Spleen is probably never primary. It is part of a general tuberculosis.

The spleen is studded with round, yellow nodules, about the size of a millet or hemp seed, and said to be washed away by a stream of water, whereas Malpighian granules are not. [According to Dr. Carrington, it is doubtful if this be true.—Eds.]

Tubercle of Liver.—Here, again, we have to deal with a secondary disease.

Note.—In children with jaundice and fever there is

probably tuberculosis of liver, which may be associated with general tuberculosis. As part of a general acute tuberculosis, the liver may be studded with numberless grey granulations; in a later stage yellow tubercles are met with, the nodules varying in size from a hemp seed to a hazel nut, or larger.

The tubercles seem to be deposited in the course of the bile-duct; they are always bile-stained, and there is generally a central cavity. It used to be said that the cavity represented the lumen of a bile-duct; this, however, appears not to be the case. The cavity results from the softening of the tubercle; at all events, continuity with a bile-duct cannot be established.

Tuberculosis of Brain and Spinal Cord as a primary disease is limited to *young children* (under ten, probably under five years of age), but may occur in adults as a secondary effect of phthisis, or some other form of tuberculosis.

The most common seat is the *cerebellum*, after this the *pons*, medulla, crura cerebri, optic thalamus, corpus striatum, and, besides this, the central hemispheres and the cord.

The growth is very often *solitary*, but there may be several. It varies greatly in size, from the smallest possible to the size of a billiard ball, or larger. It usually presents itself as a more or less rounded mass of firm consistency, and yellow and caseous throughout; there is *very rarely* softening in the centre.

At the periphery will be seen a greyish translucent border. This is the growing edge, for, when examined by the microscope, it will be found to present the typical tubercular structure.

Addison's Disease.—This is said by many to be a tubercular disease of the supra-renal capsules.

Tubercle of the supra-renal capsule may be a part of a general tuberculosis, but this can scarcely be called Addison's disease, for the pigmentation and other symptoms may be entirely absent. Whether Addison's disease is entirely tubercular is open to question.

The earliest appearance is of a greyish deposit in the medulla of the supra-renal capsule. Then the deposit invades the cortex, and the organ becomes enlarged to two or three times its bulk, or even more. Then this deposit undergoes fatty degeneration and caseation. At first yellow caseous nodules of the size of a pea are seen, but ultimately the capsule is converted into a yellow caseous mass.

Upon this a stage of atrophy supervenes, and the capsule becomes small and contracted, and even converted into a little fibrous body without any trace of the original structure.

One capsule may probably be affected before the other; but *post mortem*, in true cases of Addison's disease, both capsules are affected, although the disease in one may be in advance of the other.

Microscopically.—The new growth is found to consist, in the first place, of numerous leucocytes and an indistinct fibrillated stroma; further, the leucocytes undergo fatty degeneration, and break down into a granular debris.

Many reliable observers have found the bacillus tuberculosis in the supra-renals in cases of Addison's disease.

This affection of the capsule is frequently associated with caries of the spine, generally the lower dorsal and upper lumbar vertebræ.

Relation of Addison's Disease to Tuberculosis.—Dr. Moxon investigated those cases of Addison's disease in

the museum of Guy's Hospital in which the lungs had been preserved, and he almost invariably found tubercle at the apices of the lungs.

Dr. Wilks believes the disease to be a special one, and that only the affection above described will give rise to the principal symptoms of the disease, viz.,—

Pigmentation.
Asthenia.
Feeble circulation.
Vomiting.
Little or no emaciation.

But of late other cases have been recorded in which all the symptoms have been present during life, and in which after death the capsules have been found diminished to thin-walled cysts, or just a relic of supra-renal tissue.

The question must to some extent be regarded as undecided.

No doubt the vast majority of cases are accompanied by caseous disease, and often by tubercular disease. Whether Addison's disease may be due to a simple atrophy, or whether atrophy, such that the capsule may be reduced to a mere cyst, is a later result, is undetermined.

Such cases of atrophy have been described by Wickham Legg ("St. Bartholomew's Hospital Reports"), Dr. Barlow ("Pathological Transactions"), Dr. Coupland ("Pathological Transactions"), and Dr. Goodhart ("Pathological Transactions," vol. xxxiii., p. 340).

CHAPTER II.

THE LUNG.

(For *Phthisis vide* Chapter on Tubercle.)

Acute Lobar Pneumonia.

PRESENTS three stages:—

1. Engorgement.
2. Red Hepatisation.
3. Grey Hepatisation.

To which have been added two others:—

a. A stage of simple hyperæmia antecedent to the first.

b. A stage called by Dr. Wilks "purulent infiltration," which, however, is generally described under Grey Hepatisation.

1. **Engorgement.**—The lung is more bulky, it pits on pressure, it is of a deep red colour, and on section abundant blood-stained frothy fluid escapes; and it is more readily broken down than is usually the case.

Microscopically.—The vessels are gorged with blood, and a few cells are found in the pulmonary alveoli, probably derived from their epithelial lining, the cells of which are seen to be swollen and proliferating.

2. **Red Hepatisation.**—Lung is bulky and much heavier than normal; invariably there is some amount of pleurisy, varying in degree from a mere loss of

polish of pleural surface to a thick layer of plastic lymph.

On Section.—The colour is red, mottled with pigment, of very great intensity. (N.B.—This colour is not a P.M. change.) The surface is not smooth, but minutely granular. The lung tissue breaks down with very little pressure. Portions of lung sink in water. The swollen bronchial tubes are full of muco-pus.

Microscopically.—The alveoli are fitted with plugs of fibrin, of which the fibrils can be made out. In the meshes of this fibrin cells of different kinds can be seen, red blood cells, leucocytes, epithelial cells, these latter being found in greater numbers at the periphery. The walls of the alveoli are thickened somewhat by the presence of leucocytes in the connective tissue. Lastly, the blood-vessels are engorged with blood.

As the inflammation progresses the epithelial proliferation becomes a prominent feature. The alveoli in consequence become more and more distended. This presses the blood out of the vessels, the red colour in consequence is lost, and then ensues—

3. **Grey Hepatisation,** in which the lung is bulky, heavy, yellowish grey in colour, mottled with pigment. Pleurisy still present.

On Section.—The colour is yellowish grey mottled with pigment; the surface is granular, the lung breaks down on pressure more readily, and the tubes copiously exude muco-pus.

In most cases the stage of purulent infiltration is included under this head.

In this condition the exudation has undergone "fatty degeneration," and broken down into a purulent fluid; we may meet with every degree of this condition till the lung may be described as a sponge soaked with pus.

Microscopically.

a. The blood has nearly disappeared from the vessels.

b. The fibrinous plugs have broken up into fatty granules.

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c. The red blood cells have also disintegrated, and practically the alveoli are occupied with cells indistinguishable from leucocytes, and epithelial cells more or less on the way to become leucocytes. The cellular elements filling the alveoli are partly derived from blood, partly from epithelial proliferation.

It is important to note that acute pneumonia may clear up without there being a trace of sputum from beginning to end. It is supposed that the products of inflammation, having undergone fatty degeneration, have been absorbed by blood-vessels; and the question is raised whether this may not be the normal process of resolution, and therefore the condition of purulent infiltration necessarily fatal.

Termination of acute pneumonia:

1. Resolution.
2. Abscess.
3. Gangrene.
4. Chronic Pneumonia or Cirrhosis of Lung.

Abscess is excessively rare as a result of lobar pneumonia, and is invariably found in the lobular pneumonia resulting from pyæmia.

Gangrene is also a rare result of acute pneumonia. Perhaps it is most frequently found in the super-acute pneumonia, resulting from immersion; but it must be allowed that it is an occasional result of ordinary acute pneumonia. In fact, it is very doubtful whether pul-

monary gangrene ever arises without an antecedent pneumonic change in the lung.

Taking the question of gangrene generally, it has, in addition to the above, the following causes. It may occur—

1. Around masses of pulmonary tubercle.

2. In cases of thoracic aneurism and mediastinal tumours, which press on the trachea and bronchi.

N.B.—In this case the question arises as to whether the gangrene results from pressure on the vagus and the consequent withdrawal of its trophic influence on the lung, or whether the gangrene is due to accumulation of secretion behind the obstruction and its consequent decomposition. Certainly this last view is probable from the consideration that the commonest cause of pulmonary gangrene is septic poisoning, as from œsophageal cancer perforating into the lung, from embolism in suppuration of internal ear followed by thrombosis of the sinuses of the dura mater, or from round substances capable of decomposition sucked into the larger air passages (*e.g.*, pieces of bone in two cases in "Guy's Reports," vomited matter, etc.).

3. As a result of pulmonary infarcts.

Appearances presented by gangrene of the lung:—

1. There is in most cases a zone of pneumonia, into which the gangrenous part passes insensibly; but sometimes this zone is absent.

2. The gangrenous part copiously exudes a brownish green extremely fœtid fluid.

3. The gangrenous lung tissue is soft, often diffluent, and also fœtid.

Microscopically.—The lung tissue is everywhere broken down, and in the most advanced parts consists of granular detritus.

Gangrene of lung must not be confounded with fœtid bronchitis. In some cases of chronic bronchitis with dilated tubes, the retained secretion undergoes chemical changes, which cause the sputa to give off the odour of gangrene.

The tubes in these cases are occupied by plugs which are called "Dittrich's plugs." They are composed of granular matter, degenerated epithelium, and a large amount of crystals of the fatty acids (butyric acid chiefly). These plugs are yellowish brown in colour, and protrude from the divided ends of the tubes.

[There is a strong tendency at present to class pneumonia as an epidemic disease, and to attribute the incidence of the disease to a specific microbe; the following have been considered at different times to be the specific microbe :—

1. **Friedländer's Micrococcus Pneumoniæ.**—These are large, oval, and capsulated micrococci, and are found in large numbers in the affected lungs, and in the blood and sputa. Pneumonia has been produced in rabbits by the injection of cultivations of these micrococci.

2. **Fränkel and Weichselbaum's Diplococcus.**—This is also capsulated and asserted to be the true pneumonic microbe by its discoverers; it is indistinguishable from one found in large numbers in normal saliva.

3. **Klein's Bacillus Pneumoniæ.**—In a widespread and fatal epidemic of pneumonia, which occurred at Middlesborough in 1888, Klein found in the morbid tissues

large numbers of short bacilli; inoculations of the lung-juice or of cultivations of the bacilli into mice produced an acute disease, the chief and constant feature of which was pneumonia: further inoculations and cultivations from these mice produced the disease in other mice. Samples of bacon purchased in the district gave the same disease to mice when fed on it, and in this bacon were found similar bacilli which could be cultivated and inoculated with precisely the same results as those taken from the human lungs. Whether the bacon had or had not become infected by human cases of pneumonia is not clear, but there is hardly any doubt that the disease was capable of being spread by means of infected food.—EDS.]

Chronic Pneumonia.

It has already been stated that most cases of chronic pneumonia (*i.e.*, chronic fibroid phthisis) are really cases of chronic tuberculosis. But there are some few cases which commence with an attack of acute pneumonia, which never entirely clears up, and in which the patient ultimately dies, his lungs being found in the following condition:—

1. Pleura immensely thickened, and this thickening extending to the pleura between the lobes.

2. Lung greatly reduced in size, firm, dense and fibroid, greyish in colour and mottled with pigment, and more or less airless.

3. The other lung is healthy.

4. There is no tubercle in the body.

This is the condition described (by Dr. Wilks) as "cirrhosis of lung." The view given by Dr. Wilks is that the primary disease was pleurisy, causing the

thickening of this membrane, and that from the thick pleura the fibroid tissue invaded the lung in all directions, first passing into the lobe and then between the lobules, and ultimately between the alveoli. It appears, however, certain that there are a few cases without such thickening of pleura, and therefore it is probably true that pneumonia in some few instances gives rise to a chronic disease.

Syphilis of Lung.

Depaul described a yellowish grey nodule in the lung of children dead of congenital syphilis, and Virchow described what was called "pneumonia alba." The lung is yellowish grey, solid, and firm, but retains the impress of the fingers. The difficulty in making sure that there is a true syphilitic phthisis arises from the fact that the histological structure of tubercle and gumma is identical (cf. page 21), and that gummata caseate in exactly the same way as tubercle. Of course ordinary tubercular phthisis may occur in a syphilitic individual.

The evidence is as follows :—

1. Nodules surrounded by a fibroid zone yellow, opaque, and caseous, are associated with gummata of the liver, testis, and other parts.

2. Disease of the apex of the lung, consisting of caseous nodules indistinguishable from yellow tubercle, but surrounded by very great excess of fibrous tissue, is found associated with gummata of other organs.

3. Syphilitic individuals sometimes contract chronic disease of the lungs, commencing at the apex, and which ultimately is indistinguishable from phthisis, but rapidly gets well under anti-syphilitic remedies (Dr. Moxon).

Goodhart and Greenfield describe the fibroid material of what they believe to be syphilitic phthisis, as distinguished by its greater vascularity.

Broncho-pneumonia.

Is secondary to bronchitis, and occurs in the extremes of life, in children and old people.

Naked Eye Appearances as we see them in a child's lung :—

a. The lobules, or groups of them, are distended with inflammatory products, so that on the surface they project and render the surface uneven.

b. The bronchioles and bronchial tubes are full of pus and mucus.

c. Scattered between the pneumonic lobules are collapsed portions. These are of a slate blue colour, pyramidal in shape, airless so that they sink in water, and are incapable of inflation. These lobules are depressed below the surrounding lobules, and make the irregularity of the surface on section more marked.

d. The lung tissue surrounding the collapsed or pneumonic parts is in a state of hyper-distension. It is not emphysematous, because the alveolar septa are in no case broken down.

Microscopically. — Broncho-pneumonia is distinguished by the far greater amount of cellular epithelial products in the affected alveoli. These cells are derived from—

1. Proliferation of epithelium of the bronchial tubes being sucked into the alveoli.

2. The proliferated epithelium of the alveolar walls.

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There may be a small amount of fibrin exuded, and perhaps a few red cells; these are in extremely small proportion, and only found immediately around the bronchioles.

3. Large numbers of leucocytes derived from emigrated white blood cells. The bronchioles have their walls thickened in consequence of infiltration of leucocytes.

Acute Bronchitis.

Naked Eye Appearances.

1. Injection of mucous membranes.
2. Tubes filled with muco-pus.

3 (often). Hyper-distension of alveoli. Owing to the accumulated secretion, and perhaps because the large tubes have their walls softened, these tubes are unable to collapse and allow the air to escape. There is no destruction of alveoli, etc., no true emphysema.

Microscopically.

1. There is great vascularity.
2. The columnar ciliated epithelium is discharged, and its place is taken by ill-formed epithelial cells and leucocytes.
3. Swelling of basement membrane.
4. Infiltration of leucocytes into different layers of the tubes and even outside them.

Chronic Bronchitis.

Naked Eye Appearances.—These are by no means marked. The vascularity has disappeared, and the mucous membrane is of a slate colour, often mottled with dark patches. Bronchiectasis and emphysema are invariably present to a greater or less degree.

Microscopically.—The changes are identical with what

has been described under acute bronchitis, but in addition the walls of the tubes show atrophic changes.

Many of the muscular fibres are atrophied.

The cartilages also undergo atrophy.

Emphysema.

Two kinds:—

1. Interstitial.
2. Vesicular.

Interstitial Emphysema is characterised by the fact that the air is extravasated into the connective tissue of the lung, so that the lobules are surrounded by bead-like rows of air bubbles.

Sometimes in extreme cases the air passes inwards to the root and reaches the connective tissue of the mediastinum or even the neck.

This form only occurs in a case in which the connective tissue is abundant—*i.e.*, in children—and it is most commonly found in pertussis.

Vesicular Emphysema.

Sir William Jenner described two forms: large-lunged or hypertrophous emphysema; small-lunged or atrophous emphysema.

Atrophied or **Atrophous Emphysema** is synonymous with senile atrophy of the lungs, which will be treated of afterwards.

Large-lunged Emphysema (of adults).

Naked Eye Appearances.

1. Lungs are excessively bulky, and retain their size when removed from the body; *i.e.*, they will not collapse.

2. They pit on pressure and do not crepitate. They are described as feeling like a down pillow. The

absence of collapse and the pitting on pressure are due to loss of elastic tissue.

3. The parts which are affected first are the apices and anterior border, but the process in extreme cases extends throughout.

Microscopically.

a. The first change is hyper-distension of the infundibula and subsequently of the alveoli. Partly by atrophy of the alveolar walls, partly by distension, the air cells disappear, and the whole infundibulum is converted into a pyriform sac. The hyper-distension continues, and contiguous sacs break into one another so as to form greatly enlarged cavities and bullæ, which are seen when the disease is extreme. The bullæ often project from the surface beneath the pleura.

N.B.—On account of the hyper-distension of these enlarged air sacs, emphysematous lung when incised collapses completely. In consequence of the distension of the alveolar walls it follows that there is a corresponding destruction of the capillaries, and this is to a large extent the cause of the cyanosis and dropsy of chronic bronchitis.

Theories of Emphysema.

1. **Theory of Laennec.**—He supposed that some of the tubes, as the result of bronchitis, became blocked with secretion; that in consequence of this the air could not be driven out of the lungs, but that it could be drawn into the cells by forced inspiratory efforts; that in coughing, the air, retained in some cells by the

plug of secretion being subjected to pressure, ruptured the air cells.

2. **Gairdner of Glasgow.**—He showed that the effect of the accumulation of mucus in the tubes would be collapse, not hyper-distension. The tube being conical and gradually diminishing in size, inspiratory efforts would only have the effect of fitting the plug of secretion more tightly. Expiratory efforts would drive it into a wider part and allow it to escape. The final result would be that portions of lung would be emptied of air. Even if the air cells were distended, any amount of compressing force would not rupture. The air cells might be regarded as spheres, and any amount of pressure equal in all directions would not rupture such a sphere. He therefore supposes the sequence to be as follows:—

I. Bronchitis.
II. Collapse.
III. Hyper-distension of other parts of the lung by forced inspiration, consequent atrophy of alveolar walls, and emphysema.

This hyper-distension is due to the fact that the healthy part of the lung expands to make up for the part collapsed.

3. Sir William Jenner advocates the expiratory theory, but on different grounds from Laennec. He points out that the pressure at all parts of the chest is not equal, that the apices are less supported than any part on account of the mobility of the sternum and costal cartilages; the adjacent part of the lung is supported lung.

A fissured sternum shows that in coughing these parts of the lung are projected forwards; that is,

parts of the lungs are rendered functionless by collapse or accumulated secretion; other parts will become hyper-distended to compensate for these; and during coughing those parts of the lungs least protected will become emphysematous. These are the parts, apices, and anterior borders, which are affected as a matter of common experience.

Besides the inspiratory or expiratory theories of emphysema, there is another which refers to a general degeneration of the alveolar wall throughout the lung. Fatty granules in such cases are found in the walls of the alveoli. But it is objected that this fact does not prove that the primary process is the fatty degeneration. This may be called the "nutritive theory," and it leads to what is the worst form of emphysema, viz., a general destruction throughout the lung.

To sum up, the probability is that all these causes of emphysema are true in particular cases.

a. That the emphysema which is found surrounding tubercular nodules, or which is secondary to a chronic pleurisy, is really inspiratory.

b. That the emphysema associated with chronic bronchitis is produced, as Sir William Jenner explains it, by expiration.

c. That nutritive changes are sometimes primary, and no doubt are often secondary. The air cells are in a state of hyper-distension; the nutrition of the lung suffers partly from this and partly from the condition of the body in general; and so the two last causes are in operation at the same time. With regard to the ultimate cause of emphysema, the following facts seem established:—

1. It is hereditary.

2. It may be produced by acute pneumonia weakening the pulmonary tissue; evidence of this is a case quoted by Hertza.

3. It may be produced by overexertion. This is undoubtedly the case in draught horses, in which the apices and anterior parts of the lung become emphysematous. In man there are undoubted cases in which severe physical exertion has caused emphysema.

4. The use of wind instruments, shouting, and the like are causes of emphysema. A military cornet-player was attacked by double pneumonia; previous to that he had been playing his instrument as usual, but after his recovery he was unable to play. He died of some intercurrent disease shortly after, and both lungs were found to be emphysematous.

Atrophous Emphysema.

(Senile atrophy of lungs.)

This condition is commonly met with in old people with narrow, shrunken chests, stooping shoulders, and, in fact, the usual accompaniments of old age. The lung tissue is atrophied throughout. The lungs collapse remarkably on opening the chest. The change is simple atrophy of the alveolar walls, leading to emphysema.

[Malignant Disease of Lungs.

Malignant disease of the lungs may occur as—

1. **Primary in the lungs** (rare).
2. **Secondary to—**
   - *a.* Adjacent parts (*e.g.*, infiltration into the root of the lung from the mediastinum).
   - *b.* Malignant disease of other parts (*e.g.*, testis).

Varieties.

1. Soft carcinoma (encephaloid), most common.
2. Various kinds of sarcomata.
3. Scirrhus, epithelioma, and colloid; these are all very rare.

It may occur in persons of all ages, but the middle periods of life are the most liable, almost equally in the two sexes.

When *primary*, it usually constitutes a solitary mass in the substance of the lung.

When *secondary to surrounding parts*, it runs along the bronchial tubes, infiltrating the connective tissue surrounding them, and finally involving the walls of the tubes and the surrounding lung tissue.

When *secondary to disease in distant organs*, multiple nodules are found of various sizes in the substance of the lung and beneath the pleura.

Malignant growths of the lung, like those of other organs, tend rapidly to undergo degenerative changes, and thus to break down and form vomicæ.—Eds.]

CHAPTER III.

THE HEART.

Heart Disease.

ENDOCARDITIS is of three forms:—

Acute.
Chronic.
Ulcerative.

CAUSATION.

Acute Endocarditis.

1. Rheumatism in about half the cases.
2. Chorea.
3. Pyæmia.
4. It is universally stated that some of the specific fevers are causes, *e.g.*, scarlatina, diphtheria. It is doubtful if scarlatina ever causes it apart from the rheumatism which so frequently accompanies it; that it may be caused by diphtheria is still more doubtful.

Chronic Endocarditis.

1. Is a sequel of acute, so that the causes are the same, except that we must exclude pyæmia on account of its fatality.
2. May be chronic from the first, and then its causes will be—

Rheumatism.
Strain.
Chronic Bright's disease.

Ulcerative Endocarditis.

1. In the large majority of cases is secondary to chronic endocarditis.

2. It may be idiopathic (unless it is shown that in all cases it is due to an organism called "mycosis endocardii," a micrococcus).

3. Pyæmia, especially puerperal pyæmia. It is a fact of clinical importance that puerperal endocarditis affects the valves of the right side, especially the tricuspid.

4. There is the view that it is due to the growth of the specific organism mentioned above.

Morbid Anatomy.

Acute Endocarditis.

Naked Eye Appearances.

1. A row of small bead-like granulations along the contact edges of the valves, towards the blood stream. This is on the auricular surface of the mitral and the ventricular of the aortic.

2. These granulations may remain single, or may run together into warty masses of variable size. Upon these fibrin is deposited, at first as a little cup, but ultimately it may be in long branching clots.

3. These masses of granulations or clots, waving to and fro in the blood stream, come in contact with other parts of the endocardium, and set up acute endocarditis at their points of contact, so that it is said that acute endocarditis is a disease of the valves; and if other parts of the lining membrane are affected, this is always secondary (Moxon).

Sometimes the inflamed valve undergoes softening, and the result is an ulcer, and this may go on to the

formation of an aneurism of the valve, or even perforation. This perforation is rapidly closed by granulations or clotting; and so the valve may be capable of exercising its function, and after death it may be overlooked. This softening may involve the chordæ tendineæ and lead to their rupture, and thus cause a very severe form of heart disease.

Microscopically.—The vegetations present throughout numerous nuclei derived partly from proliferation of connective tissue and partly from extravasated leucocytes. At the apices of the granulations irregular masses of protoplasm are found, which are the altered endothelial cells. The infiltration of leucocytes extends downwards into the substance of the valve.

Ulcerative Endocarditis.

In ulcerative endocarditis (or malignant endocarditis) groups of micrococci are found in the vegetations. These have been designated "mycosis endocardii." The ulcerative process is believed to be due to the presence of these organisms. Further than that, the shedding of these organisms into the blood stream, together with portions of the affected valves, is believed to be the cause of the pyæmic symptoms which characterise the disease. Dr. Wilks calls it "arterial pyæmia."

N.B.—Ulcerative endocarditis in the vast majority of cases is a complication of chronic endocarditis. Now and then it seems to arise without previous disease, especially in puerperal cases, and it is to be noted that in these cases it attacks the valves of the *right side*.

Chronic Endocarditis.

The extravasated nuclei become converted into fibrous tissue, and this undergoes the usual changes of contraction, producing deformity of the valves to a greater or less degree.

Naked Eye Changes in Aortic Valves.

They become more or less deformed on account of cicatricial contraction, so that—

1. They may be little functionless masses projecting like shelves.

2. A cusp may be torn away at one side and hang down.

3. Eversion of the edges of the cusps may take place (not a dangerous result). The valves being previously diseased, fail to meet at their free edges and to support one another, so that when the regurgitant stream bears on the edges it everts them towards the ventricle.

Perhaps the most important form of aortic disease is one not due at all to the valves, but to primary disease of the aorta. This occurs in young hard-working men, and also there is good reason to suppose it to be the result of syphilis.

The first part of the aorta undergoes softening, so that under exertion it becomes dilated. The valves themselves, being formed of white fibrous tissue, will not stretch, but the aorta between them yields, partly from its natural elasticity and partly from its softened condition. The result is that the valves are separated from each other at their attachment perhaps a quarter of an inch. Also, on account of tension, they become con-

verted into deep, narrow pockets. Eversion of valve may take place and complicate the result.

In a third form of disease the valves become adherent at their edges. The septa between the original valves may disappear; and a sort of funnel-shaped orifice results. Sometimes only one septum disappears, and an aortic valve with only two cusps is the result. All intermediate changes are met with.

Changes in a Cuspid Valve.

1. The cusps of the valve become thickened and contracted.

2. The chordæ tendineæ become thickened, often adherent to one another (so as to form large, thick bundles), and shortened, so as to draw down and fix the thickened cusps.

3. The musculi papillares undergo fibroid changes at the apex and increase the contraction.

4. The cusps become adherent at their edges, and so—

5. The orifice becomes contracted. The result is stenosis ("button-hole mitral").

This is the commonest form of mitral disease pathologically, although mitral regurgitation is the most common clinically. The explanation is this: that the stenosed valve will permit regurgitation as well; and the clinical symptoms of the latter will altogether overshadow those of stenosis. Sometimes, however, undoubted regurgitation is met with, the orifice being held rigidly open and also enlarged; and sometimes as a result of ulcerative endocarditis the chordæ become divided, so that the cusp flaps backward and forward and is quite functionless.

Results of Morbid Changes—Mitral Stenosis.—Left auricle becomes dilated and hypertrophied.

Right auricle and ventricle follow.

Left ventricle remains small.

N.B.—In a few cases of mitral stenosis the left ventricle is hypertrophied, probably from coincident regurgitation.

Mitral Regurgitation.—Same as above, with the addition of hypertrophy and dilatation of left ventricle.

The question suggests itself as to why the left ventricle is hypertrophied in mitral regurgitation.

1. It cannot be that first there is increased pressure in the veins and that this is ultimately transmitted through the capillaries to the arteries and the left ventricle, for the same condition obtains in mitral stenosis.

2. Niemeyer suggests that if the ventricle did not thicken it would not be able to do its extra work, and the heart must stop.

3. Possibly the hypertrophy is due to the increased pressure in the left side of the heart. In regurgitation blood is driven back into the left auricle. This causes hypertrophy of the walls of the left cavity, and this in contracting drives the blood back again into the ventricle, so that on account of this increased work both cavities thicken.

Aortic Disease.—Stenosis and then regurgitation.

N.B.—Practically these two conditions go together except in a very few cases. Aortic disease is very quickly followed by regurgitation.

Aortic stenosis leads first to hypertrophy of left ven-

tricle. There must be a limit to this, and when this is reached the muscular fibres undergo fatty degeneration. Then dilatation ensues. The original mischief increases; and regurgitation follows the dilatation and hypertrophy. Ultimately the right cavity becomes affected, and so the result is the immense *bovine heart* of aortic disease. The largest specimen is that of Dr. Peacock—forty-eight ounces.

N.B.—Immense hearts with hypertrophy and dilatation of all cavities are met with in chronic Bright's disease. In these cases the valves are healthy or only a little diseased by thickening or atheroma.

The effect of left-sided disease, as already seen, ultimately results in hypertrophy and dilatation of the right cavity; but hypertrophy and dilatation of the right heart *alone* is almost invariably due to chronic bronchitis and emphysema or chronic fibroid phthisis.

Primary Pulmonary or Tricuspid Disease is excessively rare.

In these cases—

a. The heart is described as square.

b. The right ventricle takes part in the formation of the apex, equally with, if not more than, the left.

Effects of Heart Disease on Other Organs.

I. Embolic effects.
II. Backward effects.
III. Forward effects.

I. Embolic Effects.

The effects of an embolus depend entirely on the process going on in the site from which it was derived.

Simple embolism blocks arteries.

Ulcerative embolism sets up ulceration.

Gangrenous embolism sets up gangrene.

N.B.—An embolus invariably becomes impacted at the bifurcation of the artery.

Brain.—Simple embolism, very rarely a wedge-shaped infarct, such as will be described hereafter, is seen. Generally a form of softening is the result.

In brain, kidney, spleen, and lung the arteries do not anastomose. Now, in these viscera, if an artery be blocked by an embolus, the effect is not anæmia of the embolic area, but hyperæmia and hæmorrhage.

This is explained by Wilks and Moxon as follows:—"When an artery is blocked, not only is the blood cut off from the area or tissue which it supplies, but also from the artery itself. The result of this will be malnutrition of the arterial walls beyond the embolus and passive dilatation. This creates negative pressure, and the blood rushes back from the veins into the capillaries, and with such force as to rupture the weakened arteries and cause hæmorrhage."

In the brain the usual effect is **red softening**. The tissue becomes soft and diffluent, red in colour from hæmorrhage, and the nerve tissue is spoilt, so that only a granular debris, with more or less broken-up nerve-fibres and cells, is found, with many **compound granule cells**. These are large cells crowded with granules. They are the only pathognomonic evidence of softening of nerve tissue.

The arteries usually blocked in the brain are the **left middle cerebral** and the **basilar.** Dr. Fagge does not confirm the usual assertion about the left middle cerebral. Twenty cases of right and twenty-one of left were collected by him from Guy's P.M. Reports.

In red softening, as described, the extravasated blood ultimately undergoes decolorisation, and the result is a yellow ochrey colour. That is one variety of **yellow softening.**

Lung.—The effect is pulmonary apoplexy, variable-sized, wedge-shaped extravasations of blood, with the base to the surface, and the artery leading to such patches blocked by embolism. This is always a late result of heart disease, and the infarct does not go through the changes of decolorisation, atrophy, and so on, as in the case of the kidney and spleen.

N.B.—The above refers to simple embolus. Ulcerative or gangrenous embolus would set up local ulceration or gangrene.

Liver and Intestine.—Embolism of the hepatic artery on account of the frequent anastomoses seldom leads to infarcts. Embolism of a mesenteric artery leads to acute enteritis, probably because the blood from the veins runs back into the embolised area, and leads to hyperæmia.

Spleen and Kidney.—As in the lungs, there are two important facts in the vascular supply :—

a. The arteries do not anastomose.
b. The veins have not any valves.

When an artery is blocked by a simple embolism the first effect is of course a momentary anæmia. Of this

we have no *post-mortem* evidences; but, for the reason already given, the blood rushes back from the veins through the capillaries into the embolic areas with such force as to cause hæmorrhage. The result is a wedge-shaped patch, with its base to the surface of the organ, of a dark purple colour like a recent extravasation. The subsequent series of changes consists in alteration and absorption of the blood colouring matter, so that it becomes at first a yellowish brown, and then there is atrophy and contraction of the tissue in the embolised area, and ultimately a fibrous scar. Caseation also takes place in the wedge-shaped infarctions, and may begin certainly within thirty-six hours.

The above might be applied to pulmonary infarcts. But this condition in the lung occurs at the end of heart disease, and the change rarely, if ever, goes beyond the state of hæmorrhage. The converse is also true: given an infarct, the artery supplying its areas will be found to be blocked.

The above applies to simple embolism, but if the embolus be derived from a source of suppuration or gangrene the result will still be a wedge-shaped patch, which frequently sloughs or becomes gangrenous *en masse*.

Embolism of Peripheral Artery.—Simple Embolism. In this case the veins contain valves, and the arteries anastomose; the blood therefore cannot return from the veins. If the anastomosis in the particular part in which the embolus is impacted be not free enough, the result is dry gangrene. Gangrene in a young person is almost invariably due to heart disease. If the embolus be suppurative or gangrenous, it sets up the corresponding process in the artery in which it becomes impacted, and the result is hæmorrhage or aneurism.

If a cerebral artery, hæmorrhage.

If a peripheral artery, false aneurism, consisting of extravasated blood, of which the sac is formed by the surrounding tissue.

But the process need not go on as far as actual hæmorrhage, but may cause inflammation of the arterial wall, and consequent softening and yielding of the wall, with formation of a true aneurism.

Cerebral hæmorrhage in a young person is frequently, but not invariably, the result of heart disease.

II. Backward Effects.

Lung.—"Brown induration." The organs are firm and dense. On section they are dark brown in colour, more or less airless, and frequently there is coincident bronchitis. The tissue is very dense, scarcely breaking down even under extreme pressure.

Microscopically.—The first change is dilatation of the pulmonary capillaries, which become enlarged and varicose. As a result of this the air-cells are encroached upon, and the lung becomes comparatively airless.

Secondly, on account of the extreme pressure, the colouring matter of the blood exudes and undergoes changes producing the brown colour.

Lastly, on account of the continued hyperæmia, the connective tissue of the lung undergoes proliferation, and this process surrounding the walls of the alveoli renders them still more airless. The result is a lung dark brown, extremely hard and dense, and more or less airless.

Liver.—Nutmeg liver. The liver is enlarged, extremely hard, and on section has the nutmeg pattern. The process is—

a. Firstly, dilatation of the hepatic vein.

b. Secondly, the pressure of the dilated veins causes atrophy of the immediately surrounding liver cells, which become white and granular.

c. Lastly, the peripheral cells pick out fat from the portal veins and become yellow, so that a lobule of the nutmeg liver is normally described as showing three zones:—

i. *Red* in centre.
ii. *White* surrounding.
iii. *Yellow* at periphery.

With these changes there is an increase of the connective tissue of the portal canal, with a slight increase round the hepatic vein; indeed, it is stated by numerous authorities that heart disease is a cause of *cirrhosis of the liver.* It is to a modified degree; but that it ever leads to the atrophic and hypertrophic cirrhosed liver is certainly not true. The heart disease is invariably fatal before such an extreme process can come about.

Kidney.—Here, again, as in the case of liver, it is a moot point whether heart disease leads to granular kidney (cirrhosis of kidney or chronic interstitial nephritis). The probability is that it does not, the patient dying long before such an effect can take place. In heart disease the kidneys are extremely hard and elastic, deep red in colour, and very frequently secrete albumen. No doubt cases of interstitial nephritis with a dilated and hypertrophied heart are often met with; but these kidneys are in all probability examples of primary renal and secondary heart disease.

The heart in such a case will be found more or less enlarged, sometimes enormously so; all the cavities

are dilated, and the walls thickened; the valves are thickened and atheromatous, and the orifices dilated, but there is no evidence of endocarditis—no granulations are found, and there is no puckering or contraction. In fact, the appearance of the heart is that of an organ overworked, all the parts of which have become hypertrophied.

N.B.—Such a condition is almost invariably met with in adult life. In fact, all cardiac disease beginning in adults is generally due to renal disease.

Systemic Venous Circulation.—The result is at first *cyanosis*, seen first at the extremities (ears, nose, fingers, toes). In chronic heart disease the ends of the fingers become "clubbed." This is often the case in phthisis, but there is this distinction: in phthisis the nails also become incurved, in consequence of the atrophy of the fat beneath the nail. The excessive size of the ends of the fingers is due to the chronic hyperæmia.

N.B.—The most extreme cyanosis is not met with in heart disease even when it is congenital, although in the latter condition it is often very severe; but it is found in chronic bronchitis and emphysema.

The next result of heart disease is **Dropsy**. **Cardiac Dropsy** begins in the feet and extends upwards. The genitals, if they are affected at all, are so secondarily. There is almost invariably a tinge of **Jaundice**.

Renal Dropsy begins in the eyelids and genitals, and the patient is markedly white.

Spleen.—The spleen in heart disease not complicated with ulcerative endocarditis is small, hard, of a dark purple colour. Considered mechanically, it should be enlarged, but it is true of heart disease, as of about half the cases of cirrhosis of the liver, that the spleen is small, showing that the condition is not a mechanical one.

N.B.—It may be laid down as a rule that if the spleen in heart disease is enlarged, we have to deal with ulcerative endocarditis. In other words, it is the large soft spleen of pyæmia. There may be exceptions to this rule, but they are very few.

III. Forward Effects.

Forward effects of heart disease will be manifested in the pulse.

a. **Mitral Stenosis.**—Nothing noteworthy till regurgitation is superadded. The pulse may be small.

b. **Mitral Regurgitation.**—Pulse small, feeble, irregular or intermittent, or both.

c. **Aortic Stenosis.**—Pulse hard, slow (forty per minute).

d. **Aortic Regurgitation.**—Low tension pulse, which strikes the finger suddenly and suddenly recedes. It is best observed by grasping the forearm with the hand, and not by feeling the pulse in the ordinary way. Water-hammer sensation increased by raising the arm.

Subjective Effects of Heart Disease.

1. **Pain**, increased by exertion, starting in cardiac region and shooting down the left and occasionally the right arm, and sometimes accompanied by numbness or anæmia of the affected part.

2. **Palpitation.**—Pain and palpitation are, however, more frequently the result of functional than of organic affections.

3. **Dyspnœa** is caused by exertion.

Orthopnœa, whilst sitting, is probably due to the fact that in the sitting position the patient takes the weight of the abdominal viscera off the diaphragm, so that the lungs and heart act to better advantage.

4. **Sleeplessness.**—The patient dozes off, but wakes up with a start and a feeling of impending suffocation. In this condition full doses of morphia should be given.

N.B.—The preceding notes all refer to heart disease due to endocarditis. The other forms of heart disease of clinical importance are—

1. Hypertrophy and dilatation of heart following on pericarditis.

2. The two forms of fatty heart.

3. Heart disease which follows Bright's disease (*vide* chapter on the Kidney).

Pericarditis.

In its earliest stage it is best seen as the result of Bright's disease, and is always fatal.

1. There is hyperæmia and minute injection of vessels.

2. There is exudation of plastic lymph containing

fibrin factors. This is deposited on the surface of the pericardium, and on account of the movements of the heart becomes deposited in various ways :—

a. Ridged like the sea-shore.
b. As if two surfaces, covered by a sticky substance, had been pulled asunder.

As a rule the amount of fluid is small.

3. Adhesions form, and if the adhesions are of any degree of thickness, we have a fertile source of heart disease acting as follows :—

a. Pericarditis is almost invariably accompanied by myocarditis, and the inflamed muscle becomes more or less paralysed. This condition will lead to dilatation. Myocarditis presents no marked features, P.M. But the layer of fat which is met with beneath a thickened and adherent pericardium is taken as evidence of it.

b. The thick and adherent membrane interferes seriously with the action of the heart, and prevents the cavities emptying themselves. Therefore dilatation results from the myocarditis and the impeded action. As the dilatation increases the heart hypertrophies to compensate for it. These two processes go on side by side, till in time the mitral or tricuspid orifice enlarges. The cusps then fail to meet, and the result is mitral or tricuspid regurgitation.

We find, P.M., in a pericarditic heart the following :—

a. A very large heart.
b. Thick adherent pericardium.

c. All cavities dilated and hypertrophied.

d. Valves not punctured or contracted, but probably thickened from overwork.

e. Kidney healthy, or only congested. This last point is important, in order to distinguish such hearts from those of Bright's disease. A further distinction is that the pericardial heart occurs in young subjects, the heart of Bright's disease in those of middle age. It is important to note that the pericardium, even when adherent, is liable to further attacks of inflammation, the evidence of which is that the layers of inflammatory products are of different dates, varying from tough fibrous tissue to soft unorganised lymph. Sometimes a considerable quantity of serous fluid or pus will be found in the meshes of the old adhesions.

Fatty Heart.

This form of cardiac disease is not followed by the usual results except in very rare cases ; *i.e.*, there is no dropsy and no enlargement of liver.

There are two forms of fatty heart :—

a. Fatty infiltration.

b. Fatty degeneration.

a. **Fatty Infiltration** is generally most marked in the left auricle. It consists of an overgrowth of fat, which passes into the muscular fasciculi, and causes atrophy or absorption. Often the atrophy is preceded by true fatty degeneration of the muscular fibres. In an extreme case the entire ventricular wall seems to be made up of fat.

Microscopically.—A considerable quantity of muscular fibre will be found.

Such a condition of heart is generally met with associated with obesity, but sometimes without it. Alcohol is perhaps the most frequent cause, but alcohol is also a cause of obesity.

b. **Fatty Degeneration.**—In this case there is an actual deposit of fatty matter in the muscular fibres, at first in the granular, but later in the globular form ; so that in an extreme case the sarcolemma is full of oil globules, the muscular structure having entirely disappeared. It appears that there is no (or only very slight) absolute increase of fat, the analysis of a fatty degenerated heart being almost the same as a healthy heart.

Causes.

1. Obesity.
2. Wasting diseases: phthisis, cancer, long-continued suppuration.
3. Poisoning, especially by phosphorus.
4. Specific fevers (actual cause is high temperature).
5. Anæmia from any cause, but especially idiopathic.

One of the most important forms is local anæmia, the result of disease of the coronary arteries, for this often leads to rupture of the heart. The earliest appearance of fatty degeneration is found in the musculi pectinati of the mitral columns. These will be found transversely striated in a zigzag manner, with alternate layers of fatty deposit and normal muscle, called the "*Tabby Cat*" striation.

Fibroid Disease of the Heart.

Patches of fibroid tissue are met with occasionally in the substance of the wall of the heart, generally the left ventricle or the septum.

Sometimes in such a condition the cavity is found dilated. But this condition usually ends in sudden death. The question is raised as to whether such patches are not the result of gummata.

[Dr. Fagge gives the following as possible additional causes :—

1. It may result from a primary process of chronic inflammation, *a myocarditis*, arising in the cardiac muscular tissue spontaneously or from rheumatism, or perhaps in consequence of a blow or fall on the chest.

2. The formation of *thrombi* in the cavities of the heart may give rise to an inflammatory change in the wall of the part of the heart to which they adhere ; the ultimate result of this may be the formation of a fibroid patch.

3. Interference with the blood supply of the cardiac wall due to embolism, thrombosis, or atheroma of a branch of the coronary artery.

It is probable, however, that syphilis is by far the most usual cause.—Eds.]

Congenital Heart Disease.

Is believed to arise from intra-uterine disease, not from arrest of development.

1. The most common condition is as follows :—

   a. Before the septum of the ventricle is perfected, endocarditis of the pulmonary valve takes place, and causes various degrees of obliteration of the pulmonary artery.

   b. The blood being prevented from flowing through the pulmonary artery, will pass from the right ventricle to the left over the top of the incomplete septum, and will prevent its closure.

c. The *foramen ovale* may or may not be prevented from closure from the same causes.

d. Frequently the *ductus arteriosus* also remains patent.

2. Sometimes there is a common ventricle from which both aorta and pulmonary artery arise.

3. Sometimes the *foramen ovale* remains patent, sometimes the *ductus arteriosus.*

4. Sometimes there is only one auricle and one ventricle.

5. Sometimes there are two auricles and one ventricle.

Hypertrophy and Dilatation.

Hypertrophy of the heart is always secondary to some disease or to overwork. Probably the only instance of simple hypertrophy without dilatation is met with in Bright's disease (*q.v.*); but even in this case we rarely meet with a greatly thickened left ventricle without any dilatation.

Dilatation and Hypertrophy in other cases are secondary to—

a. Valvular disease.

b. Adherent pericardium.

c. Bright's disease.

d. Fibroid disease of heart.

CHAPTER IV.

THE LIVER.

Murchison's Classification.

Enlargements.

Painless.—Lardaceous. Fatty. Hydatid. Simple hypertrophy. Leukæmia.

Painful.—Cancer. Pyæmic abscess. Tropical abscess. Hypertrophic cirrhosis. Congestion. Catarrh of ducts. Tuberculosis. Tumours. Cysts.

Contractions.—Simple atrophy. Acute yellow atrophy. Atrophic cirrhosis.

Lardaceous Disease.

General Pathology—Causes.

i. Suppuration, using the term in its widest sense, the commonest cause being chronic bone disease and phthisis.

ii. Syphilis apart from suppuration.

Other alleged causes are chronic ague and malignant disease.

Tests.

1. Lardaceous viscera stain a deep mahogany brown with solution of iodine in potassium iodide.

2. For microscopical demonstration methylaniline violet is best to use. The sections are first stained, and then washed with dilute acetic acid. The result is that the lardaceous matter shows a rose pink colour.

3. Lardaceous material is said to turn blue with iodine and sulphuric acid. This probably never occurs unless cholesterine be present.

Pathology.—The two points in dispute are—

1. Is the lardaceous material an exudation from the blood ?

2. Is it a degeneration ?

In composition it is nitrogenous, and closely resembles that of fibrin. The term "amyloid" was originated on the theory that the substance was *starchy* on account of the reaction obtained with iodine and sulphuric acid.

The evidence of its being a degeneration rests upon histological grounds. If an early specimen be examined which contains a small artery, and be stained with aniline violet, individual muscular fibres will be found to be affected, and these will be seen to be surrounded by healthy cells. This of course is difficult to explain on the supposition of exudation. Dr. Dickinson is the author and advocate of the exudation theory. This theory is based on the following facts :—

1. That lardaceous material is very soluble in alkali.

2. That after treating with alkali it no longer stains with iodine.

3. That if after treatment with alkali it be treated with acid it will again stain with iodine.

4. That chemical analysis shows lardaceous organs to be largely deficient in potash.

5. That if fibrin be treated with dilute acids it will take the iodine reaction as well and as deeply as lardaceous matter.

Therefore he suggests that lardaceous matter is deposited from the blood because from deficiency of potash it has become insoluble. The cause of this deficiency in suppuration is the loss of corpuscles,

and these elements of the blood contain a large proportion of potassium.

The question of the influence of syphilis is more difficult to deal with.

But Dickinson suggests that deficient formation of corpuscles is as bad as excessive loss.

Syphilis produces anæmia (*i.e.*, a deficiency of corpuscles), and this may act in a similar way, the salts of potassium not being in sufficient amount to keep the fibrin factors soluble.

A view has been strongly advocated (Goodhart) that there is some connection between lardaceous disease and fever.

It is pointed out that in febrile conditions the coats of the arteries assume a translucent appearance, comparable with that of early lardaceous disease.

Order in which the viscera are affected.

1. Kidney.
2. Liver.
3. Spleen.
4. Small intestines.
5. Stomach.
6. Large intestines.
7. Adrenals.

Murchison, however, puts liver first.

Scheme for describing condition of liver.

1. Amount of enlargement or contraction.
2. Uniform or non-uniform.
3. Surface.
4. Consistence.
5. Section to the naked eye.
6. Microscopical appearance.

Morbid Anatomy of Lardaceous Liver.

Synonym.—Waxy, amyloid (depurative, Dickinson).

The increase in weight is often very great, a hundred and eighty ounces being a common weight.

Enlargement uniform, except where lardaceous disease supervenes on a liver affected by syphilitic scarring. This may be mistaken for cancer.

Surface is quite smooth.

Consistence is doughy and inelastic; instead of displacing organs in its growth, it moulds itself around them, often receiving the impress of the ribs. It takes and retains the impress of the fingers.

Section.—Its appearance will vary according to the amount of fat (which is almost always found associated with lardaceous disease).

Generally the parts thus differently affected remain separate. The lardaceous part is semi-translucent and of a pinkish grey colour.

The fatty part is represented by lines of opaque yellowish grey material.

The section is quite dry, or at the most a reddish blood-stained fluid escapes from the vessels.

Sometimes the deposit of fat is more uniformly diffused, and then the colour will become a yellowish grey.

Finally, lardaceous material undergoes occasionally calcareous degeneration, so that deposits of earthy matter may be met with.

Lardaceous disease is usually universally diffused throughout the organ, or may in rare cases be partial.

Microscopically.—The first change appears in the arterioles of the hepatic artery and the capillaries.

These become semi-translucent, blurred, and indistinct.

N.B.—The inter- and intra-lobular veins remain unaffected.

The question is raised as to whether the hepatic cells undergo lardaceous change or whether they are simply destroyed by the exudation of lardaceous material around.

This is left an open question by Dr. Goodhart, who says (Sydenham Society) that if the change does take place, the cells become enlarged and glassy, the nuclei disappear, and the outlines become blurred. Then the altered cells become aggregated together into large irregular masses.

The final result is that a section of liver presents a homogeneous, shiny, structureless appearance in the midst of a delicate reticulum of connective tissue. The cells in the neighbourhood of the portal veins take up fat. The cells surrounding the hepatic vein either remain unchanged or undergo atrophy. The zones of the portal vein, the hepatic artery, and the hepatic vein are generally followed, but by no means closely.

Hydatid.

Is caused by the growth and development of some form of tænia. In man two forms are met with :—

i. The cysticercus cellulosæ, which is the hydatid form of tænia solium.

ii. Echinococcus or common hydatid.

The cysticercus cellulosæ is usually met with very numerously and widely distributed, especially in the

muscles. It is rare. The common hydatid as a rule occupies one of the larger viscera, in the vast majority of cases the liver. It may remain barren, but it usually multiplies either by an exogenous or endogenous process.

Description of Tæniadæ.

1. Tænia solium.
2. Tænia mediocanellata.
3. Bothriocephalus latus.

1. **Tænia solium** is found in the ileum.

It is three to four yards long.

Head globular, four suckers, a rostellum of twenty-six hooklets.

Joints or proglottides are longer than broad.

The genital aperture is on the side, a little beyond the middle.

The uterus consists of a central linear cavity with from seven to ten lateral branches.

2. **Tænia mediocanellata.**

Head, four pigmented suckers, no hooklets.

Proglottides are very irregular, but as a rule broader than long. They are larger than tænia solium.

Genital aperture is in the middle or behind middle.

Uterus branches dichotomously.

3. **Bothriocephalus latus.**

Head presents two lateral grooves.

Proglottides much broader than long.

Genital aperture in the centre of the flat surface.

Uterus is rosette-shaped.

Life History of Tæniadæ.

All these creatures need two hosts : one a carnivor for the tape-worm ; one a herbivor for the hydatid.

There are a few exceptions, the most important being the tænia solium, both forms of which occasionally infest a man.

The ova of the tape-worm are discharged from the alimentary canal of their host in vast numbers. Then in some way, probably by means of some vegetable, they are swallowed by a herbivorous animal. This being received into the stomach, the chitinous capsule of the ovum is destroyed, and what is known as the *six-hooked embryo* is set free, which at once proceeds to burrow its way through the walls of the stomach. Some will pass into a blood-vessel, and will be conveyed to distant parts of the blood-stream, the most common seat being the *liver*. Others simply make their way by burrowing through the tissues. As soon as the embryo reaches its nidus, it fixes itself. The six hooklets disappear, and the embryo develops *into a cyst*.

From the interior of this cyst a *head with rostellum* and suckers is developed.

This is *the hydatid*. The development may go no further, and in this case we have a "barren cyst," or, as it is called, "acephalous cyst." But in most cases both endogenous and exogenous reproduction takes place—*i.e.*, the cyst becomes filled with secondary cysts, and others are produced and thrown off externally. The endogenous growth takes place by means of so-called "*brood capsules*." These are saccular protrusions from the inner wall of the primary hydatid. From the

inner wall of this other protrusions take place, which develop a head with rostellum and suckers. These grow and produce cysts in the interior of the primary cyst. The exogenous production takes place by so-called *daughter cysts.* These are cysts formed between the layers of the cuticle, the brood capsules being the product of the parenchyma. Cysts are thus produced which at first project from the surface and then are extruded. These cysts may produce brood capsules or daughter cysts, and so the multiplication of the hydatid goes on indefinitely.

The hydatid is devoured by a carnivorous animal, and then the head of the hydatid fixes itself to some part of the alimentary tract. It then consists of the head and a little cyst. Then it loses the cyst and begins to bud from the distal extremity, and in this way the proglottides are produced. These develop sexual organs (ovary and testis); ova are produced, discharged from the alimentary canal; some find their way into the stomach of a herbivorous animal, and the same circle goes on.

The hydatid form of the tape-worm of cat is found in man.

Tænia solium has its hydatid form in pig (measly pork), tænia mediocanellata in oxen.

Tænia solium in the case of man is an exception, for in rare instances man infects himself; and there is no doubt that tænia solium and cysticercus cellulosæ are now and then found in the same individual.

Tænia echinococcus hominis of the dog is important as the source of the hydatid of man, a quarter of an inch long; head with rostellum, four suckers, four joints. The last sexual ova swallowed produce common hydatid in man.

Following terms are in common use :—

Strobila = Whole tænia.
Proglottides = The individual joints.
Rostellum = The circle of hooklets.

Hydatid of Liver.

This is the most common seat of the parasite ; perhaps the next is the connective tissue of the pelvis ; then the peritoneum, and then the kidney ; the brain is very rarely attacked. The enlargement is variable in amount, from a tumour the size of a hazel-nut to one filling the abdomen.

It is not uniform. It is commonly situated in the right lobe (frequently in the upper and back part). It may grow in any direction, but generally downwards. Its surface is smooth, and there is not usually any peritonitis over the tumour.

If near enough to the surface there may be fluctuation, and the *hydatid* fremitus may be obtained. (If three fingers are laid over the tumour, and the middle one struck sharply, a trembling sensation is said to be experienced in the other two.) N.B.—There is not the least doubt that this physical sign is common to all cysts tensely full of fluid.

There may be more than one hydatid in the same liver.

Hydatid disease may terminate as follows :—

1. Spontaneous cure is rare. It may arise—

a. From bile getting into a cyst, from ulceration of a duct. N.B.—The more common result of this occurrence is suppuration of the cyst, and often in consequence multiple pyæmic abscesses in the liver.

b. From calcification of the primary cyst.

c. From the secondary hydatids within growing out of all proportion to the primary cyst.

2. It may open into the stomach or intestine. Rupture into the intestine is most favourable, and even into the stomach is generally favourable.

3. It may rupture into the pleura, and set up a rapidly fatal pleurisy.

4. Sometimes adhesions form between the lung and pleura, and the hydatid opens into the lung, and is sometimes expectorated. Hæmoptysis is a frequent result, and the disease generally ends fatally by destructive pneumonia or gangrene.

5. It may rupture into the pericardium, and this is rapidly fatal.

6. Rupture into the peritoneum. The usual result is acute peritonitis; but (and this probably applies to the pleura as well) it seems that the fluid itself will not set up peritonitis. It probably only arises when secondary hydatids are extravasated. Hydatid fluid absorbed by peritoneum frequently sets up urticaria.

7. Rupture externally. This is also unfavourable from suppuration of the cyst and the consequences, or from hæmorrhage.

8. Rarely it may open into the urinary passage.

9. Rupture into portal vein, the result being pylephlebitis, also into vena cava, the result being hydatid in the heart.

Hydatid may prove fatal in other ways: by general marasmus and asthenia; by suppuration of cyst, with or without the formation of pyæmic abscesses; by exogenous production of cysts which may press upon other organs—*e.g.*, one pressing on the spinal cord.

Fatty Liver.

Fatty liver occurs under the following conditions :—

1. General obesity.

2. Wasting disease, especially phthisis; but it may be found in any chronic bedridden subject.

3. Chronic alcoholism.

The explanation that is offered is that in obesity too much fat is produced and stored up in the liver; and in the other case (phthisis) the respiratory function is at so low an ebb that the fat is not destroyed, but circulates in the blood, and consequently is taken up by the liver cells.

N.B.—The hepatic cells have an especial affinity for fat, and inasmuch as it is brought to them by the portal vein, the early stage of fatty infiltration is seen in the outer zone of cells. This is "peripheral fatty infiltration."

In the extreme stage all the cells are filled with fat, and the liver is uniformly of a pale bright colour. In two conditions, pregnancy and lactation, only the zone of cells surrounding the intra-lobular vein is fatty; this is called "central fatty infiltration."

The explanation is that in these conditions there is a large demand for fat.

The liver is caught at the vanishing point of fat (Goodhart). The fat being absorbed from the central cells, the granules are passed on from the peripheral, and none is supplied to make up the deficiency.

Chronic alcoholism probably acts in the same way. Alcohol is readily absorbed and oxidised. It is understood that chronic alcoholism produces fat not only in the liver, but in other organs.

Liver Cells.—The change in the liver cells is described under two headings: "fatty infiltration" and "fatty degeneration." In the former, fat globules are simply deposited in the liver cells; in the other, the nucleus and protoplasm of the liver cells become granular and finally destroyed.

Fatty Infiltration occurs in wasting disease, obesity, and in chronic alcoholism. The cells in this case simply take up fat granules, and these may run together; the final result being that the liver cells are simply full of fat. The nucleus becomes hidden, and a section of liver looks like one of adipose tissue.

Fatty Degeneration occurs in acute yellow atrophy, in phosphorus poisoning, in anæmia, in some of the cells in cirrhosis of the liver, and in acute alcoholism.

Naked Eye Appearances of fatty liver: Liver moderately and uniformly enlarged; pale buff colour; anterior border rounded off; consistence soft and flabby; *on section* either uniformly pale buff without lobulation, or if the change is partial the outer parts of the lobule will be yellow, the inner normal, in colour. It will be distinguished from the nutmeg liver by being very readily lacerable.

Hypertrophy of the Liver.

Is met with in diabetes, in leukæmia, in Hodgkin's disease, and in cases in which part of the liver has been destroyed by syphilitic disease or abscess; *e.g.*, the right lobe destroyed by specific disease weighed two to three ounces, the left forty-six ounces.

Finally, as a physiological condition, some individuals have a larger liver than others, although the body weight is normal.

Here the question is raised as to whether the liver cells themselves increase in size. Cohnheim and Ranvier say that in diabetes they do, and that they contain more glycogen. Rindfleisch thinks that perhaps they do. Other pathologists, having in view the variable size of the liver cells normally, leave it an open question.

N.B.—When the enlargement is uniform it is not of any extent, neither is it of any clinical importance.

Cirrhosis of Liver.

Causation.

Alcohol.

Syphilis (acquired, congenital); characters are widely different, as will be seen later on.

Malarial poisoning.

Obstruction to bile duct by gall stones or cancer.

Thrombosis of portal vein in rare cases.

In hydatids and dried-up abscesses, a partial cirrhosis may be met with.

The following forms of cirrhosis of the liver have been met with :—

1. The large smooth cirrhotic liver; hypertrophic cirrhosis, or biliary cirrhosis.

2. The small hobnailed liver; atrophic cirrhosis, or portal cirrhosis, multilobular

3. Cirrhosis of acquired syphilis.

4. Congenital syphilis, very rare.

5. The cirrhotic liver of chronic ague, very rarely met with in England (probably never). This form is Rokitansky's red atrophy of liver.

The organ is small and contracted, dense and hard

and of purple grey colour. Here the chief change microscopically is destruction of the terminal branches of the portal vein within the liver, so that they end in club-shaped extremities.

[It is more than probable, from the researches of Crookes and others, that some of the exanthemata initiate proliferative changes in the liver which may ultimately end in cirrhosis.—EDS.]

Cirrhosis of acquired syphilis.—The main feature of this form of cirrhosis is that it is irregularly scarred by varying degrees of fibrous tissue; or gummata are met with generally on the surface, but sometimes in the substance, and these are surrounded by capsules of fibrous tissue; or as a later process bands of fibrous tissue start from them. The extreme result is a liver cut up in all directions by cicatricial tissue forming a number of abnormal lobes.

Congenital Syphilitic Liver is rare. Liver is uniformly enlarged, dense, hard, and whitish grey in colour.

N.B.—Congenital syphilis has an important bearing upon the cirrhotic livers met with in young children, which are either large and smooth, or small and hobnailed.

In a few extremely rare cases, portal thrombosis has apparently undergone cure, and from the portal vein little nodules of fibrous tissue have started.

N.B.—Practically the first three forms of cirrhosis are the important ones.

Heart diseases and other causes of chronic pulmonary obstruction lead, no doubt, to the nutmeg liver, which will be described presently. But whether they ever result in such an amount of fibrous growth as may be

termed cirrhosis is extremely doubtful. The probability is that the heart disease kills the patient long before any cirrhotic changes to any degree can take place.

1. The large smooth cirrhotic liver is much increased in size. (Some of the largest livers are cirrhotic.) Surface is smooth; *section* pale yellow in colour; lobulations ill marked, extremely dense and hard.

2. The small cirrhotic liver is irregular on the surface, but somewhat minutely so. It is studded all over and uniformly with small hemispherical projections. Hence the name "hobnailed."

3. The liver of acquired syphilis has been described.

4. In children we either meet with the small hobnailed or the large smooth cirrhotic.

Microscopical Appearances.—There are two changes of which we have to take note:—

1. The growth in the liver of granulations culminating in fibrous tissue.

2. The appearance of double rows of cells in the fibrous tissue. The nature of these is variously interpreted, but is described by Charcot and others as being new biliary ducts.

As regards the first point—*i.e.*, the fibrous tissue—cirrhosis is said to be—

a. Multilobular = irregular groups of lobules are surrounded by fibrous tissue.

b. Monolobular = each lobule is surrounded by fibrous tissue.

c. Intercellular = new-formed elements are found between the cells.

Multilobular Cirrhosis is the small hobnailed liver; *i.e.*, groups of lobules are surrounded with connective tissue.

Monolobular Cirrhosis is the large smooth cirrhotic liver. The reservation stated below must, however, be remembered.

Intercellular Cirrhosis is the liver of congenital syphilis, and probably some cases of adult syphilis; but there is no doubt that the large smooth cirrhotic liver frequently, if not uniformly, exhibits intercellular changes.

Relation of the large cirrhotic liver to the small.—The common statement is that the liver is first large and then contracts down to the small. There is no evidence of this, either clinically or pathologically. Bright is reported to have observed a case, but the report shows that this was by no means definitely proved. A man died at Guy's with a hobnailed liver. He had been in the hospital ten years before, and the report shows that even then he had a small liver.

More cases of fatal cirrhosis have large livers than small; so that at all events the large liver is a very fatal condition.

The inference is that it is not proved that the large liver becomes small and contracted. It has been suggested that the character of the alcoholic drink determines whether the liver will be large or small—that beer-drinkers get the large liver, and spirit-drinkers the small. This is probably generally true, but there is not the least doubt that spirit-drinkers sometimes die with the large liver (but they may drink both, with beer as the beginning).

Charcot's statement is that the **large cirrhotic liver** is independent of alcohol, and is due to the growth of connective tissue about the biliary ducts, so that he calls it **biliary cirrhosis.**

Wickham Legge has shown that ligature of the bile

ducts will produce a certain amount of proliferation of the connective tissue in the portal canals. In men obstruction by gall stones is said to be followed by the same effect; that it ever leads to cirrhosis, that can be recognised as such during life, is denied by some (Goodhart), but all (with the exception of Goodhart, who says he has never seen it do so) admit that obstruction of the duct by cancer will produce a large smooth cirrhotic liver.

But the chief feature of Charcot's description is the so-called "new duct formation," that is to say the presence in the newly formed connective tissue of certain double rows of nuclei. These are described by Charcot as occurring only in the large liver. He supposes them to be a vicarious but imperfect formation of liver tissue, and hence the name "hypertrophic cirrhosis." But now he must without doubt allow that these "duct formations" are met with in many other conditions—*e.g.*, acute yellow atrophy, tubercular liver, cancer of the liver, and even small cirrhotic liver—so that their significance as characteristic of the large cirrhotic liver is lost.

In the normal liver the capillary ducts surround the lobule, and the biliary capillaries are found in the form of channels between the liver cells. The capillaries have no definite wall, but appear to be marked out by rows of nuclei.

So much to make clear the various alleged modes of origin of these "duct formations."

1. That they are *not* ducts; that they are produced by the new-formed connective tissue contracting down upon the rows of liver cells, and causing their atrophy. If two rows of liver cells happen to be included a duct formation is the result (Hamilton).

2. That they arise by a process of budding from the biliary ducts (Charcot).

3. That they are original bile capillaries rendered unduly prominent by the atrophy of the liver cells (Goodhart).

Origin of the connective tissue.—There is no doubt that some of it, if not the greater proportion, arises by proliferation of the connective tissue of the portal canals (Glisson's capsule). But the view is strongly advocated by Hamilton that connective tissue is produced by conversion of the liver cells themselves. No doubt in sections of the cirrhotic liver, cells may be found which appear to be lengthening into fibres, but it does not appear that this process is a very general one.

In cirrhosis the cells become—

1. Fatty.
2. Granular, atrophied, or full of—
3. Bile pigment.

And there is not the least doubt but that in the large liver the tendency is for them to become fatty, and no doubt much of, if not all, the increased bulk is due to this cause.

Theories of the relation of large to small cirrhotic liver.

1. The large is an early stage of the small. There is no direct proof of this (*vide* above). The large is often fatal.

2. The large is due to biliary obstruction, and is independent of alcohol, the small to portal obstruction, and is produced by alcohol. There is undoubted proof that the large liver may be due to alcohol (beer).

3. The large liver presents new ducts in the newly

formed connective tissue. This is true, but they are also found, though to a less degree, in the small liver as well as in other conditions. One very noteworthy difference is the great excess of fat deposited in the cells of the large liver, and much of the bulk is due to this.

Nutmeg Liver.

Brown induration.

Causation.—Any circumstance interfering with the outlet of blood from the hepatic vein: chronic heart disease, especially mitral; chronic lung disease, especially chronic bronchitis and emphysema; other rare causes are thrombosis in the hepatic vein, cancer growing into hepatic veins, and perihepatitis.

Character.—The liver is large, dense, and hard; smooth in the early conditions, but it becomes irregular in chronic cases; not uniformly and minutely irregular, but large projecting tracts alternating with depressed portions.

The Capsule is thickened at various points and sometimes beset with minute translucent granulations which are quite indistinguishable from tubercle.

On Section.—It presents a blending of purple parts and a brick-red pattern, and looks curiously like the pattern of a nutmeg.

In the centre of each lobule is the intralobular vein, much dilated; this is *red.*

Next a *whitish grey* zone of atrophied liver cells, possibly caused by the presence of the large vein.

Outside a zone *yellow* from fatty cells. The outer zone of cells pick up the fat brought by the portal vein; so that a nutmeg liver is a compound of red, grey, and yellow portions. Sometimes these three portions are

not well marked; red and yellow generally predominate. In extremely chronic cases atrophy and shrivelling of the liver takes place, especially in the tract of hepatic vein. This is the **racemose atrophy** of Wilks and Moxon.

Microscopically.

a. Dilatation of intralobular vein.
b. Dilatation of intralobular capillaries.
c. Atrophy of liver cells, which become full of fine granules.
d. Fatty infiltration of peripheral cells.
e. Some describe an excess of leucocytes around the hepatic vein. This does occur, but never to an extent that might be called cirrhosis.

Further the connective tissue of the portal vein does undergo proliferation; but here again the change is never sufficiently extensive to constitute cirrhosis. The fact is, the primary disease (usually cardiac) generally takes the patient off before such extensive changes are reached.

Syphilitic Liver.

Congenital and Acquired.

Congenital Liver is enlarged and heavy, anæmic, grey in colour, or mottled by a mixture of anæmic grey and fatty parts.

Microscopically.—The liver shows a diffused cellular growth throughout the organ, in the portal canal between the rows of cells and between the cells themselves. In another form rounded collections of small cells are met with throughout the liver, and are called

miliary gummata. These are, however, associated with the diffused cellular growth. Gummata and scars like those of the acquired form are rare in congenital syphilis.

Cirrhosis in Children.—Extremely cirrhotic livers are sometimes met with. The livers may be large and smooth or contracted and hobnailed. The microscopical appearances are those of the similar conditions in the adult. Many of these cases are of alcoholic origin. But in a few it seems as if this mode of origin is impossible. These have been supposed to be late results of congenital syphilis.

Syphilitic liver of acquired disease.—Liver is moderately enlarged, more or less cut up by cicatrices into large unnatural lobes often containing gummata. These are rounded or oval bodies, yellow, firm, and elastic, sometimes softened in the centre, and surrounded by a zone of connective tissue. (Cancer has not this latter area.)

Microscopically.—The centre is amorphous and granular, the result of fatty degeneration; the next zone is fibroid tissue; the periphery is a zone of highly vascular, well-organised fibrous tissue. The yellow gummatous material becomes absorbed, and the fibrous zone sends out cicatricial bands through the liver. Gummata nearly always occur on the surface. Besides the gummatous growth there may be a diffused growth of connective tissue throughout the portal canal, and even within the lobules.

N.B.—It seems, however, to be true that patches of grey cicatricial tissue may start in the liver as the result of syphilis, without being preceded by the ordinary yellow gumma.

Tumours of Liver.

Adenoma.—Cellular, tubular.
Carcinoma.—Primary or secondary.
Sarcoma.

The relation of adenoma of the liver to primary carcinoma is important.

Two forms of adenoma are met with: the cellular and the tubular. In either case the following varieties of the disease occur:—

a. Solitary nodules.
b. Multiple nodules.
c. Nodules associated with secondary growths in other organs.

It may be said that the third form is practically indistinguishable from cancer. Its relation to the other two hangs upon its identity of structure.

The Cellular Form.—The nodule of the cellular form has a fibrous capsule, enclosing cells which have the normal arrangement of liver cells, but are often larger and fatty.

The Tubular Form.—The nodule of this form is composed of tubes, which are lined by cubical epithelium, and convoluted. Whether these two forms when unaccompanied by secondary growths may be regarded as adenomata, and not cancer, is almost a question of terms. The only point in favour of the former view is that they are composed of structure resembling the normal tissue, in one case the cells, in the other case the ducts.

Carcinoma of Liver.

Primary or Secondary.

Secondary Carcinomata may arise from any primary source, and the liver is the most frequent seat of secondary cancer. Its structure will be as varied as that of the primary growth that gives rise to it.

Primary Cancer of liver is rare, and occurs in two forms:—

a. One in which the liver is greatly and uniformly enlarged.

b. In the nodular form.

The relation of the latter to adenoma is a difficult question.

The structure of the growth will be a fibrous stroma forming alveoli containing groups of cells of epithelial character, and often presenting all the grades and showing the formation from linear cells.

To draw a line between this and cellular adenoma must be in many cases impossible.

Besides this, it is noteworthy that cancer of the liver is frequently associated with much cellular growth in the portal canal and between the groups of cells, so that the distinction between it and cirrhosis is not always evident.

In fact, here and in many other instances we have all gradations between simple inflammation on the one hand and carcinoma on the other.

Acute Yellow Atrophy.

Practically little is known as to its causation, although pregnancy is frequently assigned as its cause. Sometimes it is epidemic. Alcohol and syphilis are alleged

causes. The probability is that they have only been coincident.

But this fact is important: that an illness that has been going on for weeks as a simple or catarrhal jaundice may in a few days end as acute yellow atrophy.

It is doubtless true that in some cases there is an initial condition in which the liver is *enlarged.* But whether enlarged at first or not, it very rapidly (two to three days) undergoes diminution of its size, so as to lose one-half to one-third of its weight. Sixteen to twenty ounces have been met with; the smallest on record is thirteen ounces. The liver becomes soft and withered, and does not retain its shape; the capsule is thrown into wrinkles.

On Section.—Two parts are more or less blended:—

a. Yellow part: acute.
b. Red part: more chronic.

The general colour is a bright yellow. Lobulation gone. The gall bladder and ducts are either empty or full of colourless or slightly bile-stained mucus.

N.B.—The contents of the intestine are clay-coloured usually, but often black. It is thought by many that in these cases the colour is due to hæmorrhage and altered blood.

Microscopically.—The Yellow liver cells are profoundly altered; not many, in fact, are visible; and those that are are highly granular.

Rounded granular masses of the size of liver cells, but containing ten to twelve nuclei, are common. These are looked upon as proliferated liver cells, or, what

is more likely, as aggregations of atrophied cells. Numerous free granules and oil globules are met with, and all these elements are held together in the interstices of a stroma consisting of vessels, ducts, and connective tissue of the liver itself. In this stroma are numerous "duct formations," probably the normal ducts unduly prominent, through atrophy of the liver cells. But, as in cirrhosis, they have been described as arising—

a. By buds from biliary capillaries.
b. From the hepatic cells.

The Red Part differs only in the effect of chronicity. The hepatic cells have entirely disappeared; nothing being left but a granular detritus, composed of fat globules, pigment granules, and numerous free granules. The stroma, intercellular and portal canals, are considerably thickened by numerous leucocytes and more fully formed connective tissue. Similar fatty changes are met with in the kidney.

The liver of phosphorus poisoning differs in being large, firm, and uniform on section.

Microscopically.—The cells are infiltrated to a great extent with fat. There is no degeneration, as there is in acute atrophy.

[Abscess of Liver.

May be **multiple** or **single.**

Multiple abscesses of the liver are usually small, but sometimes, by the union of several, large irregular cavities are formed.

They are most frequently due to **pyæmia**, sometimes to some inflammatory lesion of the portal system; in these cases they are caused by emboli carried up to the

liver by the portal circulation. Inflammation of the bile ducts (caused by impaction of gall stones, etc.) also sometimes gives rise to abscesses in the liver.

The Single or Tropical Abscess is generally supposed to be associated with dysentery. But there is no doubt that cases sometimes occur when no lesion in the intestines, or any other cause, can be discovered. Of fifteen cases in Guy's, in three no cause could be detected, and of these one had been in the tropics. Local injury and cold have also been assigned as possible causes.

The size of a single abscess may become very large.

The Abscess Walls at first consist of liver tissue congested, softened, and ragged. Afterwards they become converted into a tough smooth capsule.

The Contents at first resemble healthy pus, but afterwards become blood-stained, or mixed with bile, or more or less fœtid and decomposed.—Eds.]

For enlargement of the liver due to heart disease cf. page 55.

For tubercle of liver cf. page 26.

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CHAPTER V.

THE KIDNEY

Scheme for Description of Condition of Kidney.

1. Size of kidney.
2. Shape.
3. Colour.
4. Capsule.
5. Section (cortical, medullary).
6. Microscopical appearance.

Bright's Disease.

The forms of kidney we shall describe under Bright's Disease are—

1. Acute nephritis, or "large red kidney."
2. Chronic epithelial nephritis, or "large white kidney."
3. Chronic interstitial nephritis, or "granular," "cirrhosed," or "gouty" kidney.
4. Small white kidney (a mixture of 2 and 3).
5. Cystic kidney.
6. Lardaceous kidney.

Besides these forms of Bright's disease, the following forms of renal disease are important :—

1. Acute suppurative nephritis, or "surgical kidney."

2. Hydronephrosis and pyonephrosis.
3. Tubercular disease.
4. Malignant disease.
5. Calculus.

Acute Nephritis.

The two most important causes are **cold** and **scarlet fever.** It is met with as a complication of other specific fevers, of which **diphtheria** is the most common. Probably acute alcoholism may be a cause, and in rare cases secondary syphilis.

1. **Size and Weight.**—Greatly increased, sometimes twice the normal size.

2. **Shape.**—Normal.

3. **Colour.**—Blood-red.

4. **Capsule.**—Strips easily, leaving a smooth surface.

5. **Section.**—Highly vascular, blood dripping away, so that the organ is sometimes described as the "blood-dripping kidney."

The cortex is greatly swollen, and many tubes are full of hyaline casts or blood casts, or there are extravasated blood-cells outside tubes; this condition is the earliest met with. After a time the blood-red colour disappears, and this is replaced by a yellowish white. The blood, however, collects in three places: Malpighian bodies, Malpighian pyramids, and stellate veins; so that these parts come out deeply red in the midst of the yellowish white inflamed portion. The reason is that the epithelial proliferation distends the tubes, and squeezes out the blood from the vessels in their walls, and causes it to collect in the three regions mentioned.

6. **Microscopically.**—A distinction is drawn between scarlatinal and other forms of acute nephritis. Scarlatinal is called "glomerular nephritis." In this form

the first change takes place in the Malpighian bodies, and consists in the proliferation of the epithelial cells covering the capillary tufts. Following on this, the connective tissue surrounding the capsule undergoes proliferation, and this is said to be especially marked around the interlobular arteries. The nuclei are said ultimately to develop into connective tissue, and so lead to cirrhosis. In addition to these changes, the epithelium of the convoluted tubes undergoes inflammatory changes. The first change is "cloudy swelling." The protoplasm becomes granular and indistinct. Next, proliferation of epithelial cells takes place, and the newly formed cells, instead of assuming the ordinary cubical shape, become ill-developed, and present all stages down to the leucocyte. The protoplasm of the cells becomes first granular, but ultimately contains fat globules. Non-scarlatinal acute nephritis differs from the above in that the tubal changes alone are present.

N.B.—The above is the orthodox description (Klein), but there is not the least doubt that glomerular changes occur in non-scarlatinal nephritis.

Large White Kidney.

Chronic epithelial nephritis—"branny," "mottled," "fatty" kidney.

1. **Size and Weight.**—Greatly increased (thirteen to nineteen ounces).

2. **Shape.**—Normal.

3. **Colour.**—More or less uniform, opaque, yellowish white. Sometimes the stellate veins on the surface are full of blood, and constitute a conspicuous feature. When this occurs the kidney is called "**mottled.**"

4. **Capsule.**—Strips easily, leaving a smooth surface.

5. **Section.**—The cortex is found to be greatly swollen. The normal pattern is more or less lost, and the cortex is blurred. It also presents frequently alternating stripes of yellow and grey, the yellow parts being fatty changes in the epithelium.

6. **Microscopical Appearance.**

a. The epithelial changes are simply a more advanced condition of the tubal changes described under acute nephritis. Epithelial proliferation has become extreme; the cells are filled with fatty particles, and the proliferation of granular leucocytes becomes a very evident feature, so that many of the tubes appear to be full of pus. Some of the tubes are blocked with hyaline casts, others with granular or fatty casts.

b. The interstitial changes are secondary to the epithelial (with the reservation of Klein's account of scarlatinal nephritis). They consist in the production of nuclei around the glomeruli and between the tubes, with thickening of the Malpighian corpuscles, and ultimately, it is said, in the development of the nuclei into fibrous tissue, so that in time the granular or cirrhotic kidney is produced. Interstitial changes which undoubtedly occur in chronic cases lead to dimpling and irregularities of the surface, which is often described as granular.

N.B.—Two disputed points are connected with the foregoing accounts. It is universally admitted that acute nephritis (large red kidney) leads to large white kidney. It is disputed—

a. Whether the large white kidney may not sometimes arise as a spontaneous chronic disease.

b. Whether the large white kidney ever becomes the contracted granular kidney.

Contracted Granular Kidney.

Chronic interstitial nephritis, "gouty kidney," "cirrhotic kidney."

1. **Size and Weight.**—Often more or less diminished, sometimes extremely so. May not weigh more than two ounces; half the normal weight is common.

2. **Shape.**—Much altered. Reniform shape more or less lost. Form irregular. Surface granular.

3. **Colour.**—Normal.

4. **Capsule.**—Thickened, adherent; when removed tears away part of kidney substance.

5. **Section.**—The cortex is greatly diminished in thickness, in extreme cases to a mere shell. Pattern of structure lost. Tissue blurred and indistinct.

To the above may be added two other characters:—

a. Presence of cysts on the surface or in the substance of cortex, some microscopic, some the size of a pea or larger.

b. Thickening of the arteries throughout the organ, and very often atheroma of the main renal artery, the smaller branches of which stand out like quills on section.

6. **Microscopically.**

(1) There is *apparently* to some pathologists, *certainly* to most, a great increase in the connective tissue of the kidney. The tubes are everywhere separated by great numbers of nuclei or fibres. The

different opinions arise in this way. The apparent fibrous tissue is said by Johnson and Moxon to be resolvable into atrophied and shrivelled tubes; it is stated by these observers that if high powers are used all stages from a more or less healthy tube to the apparent fibrous tissue may be made out. However this may be in a section of granular kidney, the so-called fibrous tissue certainly looks like normal fibrous tissue.

(2) The walls of the tubes are found to be more or less thickened and fibrous, the epithelium either entirely lost, or the cells altered in shape, granular, or fatty.

(3) Malpighian bodies. The capsule is more or less thickened and fibrous; the capillary tuft is atrophied and shrivelled; and an abnormal number of Malpighian bodies will be seen in the field, sometimes as many as seven or eight.

(4) The cysts are very variable in size, and are sometimes arranged in rows like the beads of a necklace. They possess a thin wall, and there is an epithelial lining consisting of a single layer of flat cells. These cysts have various modes of origin assigned to them :—

a. That they are produced from the convoluted tubes of the cortex, which become constricted at intervals by the fibrous tissue, the intervening portions expanding into cysts.

b. That they are produced from the Malpighian corpuscles, the flattened epithelial lining being very suggestive of this mode of origin (Wilks and Moxon).

c. That they are the result of overgrown dropsical cells (J. Simon).

d. Sometimes in the Malpighian pyramids bead-like rows of minute cysts are met with. These

are supposed to arise from the looped tubes of Henle. According to Virchow the process is that the contiguous walls of two or more tubes degenerate and atrophy, and by these means several tubes become converted into a cyst.

(5) The vessels become greatly thickened. The cause of this is under controversy. The affection of the renal arteries is only a part of the general change in the arteries throughout the body.

It is disputed whether—

a. The thickening is merely hypertrophy of the muscular coat (Johnson), or

b. Thickening of the external coat (Gull and Sutton).

As a matter of fact, both changes are met with, and in addition more or less atheromatous degeneration in the case of the larger branches of the artery.

Small White Kidney.

This is an important form, because it raises the question as to whether—

(1) It is the large white kidney contracted down to granular, or

(2) The red contracted kidney become affected by acute epithelial nephritis.

It may be taken almost as certain that if a young adult dies with the symptoms of granular kidney, the small white kidney will be found (*cf.* below).

1. **Size and Weight.**—Small.
2. **Shape.**—Deformed.
3. **Colour.**—Pale yellowish white.

4. **Capsule.**—Thick and adherent; the **surface** is granular and irregular.

5. **Section.**—The cortex is thin, of the same colour as the surface, and often contains cysts; the vessels are thickened.

6. **Microscopically.**—In addition to the changes in the connective tissue (viz., an increase), thickening of the capsules, and deformity of the Malpighian bodies, the tubes will be found blocked by proliferation of epithelium, and sometimes by casts. In short, the appearances are those of interstitial nephritis with those of epithelial nephritis.

Clinically.—The symptoms are generally attacks of dropsy and scanty urine (acute nephritis) in a patient who is already the subject of chronic nephritis.

Cystic Kidney.

Cysts are found in the kidney under four conditions:—

1. Thin-walled cysts, containing a clear fluid, varying in size from microscopical dimensions to a pea or hazel-nut, but found in the cortex of otherwise healthy kidneys. They are sometimes associated with cysts in the liver. A case occurred at Guy's in 1886 in which there were cysts in the kidney, liver, cerebellum, and pancreas, these organs being otherwise healthy. These cysts are of no clinical importance.

2. Cysts found in granular kidneys. These and their various modes of origin have already been described.

3. True cystic kidney is—

(*a*) Congenital, or
(*b*) Met with in adults.

a. **Congenital Cystic Kidney.**

Congenital cystic disease is bilateral; it renders the fœtus inviable, and is associated with other malformations, *e.g.*, hare-lip, cleft palate, spina bifida. Both organs are compared to masses of cysts which relatively to the body weight are enormous. Virchow's explanation is that this condition is due to atresia of the straight tubes of the Malpighian pyramids, and the consequent dilatation of the other tubes beyond the constriction, and thus the formation of cysts.

b. **Cystic Kidney of Adults.**

It is quite possible, though not proven, that this may be a late condition of congenital disease, which during fœtal life was not sufficient to destroy life, but which has undergone steady and progressive increase. At all events, this condition has no clinical feature, and is very often met with in an autopsy for some entirely different cause of death. But it is not uncommon for the final termination to be **uræmia**. This condition is bilateral.

Naked Eye Appearances.

1. **Size and Weight.**—Often enormously enlarged (three to four pounds common, eight to nine pounds sometimes).

2. **Shape.**—More or less irregular. The general renal shape is maintained frequently, but is often lost by the irregular projection of cysts from the surface.

3. **Capsule.**—Thickened and adherent.

4. **Section.**—The organ is converted into a mass of cysts of very variable size and contents. Sometimes there does not appear to be a trace of renal structure. Sometimes a small portion will be found at the surface in some part. The cysts vary greatly both in size and in the nature of their contents, which may be clear and

limpid, or thick and viscid, or gelatinous; the colour may be straw-like, red, brown, or blood-stained.

Microscopically.—The walls of the cysts are composed of renal tissue in an advanced condition of interstitial nephritis, and are actually composed of fibrous tissue. They are always lined (if there be any lining at all) with flat epithelium.

The cyst contains degenerated epithelium and blood cells, and cholesterine.

Chemically.—The salts of serum and altered blood, probably never any of the urinary constituents.

N.B.—The probable position of the adult cystic kidney is that it is a granular kidney in which cyst formation is out of proportion to the other changes.

Lardaceous Kidney.

1. **Size and Weight.**—Enlarged, and the enlargement sometimes becomes great.

2. **Shape.**—Normal.

3. **Colour.**—Pale yellowish white.

4. **Capsule.**—Strips easily and leaves a smooth surface.

5. **Section.**—The cortex is smooth, and the normal pattern remains distinct.

Tested with iodine, the part turns a deep mahogany brown. The first parts to show reaction are the bases of the Malpighian pyramids, and the Malpighian bodies.

The later stage differs from this, for the lardaceous material appears to set up irritative changes, and in consequence both tubal and interstitial changes supervene.

6. **Microscopically.**—**Early Stage.**—The small arteries are infiltrated by the translucent gelatinous material.

The change begins in the inner coat, and invades the middle; and when the condition is extreme, the arteriole is converted into a translucent tube. The Malpighian glomerulus is converted into a little translucent mass. The epithelium of the tubes remains distinct, and the tubes become full of hyaline casts. These casts are said (by Dickinson) to exhibit the lardaceous reaction.

Later Stage.—When the epithelial and interstitial changes are added, the tubes become blocked with proliferated and altered epithelium. The connective tissue undergoes great increase, and the surface becomes granular; and in cases where the interstitial changes are in excess, the organ becomes contracted, and more or less deformed.

It is universally admitted that in the small contracted granular kidneys, that exhibit the lardaceous reaction, the initial change is the lardaceous.

A kidney exactly like that described as "small white" very frequently shows the lardaceous reaction, and this furnishes one mode of origin of this form of kidney.

Relation of the Different Forms of Bright's Disease to One Another.

1. Large red = Acute nephritis.
2. Large white = Chronic epithelial nephritis.
3. Small red = Granular kidney, interstitial nephritis.
4. Cystic kidney.
5. Small white mixed 2 and 3.
6. Lardaceous.

We may dispose at once of the cystic and lardaceous forms.

Cystic Kidney is a form of granular kidney in which

cyst formation is the predominant feature, and when it has any clinical aspects they are those of granular kidney.

Lardaceous Kidney stands by itself, and has the course just described.

Acute Nephritis either gets well, or forms the large white kidney. This is universally admitted.

Large White either arises in acute nephritis, or is a chronic change from the first. The last is not universally admitted; the first is.

Large White probably in the majority of cases is fatal as such. But in a small minority it is said to run on to granular kidney. Dickinson advocates this origin of the granular kidney strongly, but only for a small proportion. He quotes several cases from children, of which the following is one of the best: "A boy died at the age of eleven, who had had good health until attacked by scarlet fever three years before. He was never well after, and suffered from headache, vomiting, and drowsiness. He got albuminuric retinitis, and urine contained albumen, granular and fatty casts. He died with granular contracted kidneys" (*cf.* above).

To the epithelial changes interstitial hypernucleation is added, and the nuclei become converted into fibrous tissue, and the kidney becomes small, contracted, and granular; *i.e.*, one mode of origin of the small white is in acute nephritis, but it seems certain only in a small proportion of cases.

Granular Kidney (small red) appears to arise as follows:—

1. Insidiously, without any known cause, in most cases.
2. In connection with gout.
3. In connection with chronic lead poisoning, probably without gout.

4. In a few cases as a result of acute nephritis through the large white kidney.

5. Probably from chronic alcoholism.

The preceding refer to the small red kidney. Now, there is little doubt that acute nephritis may arise in a red granular kidney. The result is that epithelial changes are added to the interstitial, and the small white kidney results.

Small White may arise—

1. As a consequence of lardaceous disease.

2. As an infrequent result of acute nephritis (through the large white).

3. As an acute attack in the red contracted granular kidney.

General Effects of Chronic Bright's Disease.

The following effects are for the most part common to all the kidneys described under the head of Bright's disease, but with different degrees of severity and variation in the particular forms:—

1. **A loss of albumen, imperfect excretion of the urinary products, and their accumulation in the blood.**

a. The loss of albumen in the most extreme cases is but small, probably not exceeding a few drachms a day, so that there must be some other factor than this to account for the anæmia and wasting.

b. Probably the circulation of the non-purified blood is the other cause of perverted nutrition.

The question is raised as to what the substances are which by circulation in the blood set up uræmia.

It is true that a large quantity of urea may be

injected into the veins of animals, and the only effect will be its rapid excretion by the kidneys. But it is said that if the ureters or renal arteries be tied, the injection of urea will produce uræmia. Experiments on the latter point are, however, very variable.

Frerichs thought that the noxious material was ammonium carbonate $(NH_4)_2\ CO_3$, produced by the decomposition of urea by the action of a ferment in the blood. He further points out that uræmic patients have a peculiar ammoniacal smell. Experiments have been made by—

(i.) Analysing the breath and blood of uræmic patients, but with discordant results.

(ii.) Injecting $(NH_4)_2\ CO_3$ with or without ligature of the renal arteries and ureters, or extirpation of kidney. Sometimes convulsions have been produced, but never coma; others have failed entirely.

A third view, which, however, is only introduced to make the subject complete—for it only refers to uræmic coma and convulsions—is that these conditions are due to œdema of the brain and coincident contraction of small arteries. It does not make this view more acceptable that uræmia is very common in granular kidney in which there is little or no dropsy.

2. **Thickening of the vessels.**—No doubt the arteries are thickened, but controversy has arisen as to what elements of the wall are changed.

a. Where is the obstruction?

(i.) Bright put it in the capillaries.

(ii.) Others put it in the arterioles.

In favour of the former view it is held that the tissue changes take place in the capillaries; and that when the impure blood circulates through them the vital

capillary force of the circulation is lost, and the circulation correspondingly retarded.

In favour of the obstruction being in the arterioles, an observation of Gowers is in point, for he found that the arterioles of the retina were firmly contracted in a case of uræmia.

b. What is the change?

(i.) Dr. Johnson says the change is found in the **hypertrophy of the muscular coat** of the small arteries. He says that this coat is invariably found hypertrophied, and he endows these vessels with a conservative property; that is, that these vessels contract in order to keep impure blood away from the tissues. This of course would raise the blood pressure. There is not the least reason to suppose that the small arteries have this function in health.

(ii.) Gull and Sutton's view.

Arterio-capillary Fibrosis.—These pathologists state that the change is in the outer coat, and that it results in its thickening and conversion into a hyalo-fibroid material. They affirm that Bright's disease is not a disease special to the kidneys, but that the kidneys only take part in a change common to the whole body. Of course the effect of thickened arteries will be impeded circulation, and consequently hypertrophy of the heart. Now, Gull and Sutton point out what is undoubtedly true: that the following classes of cases are met with:—

a. Common class of granular kidney with thick vessels and hypertrophied heart.
b. Moderately granular kidney with ditto.
c. Healthy kidney with ditto.

And they contend that the sequence is *c*, *b*, *a*.

No doubt there is truth in both statements. What is found generally is thickening of the outer and also of the middle coat, and this combined with more or less degeneration (atheromatous).

3. **Hypertrophy of the heart.**—Probably (Wilks and Moxon) the only case of simple hypertrophy without dilatation is met with in chronic Bright's disease. On account of the raising of the blood pressure, the left ventricle thickens ; but sooner or later this compensatory relation is lost.

There must be a limit to the hypertrophy, but the renal and arterial changes still progress. Then the heart will begin to dilate, and in time the cavities behind will begin to reach to the increased pressure ; and enormous hearts with all the cavities thickened and dilated are thus met with. The dilatation of the ventricle will drag open and enlarge the mitral orifice, and **mitral regurgitation** often ensues.

N.B.—The type of the renal heart is either simple thickening of the left ventricle or a large heart with all the cavities hypertrophied and dilated, valvular disease and adherent pericardium being *absent.*

Quain describes **sclerosis of the connective tissue** of the heart as the result of Bright's disease. But there is no doubt that the increase in size is due to true hypertrophy of the muscular fibres ; and these cases of fibroid disease belong to another category.

Masses of fibrous tissue are met with in the substance of the heart muscle, perhaps most frequently in the septum ventriculorum ; and in many cases they are clearly proved to be due to syphilis.

4. **Dropsy.**—This affects all the tissues of the body (beginning in the eyelids and scrotum), the connective tissue generally, the larynx and lungs; passive effusion into the serous cavities, which must be distinguished from inflammatory effusion.

Bright considered renal dropsy to be inflammatory, and this view is held now by some pathologists (Moxon). The point which raises the question is the partial distribution of the dropsy—*e.g.*, ascites without excess of fluid in the calibre of the intestine.

But the general view of renal dropsy is that it is mechanical, and the result of three causes :—

(i.) The watery condition of the blood.

(ii.) The high blood pressure.

(iii.) The degenerated condition of the vessels which, although thickened, have lost their vital functions.

5. **Hæmorrhages.**—The result of—

a. High tension.
b. Degenerated vessels.
c. Altered blood.

The commonest are—

(i.) **Retinal**; (ii.) **cerebral**; (iii.) **epistaxis**;

And then perhaps—

(iv.) **Cutaneous,** in the form of purpura of more or less severity.

But it may come from any source—from stomach, lungs, etc.; and of course blood is met with in the **urine,** but this is part of the disease itself in all cases of any acuteness.

6. **Tendency to Inflammation**—

(i.) Of mucous membranes; *e.g.*, gastritis, enteritis, bronchitis, pneumonia.

(ii.) Of serous membranes; *e.g.*, pleurisy, peritonitis, pericarditis.

(iii.) Of connective tissues; *e.g.*, erysipelas, œdema (erythema).

All inflammatory conditions in Bright's disease are liable to be suppurative.

7. **Functional Effects.**

(i.) Dyspnœa or hurried breathing without pulmonary lesion.

(ii.) Vomiting, diarrhœa, without lesion.

(iii.) Headache, drowsiness, delirium, mania, without cerebral lesion.

(iv.) Coma and convulsions are of course most common. All these are believed to be the result of uræmia.

All the preceding are common to Bright's disease, but the different effects vary according to the form of kidney.

a. In acute nephritis the tendency is to dropsy and uræmia.

b. The amount of cardio-vascular change appears to be a question of time, so that there is little of it in acute nephritis and much in chronic granular kidney. Dickinson maintains that even in acute nephritis the arterioles are decidedly thick. Therefore granular kidney manifests itself more frequently as heart disease than in any other way.

The vascular changes being more extreme, it follows that hæmorrhages are most common in chronic granular kidney. Cerebral hæmorrhage is a very usual mode of manifestation of Bright's disease.

c. In acute nephritis and large white kidney the tendency is to inflammatory affections. Pleurisy and pericarditis are generally suppurative.

Lardaceous kidney is characterised—

(i.) By great infrequency of cerebral complication.

(ii.) Tendency to pneumonia and inflammations generally.

(iii.) As regards dropsy, it occupies an intermediate position, having acute nephritis with extreme dropsy on the one side, and granular kidney with very slight dropsy on the other.

Surgical Kidney.

Better called **acute suppurative nephritis**. Bright's disease never leads to suppuration. This kidney results from the inflammatory condition of the urinary passages, and is secondary to—

1. Cystitis, calculus vesicæ, and enlarged prostate.
2. Cystitis, the result of spinal disease.
3. Or it is pyæmic, and the cause of the pyæmia is generally to be found in some operation on the pelvic organs. The pyæmia may also be from bone disease.

Description.

1. **Size** is increased.
2. **Capsule** strips readily.
3. **Surface** is smooth, but dotted all over with small yellow points, corresponding to minute abscesses.
4. **On Section.**—Cortex is swollen, and kidney is yellowish white in colour. If many sections are made, the yellow dot on the surface is seen to correspond to the linear collection of pus corpuscles passing in a

radiating way through the cones and cortex. This can only be made out by continuous sections on account of the tortuous arrangements of the tubes of the cortex.

The most characteristic appearance is the presence in the cortex of linear collections of pus arranged in a radiating manner, and sometimes these collections in a lucky section will be found to communicate with the yellow spot on the surface.

Microscopically.—The chief point is the recognition of the collection of leucocytes in the midst of the renal substance in which the tubes are filled with altered epithelium. It seems probable that suppurative nephritis is not of necessity fatal. Cases are met with in which there has been antecedent obstruction to the outflow of urine, *e.g.*, a calculus, which had passed away or been removed; and in these the connective tissue around the kidney has been found greatly thickened, the capsule also greatly thickened and adherent to the connective tissue, so that in removing such kidneys the capsule has remained behind, and the surface has seemed scarred.

Of course, as it is often due to urinary obstruction, it is frequently associated with "dilated pelvis and calyces," "consecutive Bright's disease," and "pyonephrosis."

Hydronephrosis.

Results from obstruction to the outflow of urine in some parts of the urinary passages. This obstruction is not always demonstrable. It may be inferred in some such cases that the calculus, the original cause, has passed down the passages or become dissolved, or perhaps undergone spontaneous disruption (Ord).

Causation.

1. Impaction of calculus.

2. Obstruction in the ureter, in the bladder, or in the urethra; *e.g.*, stricture of urethra from ulceration set up by calculus, enlarged prostate, stricture of urethra, and in female pelvic cellulitis, ovarian and other tumours. Certain causes have been described by Roberts and others which are more or less doubtful, viz.—

(1) Congenital. The strong point here is the occasional association with other congenital defects—hare-lip, etc.

(2) Too great obliquity of the junction of the ureter with the pelvis; urine collects in the pelvis and presses the fold down like a valve. When the distension becomes great it acts on the opposite wall of the ureter, and relieves the pressure by a copious flow of urine. The continuance of these processes for a long time causes hydronephrosis.

(3) Abnormal distribution of the renal artery. A branch coming off and passing across the ureter enters the substance of the kidney lower down. This is supposed to have caused a certain amount of obstruction, which would of course increase as the pelvis became dilated.

(4) In a case described by Dr. Hare, there was a large hydronephrosis, and the upper part of the ureter was twisted in a spiral, the coils of which were adherent. When the coils were separated the urine ran readily through the ureter.

Whatever the cause of obstruction, the first effect is dilatation of the pelvis, then of the calices; then the pressure begins to operate on the renal substance, and the first thing noticed is flattening of the papillæ. If

the pressure increases, first the pyramids, then the cortex, become flattened out, and the final result is a large sac either without any remnant of renal structure at all, or only a small portion at one point. The sac is often more or less divided up on account of the greater or less persistence of the calices. Of course all stages are met with. The renal changes that take place in the kidney substance are exactly the same as take place in granular kidney; *i.e.*, there is an interstitial nephritis, thickening of the connective tissue, formation of cysts, atrophy of Malpighian bodies, and thickening of arteries. This is Fagge's "consecutive Bright's disease."

The fluid of hydronephrosis is clear, pale yellow, and contains small amounts of the urinary constituents (urea, uric acid, and salts). Sometimes it consists of pus, when the condition is known as **Pyonephrosis.** The presence or absence of pus seems in many cases determined by the acuteness of the obstruction. The slow, intermittent, and gradually increasing obstruction results in hydronephrosis; a sudden and complete one, as by a calculus, results in pyonephrosis.

Scrofulous kidney sometimes produces pyonephrosis.

Tubercular Kidney.

Two forms—(1) Miliary tuberculosis, always secondary.
(2) Primary tuberculosis.

1. **Miliary Tuberculosis** is always secondary, and is part of a general diffusion of tubercle. It seems that the kidney in a case of tuberculosis follows the lung as regards frequency. This disease, which consists in the scattering of small round yellow or grey miliary tubercles, chiefly in the cortex, has no clinical significance.

2. **Tubercular Disease** may be a primary affection.

The first deposit of tubercle takes place at the apices of the Malpighian pyramids, or between the cortex and medulla. Very quickly the pelvis is involved, and tubercular pyelitis may precede the renal affection. The tubercle is deposited very quickly, and becomes yellow and caseous, and softens. The tubercular disease of the pelvis leads to thickening and swelling of the mucous membrane and obliteration of the ureter. In consequence of these two factors—

(i.) The pelvis and calices dilate, and the renal substance becomes more or less flattened out. The mucous membrane of the pelvis of the kidney becomes converted into the thick (quarter-inch to one inch) layer of quite structureless material which under the microscope is resolved into the fatty and granular debris of degenerated tubercle.

(ii.) The tubercular masses soften down and form vomicæ. The sum-total of these two processes (*i.e.*, dilatation and tubercular deposit and cavitation) is that the kidney becomes more or less enlarged, the pelvis and calices dilated and coated with a thick layer of yellow caseous material, the kidney containing cavities of various sizes, the walls of which are coated with the same yellow matter. The extreme result is an organ riddled by cavities, in part arising from dilated calices, in part from softened tubercle. These cavities are lined by yellow, caseous, softened, tubercular matter, and according to the stage of the disease more or less renal substance is left. This disease is often a primary one of the kidney. It is unilateral at first, but sooner or later affects both organs, and finally sets up in most cases general tuberculosis.

The tubercular disease passes down the ureter, and in some cases involves the mucous membrane of the

bladder. Sometimes it passes along the vas deferens to the epididymis by direct continuity. But sometimes in primary tubercular renal disease deposit takes place in the epididymis concurrently. In extreme cases the tubercular disease advances along the urethra, and the mucous membrane undergoes the same thickening and caseous degeneration, so that in rare cases symptoms of stricture of the urethra may result. In the female the same caseous change takes place in the mucous membrane of the Fallopian tubes and uterus. This condition is rare, but not extremely so. The parts affected are found to be lined by a layer, more or less thick, of yellow structureless material which under the microscope is resolved into granules and fat cells.

Malignant Disease of Kidneys.

Varieties.

(i.) Almost always **soft carcinoma (encephaloid).**

(ii.) **Round-celled Sarcoma.**—It is of interest that in sarcomata of the kidney in acute cases striated muscular fibre has been made out ("Pathological Transactions").

(iii.) **Scirrhus, Epithelioma,** and **Colloid.**—These cancers are all very rare.

Malignant disease *occurs* in—

1. **Children** under five years, who, according to Sir W. Roberts's figures, are especially liable.

2. **Adults,** from the age of twenty upwards.

In the vast majority of cases it is unilateral. It always begins in the cortex (Roberts) and in the renal epithelium, the connective tissue being involved secondarily.

It may be **nodular** or **infiltrating**.

In the first case it will lead to irregularity in form, nodules of varying size projecting from the surface.

In the second case it causes uniform enlargement, the renal shape being maintained.

It is either **secondary** or **primary**.

When secondary it is part of a general dissemination of cancer, and, *per se*, is not of importance. It occurs in the form of scattered nodules.

As primary, it is seen in children and adults.

Description.—The kidney is always enlarged, frequently enormously so. Tumours weighing thirty pounds have been taken from children; this is extreme, but the kidney generally weighs several pounds.

The **structure** of growth of the kidney is the same as that of carcinoma and sarcoma elsewhere, placed in the midst of healthy renal structure.

On cutting into the kidney it is found to be composed of large tracts of soft yellow cancerous material, in the midst of which large hæmorrhages, recent and old, are conspicuous.

Small portions of healthy renal substance may be met with.

The pelvis is nearly always affected, and the ureter blocked.

Secondary growths of the lumbar glands are met with most frequently, and increase the irregularity of the tumour. Secondary growths may be met with in any other organ.

As regards duration, there is discrepancy; but Roberts has found that it lasts longer than any other cancer. Probably an ordinary duration is *two years*.

The other kidney undergoes compensatory hypertrophy.

[**Renal Calculi.**

Renal calculi are most frequently composed of (1) uric acid, (2) oxalate of lime, and (3) phosphate of lime; carbonate of lime and cystine have also been found, but are rare.

In most cases small concretions formed in the substance of the kidney rapidly pass into the urethra and bladder, but occasionally they remain in the tubes or calices of the kidney, and these gradually increase in size. All parts of the kidney may become the seat of calculi, but the urate and oxalate calculi are more often found in the substance of the kidney than the phosphate and carbonate of lime which when present are always confined to the pelvis and infundibula.

The effects of a calculus on the kidney depend on its size; at first it will cause merely irritation and congestion, but this condition is likely to be followed by inflammation and perhaps abscess either within or without the capsule of the kidney. These changes are more likely to occur when the stone is in the substance of the kidney, but when it is confined to the pelvis chronic pyelitis is more likely to ensue, with changes of an atrophic character caused by pressure; the pelvis dilates, the renal substance wastes, and a large stone remains, sending processes into the calices, in shape like a cauliflower, and with little covering, except a thin layer of cortex beneath the capsule of the kidney. Generally the calculi are single or perhaps two or three in number, but occasionally they are so numerous that the organ feels like a bag of stones.—Eds.]

[Casts.

Urinary casts may be formed in any part of the kidney. They are mostly cylindrical in shape, often somewhat coiled and bent.

Their length varies. In diameter the casts are from $\frac{1}{500}$th to $\frac{1}{1000}$th of an inch. Casts consist of a ground substance of a transparent or very faintly granular appearance; its nature is at present uncertain. In addition to this ground substance, there are other constituents which give to the different varieties their distinguishing appearances.

The following are the different varieties:—

1. **Epithelial Casts.**—These are chiefly composed of renal epithelium more or less altered from its normal condition.

2. **Hyaline Casts.**—These mostly have a clear, transparent, and glassy appearance, but occasionally have some faint markings on the surface; though often called "waxy" and occurring in a lardaceous kidney, it is extremely doubtful if they are ever composed of amyloid material.

3. **Granular Casts.**—These contain an opaque granular material mixed with altered epithelial cells, broken-down blood corpuscles, and oxalate crystals.

4. **Fatty Casts.**—These contain fat globules, often of a large size; these globules may arise in any of the foregoing varieties.

5. **Blood Casts.**—These consist mainly of blood corpuscles, more or less altered in shape, held together by fibrin; pus casts so called may thus be produced.

Clinical significance of casts.—Although the information obtained from the examination of casts may be very

valuable, yet care should be taken that several examinations of the urine are made, and all other evidences well weighed before a conclusion be arrived at; the following few points may be borne in mind:—

1. **Epithelial Casts** point to an early stage of disease.

2. **Hyaline Casts.**—The **large** forms of this variety usually occur in chronic nephritis; being produced in tubes denuded of epithelium, they indicate an advanced state of disease.

Small hyaline casts are formed both in acute and chronic disease; they therefore must be considered only in conjunction with other signs, as the history of the case, etc.

3. **Granular Casts** point to advanced chronic disease, especially if the casts be large and coarse.

4. **Fatty Casts** show that fatty degeneration is taking place, and therefore may be of the gravest import, or may, on the other hand, indicate commencing recovery.

5. **Blood Casts** are diagnostic of hæmorrhage into the tubes, due either to arterial hyperæmia or to venous congestion.—Eds.]

CHAPTER VI.

[THE STOMACH AND INTESTINES.

Gastric Ulcer.

THE following are points of interest concerning this disease :—

1. It is scarcely ever met with in children below ten years of age (only two cases out of 226, Brinton).

2. Females are more liable than males (three to one).

3. Want of food, mental anxiety, overwork, and intemperance have been regarded as predisposing causes.

4. Virchow considers the commencement of ulceration is the blocking of an artery in the wall of the stomach, and consequent malnutrition of the part of the wall supplied by that artery; this allows the wall to be acted on by the gastric juice, and the formation of an ulcer.

Gastric ulcers are most often found singly, but there may be two or even more. Their favourite seat is on the lesser curvature near the pylorus, then the posterior wall, then the anterior wall. Occasionally the ulcer is directly on the pylorus, and sometimes in the first part of the duodenum.

The following are the characteristics of a typical gastric ulcer :—

Shape.—Circular at first, but may become irregular from extension or the junction of two or more ulcers.

Edges.—Even and sharply defined at first, "punched out"; afterwards they become indurated and thickened.

Floor.—Smooth at first, but may become sloughy, or covered with granulations. As each subsequent coat is destroyed over a smaller area than that above it, the ulcer becomes cone-shaped, the base being towards the mucous membrane.

In time, the ulceration extending through the wall of the stomach to the peritoneal coat, the peritoneum covering the floor of the ulcer becomes thickened and opaque, and adherent to whatever part may be opposed to it. Occasionally the peritoneum sloughs before any adhesion is formed, and this causes perforation into the peritoneal cavity; in other cases the ulcer may erode an artery and cause hæmorrhage if the vessel be not already thrombosed. Cicatrization may take place at any stage; the cicatrix formed may be smooth or puckered, and may give rise to a variable amount of distortion of the stomach.

Malignant Disease of Stomach.

Excepting the breast and uterus, no part is so often the seat of malignant disease; it is almost always *primary*.

Varieties.

a. **Scirrhus,** by far the most common (Dr. Brinton says three-fourths the whole number of cases).

b. **Encephaloid,** in about one-tenth of the cases.

c. **Villous Cancer** (so called).—This consists of sprouting processes made up of fibrous tissue

loaded with well-formed cells, each possessing a loop of blood-vessels.

d. **Cylinder Epithelioma,** rare.

N.B.—All the above varieties in the stomach are very liable to undergo *colloid degeneration.*

The most frequent seat of the disease is the pyloric orifice (60 per cent. of the cases), then the cardiac orifice; the fundus is scarcely ever affected.

The growth generally begins in the submucous coat, and by extension soon involves the other coats. Often degeneration takes place early, and an ulcer, with ragged edges and an uneven floor, is formed. Adhesions often form with other organs, which become involved by extension; occasionally perforation takes place into hollow viscera or other parts.

When the pylorus is affected obstruction may be caused, the stomach become dilated, sometimes enormously so (six to seven pints), and the walls become hypertrophied. When the cardiac orifice is affected, the stomach will be found shrunken and contracted. If the middle of the body of the stomach be alone involved, the cavity will be contracted at this part, and the organ assume an hour-glass shape.

Causes.—Little is known as to the causes of gastric cancer. The following points may be noticed:—

a. Age. Majority of cases occur between fifty and sixty years of age.
b. Predominance of the disease in males.
c. Hereditary predisposition (*e.g.*, Napoleon family).
d. Local injury.
e. Any long-continued irritation (*e.g.*, spirit-drinking, hot condiments).

In addition to the above, the following tumours are met with in the stomach :—

i. Sarcomata.
ii. Lipomata.
iii. Adenomata.
iv. Tubercle—very rare.

Ulcers of the Intestine.

May be divided into two classes :—

1. **Non-specific.**

a. Ulcers due to direct injury of the mucous membrane by substances in the lumen of the bowel.
b. Simple catarrhal ulcers.
c. Duodenal ulcers similar to gastric ulcer, as mentioned above. Ulcers are also met with in the duodenum as a result of burns or scalds of the surface of the body.
d. Ulceration extending into the bowel from some lesion situated outside it ; this is very rare.

2. **Specific.**

a. Tubercular.
b. Typhoid.
c. Dysenteric.
d. Syphilitic.
e. Malignant.

Typhoid Ulcers attack Peyer's patches and the solitary glands. They are mostly confined to the ileum and cæcum, but the whole of the large and small intestine may be involved. They are always more developed the nearer they are to the ileo-cæcal valve.

The primary change consists in a hyperæmia and cellular infiltration of the glands; they become of a greyish white or pale reddish colour, and stand out above the surrounding mucous membrane; the infiltration in many parts extends into the surrounding tissues.

The whole of these processes may subside, and the new elements become absorbed, but the most characteristic change is death of the new tissue, which separates as a slough, and thus the typhoid ulcer is formed.

Description of ulcer (*cf.* Tubercular Ulcer, page 23).

Edges.—Thin and undermined, and consist of a fringe of mucous membrane.

Base.—Smooth, and consists of muscular or submucous coat, in some cases of the peritoneum. There is no thickening or induration of the base.

Shape.—When in a Peyer's patch, elongated in long direction of the bowel; when in a solitary gland, more or less circular.

The ulcer tends to cicatrize; and when cicatrization is complete, there is no diminution in the calibre of the gut.

N.B.—Perforation and hæmorrhage are liable to occur.

Dysenteric Ulcers are for the most part limited to the large intestine, mostly in rectum and descending colon. They in some cases involve extensive areas, large portions of the mucous membrane being converted into black rotten sloughs. When the loss of substance has been great, so that when healing takes place the

edges of the ulcers cannot approximate, portions of the bowel are lined with connective tissue instead of mucous membrane. If the disease be at all chronic the wall of the gut tends to become thickened, indurated, and more or less contracted.

Malignant Disease of the Intestine.

Seat.—Preferably the rectum and sigmoid flexure, then the cæcum and colon generally, the jejunum and ileum being rarely affected.

Varieties.—Same as gastric. Columnar epithelioma is most often found in the rectum and sigmoid flexure.

The growth follows the course as described in gastric cancer. The scirrhus and encephaloid growths are liable to develop in an annular manner, involving the whole circumference of the bowel, and thus tending to narrow the lumen (sometimes so as to barely admit a probe).

Malignant disease of the intestine is generally **primary**.

In addition to the carcinomata, the following tumours are met with in the intestine: sarcoma, fibroma, fibromyoma, lipoma, angioma, and adenoma; the last-named is the most common.—Eds.]

[The Spleen.

The following conditions may give rise to enlargement of the spleen:—

1. Cardiac disease (obstructive, *cf.* page 58).
2. Pulmonary disease (obstructive).
3. Hepatic disease (obstructive).

The above act by causing *passive hyperæmia* of the

organ; the enlargement is often only slight, if any; the consistence of the spleen is firmer and more fleshy than usual, and the capsule generally is thickened, and sometimes presents hard thickened patches. On section the organ appears of a dark purple colour.

4. Typhus.
5. Enteric.
6. Ague.
7. Pyæmia.

} Specific Fevers.

These act by causing *active hyperæmia* of the spleen; the condition may be met with in other cases, besides the above, where there has been long-continued pyrexia.

The enlargement is often considerable, from three to four times its usual size; the capsule is stretched, and on section the spleen is soft, diffluent, and of a dark red colour; in very acute cases the condition may even lead to rupture of the organ.

N.B.—For the Infarcted Spleen of Ulcerative Endocarditis *vide* chapter on "The Heart," page 53.

8. Leukæmia.

In this condition the enlargement is usually great. The shape of the organ is not much altered, and the splenic notch can be readily felt during life. The capsule is generally thickened, and adhesions with the adjacent viscera are often met with. On section the consistence is firmer than normal, the colour is brown or brownish red, and thickened trabeculæ may be often seen. The Malpighian corpuscles are seldom prominent, especially when the enlargement is ad-

vanced. Embolic infarcts of various sizes may often be found as reddish yellow, wedge-shaped masses on the surface of the organ.

9. Rickets.

In this disease the enlargement is generally slight, and is due chiefly to interstitial connective tissue.

10. Lardaceous disease of the spleen is common; the organ is often greatly enlarged; it presents a firm yet somewhat doughy consistence, and on section has either a peculiar translucent appearance, or else it is studded with small bodies resembling boiled sago (the sago spleen), according to whether the disease be extreme or not.

The following new growths also cause enlargement of the spleen; they present the same general characteristics as in other parts of the body :—

1. Tubercle (rarely, if ever, primary, *cf.* page 26).
2. Carcinoma (secondary).
3. Sarcoma (secondary).
4. Lymphadenoma.
5. Hydatid cysts.
6. Simple, serous, or mucous cysts.—Eds.]

CHAPTER VII.

THE BRAIN.

Cerebral Hæmorrhage.

Blood may be extravasated within the cavity of the skull in four situations :—

a. Between dura mater and bone.
b. Arachnoid cavity.
c. Substance of brain.
d. Ventricles.

a. Between the dura mater and bone hæmorrhage is generally the result of an accident, and is usually accompanied by fractured skull and rupture of middle meningeal artery.

b. Into the arachnoid cavity it may result from hæmorrhage into the brain bursting into it. But true cases of arachnoid hæmorrhage occur—

i. In whooping-cough.
ii. In infants, the result of difficult parturition.
iii. In adults, the result of chronic bronchitis.

The blood is supposed to come from ruptured vessels passing between dura and pia mater.

c. Into the substance of the brain. This may occur in any part, but the most common is the corpus striatum. According to Charcot, there is an artery which runs through this body, and which is particularly

large and unsupported, and which is the source of hæmorrhage, "the artery of cerebral hæmorrhage."

N.B.—In cases of cerebral hæmorrhage the extravasation is not unfrequently multiple; that is to say, if there be *one* large cerebral extravasation there may be smaller ones in other parts, and probably the most frequent seat of the secondary extravasation is the "pons."

The question is often raised as to which is the primary lesion. It may be that all are simultaneous; but this is not likely, on clinical grounds or as a matter of probability; in the case of an individual with bad arteries, the great disturbance of cerebral circulation, due to the considerable extravasation of blood in one part, probably does account for extravasation in other parts.

The clot of fatal cerebral hæmorrhage varies up to two ounces.

Causation.—Charcot's view of the causation is that cerebral hæmorrhage is due to the rupture of minute aneurisms (miliary aneurisms). The first step in the process is inflammation of the outer coats of the artery, leading to atrophy of the muscle of the middle coat and subsequent yielding of the arterial wall ("Pathological Transactions," 1888). Whether these aneurisms are always present in cerebral hæmorrhage is a question of considerable doubt. But that they do occur is certain. They are, no doubt, identical with the arterio-capillary fibrosis of Gull and Sutton.

Another well-ascertained cause of cerebral hæmorrhage is **embolism**, under the condition that the embolus is derived from a source of suppuration, *i.e.*, practically ulcerative endocarditis. The embolus becomes impacted

at the point of bifurcation of an artery (left middle cerebral), and sets up ulceration in the coats of the artery and subsequent perforation.

Another cause seems to be **syphilis**, which leads to disease of the cerebral arteries (Heuibner; it is named after him).

a. It is limited to the vessels at the base.

b. It consists of proliferation of nuclei in the inner coat, which subsequently becomes converted into fibrous tissue. Thus it leads to narrowing of the calibre of the artery, and generally thrombosis; and probably now and then it may lead to rupture.

Another and very common cause is chronic Bright's disease, leading to atheromatous disease of the arteries, arterio-capillary fibrosis (Gull and Sutton), periarteritis and miliary aneurism (Charcot).

The effect of a recent hæmorrhage is of course more or less destructive of the brain substance. If not rapidly fatal, the extravasated blood becomes gradually absorbed. The colouring matter changes, by which the colour is altered from that of recent blood to brown or a brick-red.

N.B.—The discovery of these patches of discoloration is the most frequently found evidence of cerebral hæmorrhage. **Apoplectic cysts** are far less common. In these cases the blood is completely absorbed, and a cyst is formed, the walls of which consist of sclerosed brain material (*i.e.*, connective tissue becoming fibrous), and which contains a clear fluid. The cyst is often traversed by strands of connective tissue. It is believed that such a cyst

may be formed in six weeks. But there is no doubt that hæmorrhages of years' standing may be revealed by brownish discoloration. Hæmatoidin crystals are found in such discolorations.

Cerebral Softening.

Several forms are described.

Red Softening.—Effect of embolism; cerebritis around hæmorrhage; traumatic abscess of brain.

When a cerebral artery is blocked up by an embolus, the result is not anæmia of the embolised area, but hyperæmia. This is supposed to arise because these arteries do not anastomose. The arterial wall beyond the embolus has its blood supply cut off, and in consequence it dilates passively. This creates negative pressure, and blood rushes backwards from the veins through the capillaries, and with such force as to cause hæmorrhage. The result is, brain tissue is broken up, small extravasations occur, and further the brain tissue undergoes degeneration, a constant proof of which is the presence of **compound granule cells** (corpuscles of Gluge). These are large cells full of oil globules, which by transmitted light show as black dots, and the whole corpuscle is likened to a mulberry (cf. page 52).

N.B.—These corpuscles form the "only certain evidence" of true softening of nerve tissue.

Red softening may take place very quickly after embolism, certainly within twenty-four to thirty-six hours.

Yellow Softening takes place after a time. The extravasated colouring matter of the blood in red softening undergoes changes, one result of which is

that it becomes of a yellow-ochrey colour. Therefore one form of yellow softening is—

a. Further stage of red (granulation corpuscles found).

b. But around tumours or clots the brain substance is often yellow and somewhat soft. This is only œdema. There is no destruction of nerve elements, nor are compound granule corpuscles found.

White Softening.—True white softening is met with in aged persons with diseased arteries and feeble hearts. Possibly in such persons there may be a slow thrombosis of the diseased arteries in addition. The result is that a variable extent of brain substance undergoes slow degeneration and softening. Compound granule cells are met with. The probable actual cause is slow starvation of the nervous tissue.

White softening in meningitis (see Meningitis).

The brain substance in the neighbourhood of the lateral ventricle becomes soft, but still the colour remains unaltered. This is especially the case in the fornix and the corpus callosum, which become quite diffluent. This also is œdema, no destruction of nerve elements occurs, and no compound granule cells are found.

The important forms are—

Embolic red softening.
True white softening.

Owing to the imperfect anastomosis of the cerebral arteries, the prognosis as to recovery is more grave than in the case of destruction of the brain substance the result of hæmorrhage.

Cerebral Tumours.

There are few situations in which a greater variety of tumours may be met with than in the cranial cavity. They should be divided into—

1. **Primary.**
2. **Secondary.**

Although not strictly a cerebral tumour, sarcoma of the skull should be mentioned. The question is raised whether these commence in bone or in dura mater,

a. From the fact that they are associated with other bones; and

b. Secondly, that the inner surface of the dura mater is often quite smooth over the tumour.

The osseous origin of the tumour seems probable.

They may grow either way, and appear as pulsating tumours of scalp, or may indent the brain.

They are probably primary tumours of the skull. Their microscopical character is sarcomatous.

I. Primary Cerebral Tumours.

Large Solitary Tubercle.

The term "solitary" is not always appropriate. There may be two or three. This is the form of tumour most common in children, and occurs quite independently of pulmonary disease. The favourite situation is the cerebellum, but the pons is frequently affected. They consist of variable-sized rounded yellow masses, firm, not often softening in the centre, sometimes as large as a billiard-ball, and surrounded by a narrow zone of pinkish grey semi-translucent substance. If this zone

be examined the structure of typical miliary tubercle will be found. This is the growing edge. The bulk of the tumour is made up of structureless granular material.

Gumma.

These are nearly always the result of acquired syphilis. They originate in the membranes, and invade the brain secondarily. They have a special predilection for the region of the middle cerebral artery and the base of the brain. They are generally multiple, and so give rise to very variable and irregular symptoms. The irregularity of paralyses met with in cerebral syphilis is often of the utmost use in diagnosis.

Microscopically.—Gummata belong to the same class as tubercle, consisting of leucocytes which degenerate into a granular detritus in the centre, but which are surrounded at the periphery by a zone of well-formed fibrous tissue.

Glioma of Brain.

This is a primary tumour, generally single, but sometimes multiple. The growth shades off insensibly into the surrounding brain tissue. It may be grey in colour, resembling the grey matter of the brain, or may be more or less white, or quite of a deep red. These, however, are liable to softening and degeneration ; and being very vascular, they are often dotted with hæmorrhages.

Microscopically.—It seems to be determined that the nerve cells have nothing to do with the origin of these tumours, but that they arise from the neuroglia. In structure they are composed of round or oval cells,

resembling those of neuroglia, which give off numerous long, delicate processes, and these interlace and communicate in all directions so as to found a reticulated stroma. It is often difficult and sometimes impossible to distinguish by the naked eye a glioma from a patch of softening.

Myxoma.

This is found as a primary tumour, and often reaches an enormous size. It consists of myxomatous tissue —*i.e.*, large stellate cells giving off processes which intercommunicate (connective-tissue cells) — in the meshes a gelatinous fluid containing **mucin**, and more or less fibrous tissue. The probably correct view of myxoma is that it consists of ordinary connective tissue, the alveoli of which have become distended with a form containing mucin.

Various Forms of Sarcomata.

Almost any variety from the soft, rapidly growing, round-celled sarcoma, through the spindle-celled, to the growths largely composed of fibrous tissue (fibro-sarcoma). These tumours especially affect the base and the parts in the neighbourhood of the body of the sphenoid.

Cholesteatoma.

A rare kind of tumour consisting of a roundish mass of semi-translucent material of the appearance of white wax. Tumours have been met with, generally single, sometimes multiple, which consist simply of an over-growth of the natural grey matter.

The above constitute the most important forms of primary cerebral tumours. Clinically, however, the following are primary :—

Abscess.
Aneurism.
Hydatid.

Cerebral Abscess.

When not the result of injury, is always pyæmic ; at least, those cases which appear to be exceptions are so rare that in all probability some septic source has been overlooked.

Disease of the temporal bone or of the cribriform plate of the ethmoid.

Pulmonary disease, especially bronchiectasis and old empyema, may act in the same way.

But cerebral abscess may be associated with any source of pyæmia.

Situation. — When due to temporal diseases, it is usually in the temporo-sphenoidal lobe or cerebellum. Often there is a layer of normal brain substance between the diseased bone and the abscess.

When due to other causes, there is a special localisation.

When recent, the abscess is irregular in form with shreddy ragged walls, filled with thick yellowish-green pus.

After a time **a capsule** is acquired ; and if it be of the thickness of about a quarter of an inch the abscess is certainly of six to seven weeks' duration. This capsule is composed of granulation tissue inside, and fibrous tissue externally.

In the course of time the **contents** alter in appearance.

The pus cells undergo degeneration ; and it is thought that now and then central abscesses may dry up and furnish the calcareous masses met with rarely in the brain.

Cerebral Aneurism.

Is due to embolism or atheroma, and is met with generally in one or other middle cerebral artery.

Cerebral Hydatid.

The echinococcus is exceedingly rare (three cases in Guy's) ; has generally occurred in children. The cyst is generally barren (acephelo-cyst). In one of the Guy's cases the liver, in the other the lung, was affected. Both were quite young girls. The cysticercus cellulosæ has been found in the membranes and in various parts of the brain. It is more common than the other hydatid. The cysts are usually multiple and barren, about the size and shape of an olive.

II. Secondary Tumours.

Take after the primary origin, being secondary to breast, etc., *e.g.*, cancer, sarcoma (myeloid, etc.), enchondroma and osteoma, etc.

Meningitis.

We must distinguish between inflammation of the dura arachnoid and pia arachnoid.

In **dura arachnitis** the pus is found in the arachnoid cavity. If on opening the cranium or spinal canal the arachnoid cavity is found containing pus, except in a few cases we may be sure that there has been "injury or disease of the bones." The exceptions are certain rare

cases of epidemic meningitis, although even this disease is a pia arachnitis.

The common causes are disease of the petrous part of temporal, syphilitic caries of the calvarium; a less common cause is necrosis of the cribriform plate, which itself is often syphilitic. Fracture and injury to the dura mater may of course occur in any part.

In the case of the spinal column the cause is extensive bed-sores eroding the bones and laying open the spinal canal.

The amount of pus is often very great, and is sometimes very fœtid. As complications, thrombosis of the cranial sinuses and cerebral abscess are common.

Clinically.—The symptoms may be a mixture of those of general pyæmia and those of meningitis.

The amount of paralysis depends on the amount of pus.

Pia arachnitis, or what is generally understood by the term "meningitis."

Causation.—Injury, pyæmia, syphilis, epidemic meningitis, acute alcoholism (?), rupture of cerebral abscess, tubercle. (Tubercle is placed last because it constitutes a special form.)

It must be remembered that the pia mater constitutes the blood supply of the brain, both on its surface and (by means of the velum interpositum) the interior. If this membrane be inflamed, it will necessarily follow that the whole of the brain will be affected, and that therefore what is called meningitis is really encephalitis. In describing meningitis we must have regard to—

a. The surface.
b. Whether the vertex or base be affected.
c. The lateral ventricles.
d. The brain substance.

Meningitis **from injury** almost always affects the vertex, and the same is also true of many cases, if not all, of simple meningitis.

Tubercular Meningitis, except in very rare cases, affects the base, and the base only.

Simple Meningitis is also distinguished from tubercular—

a. By the large amount of purulent lymph effused, whilst in tubercular the lymph is almost entirely absent.

b. Sometimes by the small amount of fluid effused into the ventricles.

In both cases the inflammatory products are in the meshes of the pia mater. The arachnoid surface is generally dry and sticky-looking. But this is due to the fact that the intraventricular pressure drives out the arachnoid fluid and to a certain amount of proliferation of the endothelial lining.

Simple Meningitis.

1. Large effusion of greenish yellow pus in the sulci of the vertex, and often of the base, sometimes sufficiently copious to hide the convolutions.

2. Pia mater when stripped drags away the softened cortical grey matter.

3. The effusion of turbid fluid into the lateral ventricles usually is not great in amount.

4. White softening of boundaries of the lateral ventricles, chiefly marked in corpus callosum and fornix.

5. Usually is a most acute disease of three or four days' duration.

Tubercular Meningitis.

1. Convolutions flattened as the result of intraventricular pressure.

2. Membrane tears away the softened cortex.

3. Presence of tubercles. These are found about the middle cerebral artery; but they may spread further, and even invade the vertex.

These tubercles may be very obvious as grey or yellow grains about the size of a millet seed downwards, or they may only be ascertained microscopically (Fagge).

4. The amount of lymph is not usually great, and is found in—

a. The sylvian fissure,
b. Diamond-shaped space at base of brain, and
c. The extremity of the vermiform process, just where the veins of Galen join the straight sinus.

5. The lateral ventricles are usually distended, and in extreme cases three to four ounces of clear or generally slightly turbid fluid may be met with.

6. White softening of surrounding brain substance, especially fornix and corpus callosum.

N.B.—In the account of white softening it was stated that this form was a mere œdema, and not a true softening. It would be more correct to describe it as inflammatory œdema, so distinguishing it from passive. No compound granule cells are met with; but there are early indications of inflammatory processes.

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