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THE STUDENT'S HAND-BOOK

OF

MEDICINE & THERAPEUTICS

BY

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TO

JOHN WYLLIE, Esq., M.D., F.R.C.P.Ed.;

M.R.C.S.Eng., etc.;

Physician to Edinburgh Royal Infirmary;

JOHN PLAYFAIR, Esq., M.D., F.R.C.P.Ed.;

Physician to the Royal Hospital for Sick Children;

G. A. GIBSON, Esq., M.D., D.Sc., F.R.C.P., F.R.S.E., etc.,

Assistant Physician, Edinburgh Royal Infirmary;

This Book is respectfully Dedicated

IN TOKEN OF MANY ACTS OF KINDNESS SHOWN BY THEM
TO THE AUTHOR.

PREFACE.

IT may be thought that the number of Text-Books on Medicine are already more than sufficient — Why therefore add another? As a tutor of large experience I venture the opinion that a work such as this aims to be, is a distinct want in the Student world. Practitioners have long complained that recently qualified men are of little use to them on account of their lack of *practical* knowledge; moreover, the General Medical Council have endorsed this verdict, and given emphasis to it by adding a year of *practical* clinical work to the ordinary curriculum. But a question which should and must be answered is—"Why or how is it that in these days of splendid lectures and excellent Text-Books the recently qualified man is so impractical? The cause is not far to seek. We must admit that the Student is lectured to, far too much; each subject that he is taught is expounded to him as if it were the essential one to the exclusion of others; and the fact is ignored that a practical knowledge of Medicine can only be obtained by the application of the principles of anatomy, physiology, and pathology to the deviations from the normal standard of health as observed at the bedside of disease. The Student's day in fact, is largely taken up in listening to theories, brilliant and fascinating no doubt, but not always correct or profitable, and by evening

time he too often is bewildered or disheartened. He has then to read the excellent Text-Books that are at his command; and by the time the comprehensive descriptions given therein are waded through, little time is left for ward work. In other words, the exquisite and complete Text-Books now published are sources of danger if not used at the proper time— for to a sharp Student they afford such graphic pictures, he fears not the *absence* of ward work for his examination; and, on the other hand, the less bright Student fails to grasp the more essential points; in either case clinical or bedside experience goes to the wall. The aims of the writer of this volume are therefore two-fold:—

1. To enable the Student to digest the main features of the various diseases in the most concise manner at the time he is engaged in clinical work, thereby enabling him to verify *at the bedside* the statements he has read.

2. To enable the busy practitioner to see at a glance, the principal points of each disease, which he can elaborate by his experience.

I have further ventured to introduce an alteration on the plan usually adopted in writing small Text-Books, viz., I have placed before the reader a synopsis of the anatomy, or physiology, or both, of the organ or system dealt with, before describing the diseases of those various organs. This I hope will direct the reader to anticipate what should or ought to occur when the system in question is diseased,—*i.e.*, to *think out the probable symptoms*, not learn them off like a parrot. No one is more sensible than myself of the many short-comings of this work, and though I have no right to expect leniency at the hands of my critics, hope they will not judge me by too high a standard, but that

they will appreciate an honest attempt to present the main facts of medicine in such a way that the student need not spend the time perusing voluminous works which would be more profitably employed in acquiring a *practical* knowledge of disease at the bedside. I have left out the Diseases of the Skin, as no useful purpose would be served by giving a condensed account of them. In writing this work, the author has reaped his information from most of the leading and latest Text-Books, the monographs of Gowers, Paton, Erb, Douglas Powell, Ralfe, Horsley, the useful atlas of Byrom Bramwell, etc. I must, however, specially thank Dr John Wyllie for the liberal use of his notes, and my friend, Dr Ryland Whitaker, for valuable help in the section on parasitic diseases. I wish also to express my gratitude to my friends, Miss Euphie Cumming, Dr Teacher, and others, for reading the proof-sheets of the latter portion of the book; and to Miss Giffen for the compilation of the index. In conclusion, I return my sincere thanks to my publishers and their manager for courtesy at all times.

THE AUTHOR.

31 Morningside Drive,
February 1894.

ERRATA.

Page	92	(8th line), fourth should be	'sixth.'
,,	146,	phination ,,	'phonation.'
,,	202,	opposition ,,	'apposition.'
,,	249,	nitrate ,,	'nitrite.'
,,	74,	loop ,,	'roof.'
,,	98,	flying ,,	'fly.'

THE STUDENT'S

HAND-BOOK OF MEDICINE

AND THERAPEUTICS.

FEVERS.

FEVERS are characterised by a grave disturbance of the system, attended with great *increase of temperature* and *diminished secretions;* or in other words, *more* fire and *greater* accumulation of ashes. All the specific or "zymotic" diseases are probably due to special germs setting up chemical or fermentative changes in the blood. Some of these organisms we know something about, of others we know nothing. Pasteur was the first to elaborate the fermentation theory.

Suppose we put into a bottle—water, sugar, and barm; then cork it tightly, and let it stand in a warm place. At first *nothing* seems to happen, but after a time a remarkable change takes place—CO_2 and alcohol form; and a sediment sinks to the bottom, that is, the *sugar has been changed by the living yeast cells into poisonous compounds;* this process stops, as so many waste and poisonous products are formed that the *yeast cells themselves are killed!*

Again,—put into a bottle some sugar and a solution of *nitrogenous* material (say clear mutton broth), and instead of corking, *expose it to the air.* Again fermentation takes place, and a scum soon rises to the surface. If the scum

be examined under the microscope it will be found to consist of millions of cells = bacteria. The fluid, instead of the harmless one *it once was, has become highly poisonous;* it rapidly decomposes, and gives rise to a most disgusting odour.

Here then are two cases of fermentation: the one induced by cells *introduced by human agency,* the other by bacteria *which got in from the air.* In both cases, poisons were formed, and the *bacteria,* like the yeast cells, were killed through their own products, or through exhaustion of suitable food, and thus the fermentative process was stopped.

Now apply this to specific fevers. Take scarlet fever:—first, the patient complains of general malaise, then severe symptoms develop, and the sufferer may hang between life and death. Next a rapid improvement takes place, and health becomes more or less quickly restored. Is it not probable that certain bacteria in the air or in the food taken gained an entrance into the blood, and *having there found suitable soil* began to multiply, to set up chemical changes, and to form poisons which resulted in the production of scarlet fever — how easy to understand the whole event!

As in the yeast experiments, we have—

A quiescent period—i.e., period of incubation.

A period of brisk fermentation = development of the fever.

The arrest of fermentation = recovery of the patient, either rapidly by *crisis,* or with slight exacerbation of fever by—*lysis.*

We have then the following facts to go upon—

1. Bacteria are in the air and food.
2. Bacteria can cause disease.
3. Bacteria in order to cause disease must have *proper food or soil.*
4. Diseases which result from bacteria must have—
    (1) An incubative period.
    (2) A period of brisk multiplication, and therefore severity of symptoms.
    (3) A period of decline.

We learn more, however, for we find that as regards treatment we can—

First—help to prevent the formation of suitable soils by hygienic measures.

Secondly—help to make an already suitable soil *unsuitable*, by antiseptics, stimulants, and tonics; in other words, fight the bacteria by making the resistance greater, through increasing the vitality of the tissues.

To sum up: decrease if possible the number of bacteria attacking, and make the soil more resistant; also support the patient's strength till the fight is finished. Let me impress upon beginners, however, that "prevention" promises a far better prospect than cure: for bacteria, once they flourish gaily in the body, are fearfully difficult to exterminate.

In describing any fever, begin with the *marked* features of the invasion, *then* emphasise—

1. The *special* symptoms of the fever.
2. The manner in which it declines.
3. The complications.
4. The sequelæ.

Remember that *all* fevers have more than one type; always at least two—the simple and the severe; some, indeed, have many forms, such as small-pox and scarlet fever.

All may assume what is termed the "*Typhoid State*"—a condition of great gravity. In order to avoid constant repetition, we shall in the following pages, describe, only the peculiar feature of each "malaise," and sum up the later and more severe symptoms of each fever under the term "Typhoid State," but note particularly that this condition is *not* synonymous with Typhoid Fever.

TYPHOID STATE.

The Typhoid State is characterised by—

1. A decline of the previously more acute symptoms.

2. Pulse becoming rapid and soft.

3. Tongue dry and brown, tremulous, and protruded with difficulty.

4. Collection of sordes (a mixture of dried mucus and bacteria) around the teeth and lips.

5. Increased muscular prostration — tremors and subsultus tendinum.

6. Semi-comatose state, coma vigil = pupils dilated, but patient does not see.

7. Picking at the bed-clothes and muttering delirium.

8. Slipping down to the foot of the bed.

This condition becomes well marked in *all* the malignant forms of fevers — is very well seen in small-pox, typhus, typhoid, pneumonia, etc.

Morbid Anatomy. — Each fever has its special foci, but usually in addition to their *peculiar* lesions, *all*, *when severe*, show more or less changes in the tissues generally, viz.—

1. Blood is dark and more fluid (thick in cholera).

2. Muscles dark-coloured, and show granular degeneration.

3. Heart is softened, perhaps dilated.

4. Congestion of the viscera, *spleen*, *liver*, *and kidneys* particularly.

5. Hypostatic congestion of lungs.

PHYSIOLOGY OF FEVER.

Before discussing the treatment of fever we must consider a few physiological questions. In fever we have a disturbance of the heat-regulating centre, so that in addition to the pernicious products of the specific germ, *we have also to consider the excessive waste of tissue, and diminished excretion* (pro ratio), with, consequently, the accumulation within the blood of tissue waste products, which are also poisonous in

their nature. Thus a cycle of retrograde changes commences. The liver being engorged does its work less efficiently, proteid waste is not broken down so completely, and the kidney has to excrete *not only a greater amount of waste nitrogenous material, but material of a more irritating nature*, and this too, when by reason of the deterioration of the blood, the organ itself is but poorly nourished.

GENERAL TREATMENT OF ALL FEVERS.

I. Medicinal.—The indications of treatment are sufficiently obvious—

1. Diminish heat production.
2. Increase heat loss.
3. Help the secretory organs.
4. Keep the heart going.
5. Treat complications as they arise.

Under the first heading we may employ antipyretics—

Drugs—(1) Antipyrin group.
(2) Salicylates.
(3) Quinine.
(4) Aconite.

Under the second heading we may choose—

Diaphoretics—Liq. Ammon. Acet.
Alkaline group generally.
Jaborandi.

To help the secreting organs—

(1) Diminish the amount of nitrogenous food, and substitute milk and farinaceous material.

(2) Keep bowels moderately open. *Make the urates more soluble* by administration of citrates, tartrates, etc.

And lastly, to keep the heart going, we may employ—

(1) Digitalis.
(2) Strophanthus.
(3) Strychnine.
(4) Diffusible stimulants, brandy, ether.

No routine treatment can be prescribed, because of the many complications and peculiarities of each case. In most cases we may start with a purge, if there be no contra-indication.

℞ Hyd. Subclor. grs. iv.; followed by
℞ Haust Sennæ Co., ℥iss in 4 hours.

This may be combined with a mixture taken at intervals, say, a tablespoonful every four hours—

℞ Liq. Ammon. Acet. Conc.	℥iij.
Spt. Ætheris Nitrosi .	℥ij.
Potass. Citratis . . .	℥ij.
Aqua Camph. ad. . . .	℥viij.

This will keep skin and kidneys acting; the citrate also rendering the urates less irritating.

So much for routine treatment, the complications will be considered under each fever.

II. Hygiene.—Frequently "*drugs*" will form the least important factor in successful treatment, and in *all* cases their action will be assisted by careful diet and strict attention to hygienic principles.

The essentials are—

1. A large well-ventilated room, with blinds which may be so arranged as to let in plenty of light when wanted, or to exclude light if necessary.
2. An absence of unnecessary furniture, which only serves to form a nidus for retention of the germs.
3. A well-trained, non-officious nurse.
4. Absolute cleanliness.
5. Strict attention to physician's orders.

DELIRIUM

Is such a common complication, we may take it here. There are two types commonly met with—

1. Raving maniacal delirium, of early stages.
2. Low muttering delirium, which occurs later.

In the first case we may employ depressants—

℞ Chloral Hydras. . . ℥ss.
Pot. Bromid. grs. xl.
Aqua ad. . . . ℥iij.

One half at once, the other half repeated in one hour if required.

The second type must be met with stimulants—ammonia, alcohol in large doses is recommended by some in addition; indeed, we may sum up in similar words the *treatment of the typhoid state—i.e.*, diffusible stimulants with alcohol must be the principal remedies.

ALCOHOL IN DISEASE.

Brunton sums up as follows:—If the alcohol tends to bring the patient nearer his normal condition it is doing good; if it takes him away further from his healthy condition, it is doing harm. For instance, if alcohol renders the tongue moist, slows the *quickened* pulse or the hurried breathing, if it renders the skin cooler when hot and dry, and if it lessens delirium and brings on sleep, then use it. If the converse happens, then withhold it—typhoid or any other condition.

Alcohol is chiefly indicated during the small hours of the morning, when attendants are sleepy; the fire perhaps gets low and the external temperature is generally lowered. (Whitla.)

INCUBATION AND RASHES.

The incubative period of most fevers is from two to twelve days. Scarlet fever and chicken-pox seem to have the shortest.

The following table is fairly correct—Rash appears in—

Chicken-Pox . .	1st day.
Scarlet Fever . .	2nd day.
Small-Pox . . .	3rd day.
Measles . . .	4th day.
Typhus . . .	5th day.
Typhoid . . .	7th to 12th day.

Most rashes disappear on pressure, *if not hæmorrhagic.* If hæmorrhagic they do not disappear on pressure *or after death.*

TYPHOID FEVER.

A specific infectious fever marked clinically by diarrhœa, a rose-coloured rash (appearing in successive crops), running a course of twenty-one days, and ending by lysis.

Ætiology. — Prevails most in autumn months. Attacks both sexes from fifteen to twenty-five years of age. Rare in infancy or over sixty.

The poison is conveyed principally by—

1. Water, contaminated by stools.
2. Soiled linen of typhoid patients in public laundries.
3. Milk.
4. Drains.
5. Direct contact with stools of patients.
6. Contamination of the soil. (PETTENKOFER.)

The stools, when *fresh are not poisonous*, but become so after fermentation has commenced. The most virulent period being from twelve to thirty-six hours after having been passed.

Sewer gas, filth, etc., do not of themselves cause typhoid fever, but cause a suitable soil for the typhoid bacillus to multiply in the intestines.

Specific Germ—known as the bacillus of Eberth. It is *short*, *thick*, and has rounded ends. Some authorities say it is identical with the "bacterium coli commune" *found in all diarrhœas*, but it differs in chemical re-actions. The bacilli act by forming (1) certain ptomaïnes = typho-toxin, and (2) tox albumin. Cultures are easily destroyed by corrosive sublimate 1–2500. The bacilli are found in the intestinal glands, spleen, and stools.

Morbid Anatomy.—Principally inflammation of the lymphoid tissue of the lower portion of the ileum, with more or less catarrh throughout the bowel.

Peyer's Patches—

1st Week.—Are swollen through infiltration of lymph corpuscles, the surfaces raised and fawn-coloured—the infiltration involves the submucous coat.

2nd Week.—The surface becomes abraded; sloughs form, which are often bile-stained.

3rd Week.—Sloughs come away, leaving ulcerating surfaces. Typical typhoid ulcers are thus formed. A few solitary glands undergo the same process.

Mesenteric Glands may undergo the same changes, but more often become swollen, red, and tender only, or break down into cheesy masses.

Other Organs.—Spleen and liver are enlarged; heart is soft and flabby. The voluntary muscles undergo vitreous degeneration; in fact, similar changes to those found after death from high temperature, etc. (See page 4.)

Symptoms.—*Insidious onset*—headache, epistaxis, increasing weakness till patient is compelled to take to bed.

1st Week. — Temperature runs up in remarkable staircase manner — *i.e.*, rising two in the evening and falling *one* in

the morning. (Not always.) The malaise becomes more marked, and exhaustion increases with the onset of—

(1) Diarrhœa—the stools quickly assuming their characteristic appearance.

(2) Abdominal tenderness and enlarged spleen.

The specific rash appears on seventh to twelfth day.

2nd Week is marked by the symptoms becoming more aggravated; the morning temperature does not remit. A tendency to *typhoid state* comes on, and death may take place.

3rd Week is marked by even more profound symptoms. Perforation is much more likely to occur at this than any other period, or perhaps fatal epistaxis or the pneumonic condition may end the scene.

4th Week.—The temperature becomes normal, and usually convalescence may be now said to *commence.* Frequently, however, relapses occur.

Complications of typhoid fever are—

1. Peritonitis, with or without perforation.
2. Hæmorrhage: from bowel, nose, or mucous surfaces.
3. Embolism of femoral vein.
4. Meningitis.
5. Septic Pneumonia.
6. Early "typhoid state" or early cardiac failure.
7. Hyperpyrexia.

SPECIAL POINTS.

The *Rash* comes out in successive crops, on the seventh to twelfth day, as small rose-coloured spots raised above the surface, slightly convex; they appear first on the abdomen and chest; and fade on pressure. Frequently no rash appears.

The *Ulcer* of Typhoid Fever—

Lies in the longitudinal axis.

Edges—thin, undermined.

Base—may be formed of either—

1. Sub-mucous coat.
2. Muscular tissue, or
3. Peritoneum only.

They show a tendency to perforate, but *do not cause constriction after they heal.*

The Stools.—Very fœtid and ammoniacal; pea soup in appearance, and contain—fæcal matter, sodium chloride, triple phosphates, and frequently blood, shreds of mucous membranes, and sloughs.

	TYPHUS.	TYPHOID.
Age	Males, 25 to 35.	Both sexes, 15 to 25.
Cause	Dirt; filth; overcrowding.	Contamination with typhoid stools; defective drainage.
Onset	Abrupt.	Insidious.
Course	Ends by crisis on fourteenth day.	By lysis after the twenty-first day; frequent relapses.
Chief Symptoms	Referable to the nervous system; more delirium, and early appearance of typhoid state.	Referable more to the alimentary canal; nervous symptoms and typhoid state later.
Pupils	Contracted.	Dilated.
Bowels	Confined.	Loose.
Rash	Dark dusky mulberry; comes out on fifth day; tends to become hæmorrhagic.	Rose-coloured, like flea-bites; appear seventh to twelfth day in successive crops.
Temperature	Goes up rapidly, and comes down suddenly.	Rises 2° in the evening; falls 1° in the morning for four days; then oscillates throughout the disease.

Special Points of Treatment.—Here we have to do with a diseased alimentary canal: our treatment must be mainly dietetic.

Give no solid food, or that which would not readily pass through a fine sieve (WHITLA). Milk, *if it agrees*, arrowroot, beef-tea, and chicken-broth are the principal foods.

Medicinal.—Do not be too officious.

General principles hold good. If the diarrhœa becomes excessive give bismuth and opium, or lead acetate and morphia, or an enema of starch and opium.

If constipation be troublesome—enema of warm water, castor oil. No irritating purge.

Hæmorrhage—opium, hypodermic injection of morphia, alum.

Perforation—paralyse gut with opium.

Bed Sores—cleanliness—wash with brandy, gentle galvanism, water-bed.

TYPHUS FEVER.

An acute specific contagious fever, characterised by sudden onset, marked nervous symptoms, a maculated rash, and ending by crisis.

Ætiology.—Known as Jail fever, Ship fever, etc., is much more rare than formerly—occurs amongst the poor, overcrowded, and in dirty districts, affects principally adults between twenty-five and thirty-six years of age. Very infectious, especially to doctors *and nurses*.

Morbid Anatomy. — Those of intense fever, and blood-poisoning, plus the petechial rash. (See page 4.)

Symptoms.—After a period of incubation the fever rapidly develops with a malaise, characterised by intense headache, nausea, and vomiting, with elevation of temperature. There may be rigors, pains all over the body, constipation, contracted pupils, thick-furred tongue, and rapid exhaustion. Mousy

odour, noisy delirium and a dull, heavy, apathetic appearance. The rash appears in about the fifth day—first, on the abdomen, and the extensor aspects of the hands and wrists. As the fever progresses, the typhoid state comes on rapidly: the delirium becomes of the low muttering type—pupils before contracted, may now be dilated; the grave complications tabulated may appear: retention of urine and paralysis of the sphincters; gangrene of the extremities; all these may appear and help to terminate the disease. If the patient does not succumb, usually about the thirteenth day, the temperature falls; profuse sweating, a critical diarrhœa, and an abundance of lithates usually usher in a crisis; after which the patient gains strength rapidly, and may be, in a short time, in better health than before the attack.

Specific Poison.—The organism has not yet been isolated; it is probably a strepto-bacillus, which has been observed by more than one pathologist. Whatever the poison is, there is no doubt that it quickly succumbs to a free supply of oxygen.

The Rash—comes out rapidly about the fifth day. It shows—

1. A sub-cuticular mottling, *dusky-red* in colour.
2. Distinct papular rose spots, which tend to *become petechial*, and, therefore, neither disappear on pressure or after death.

Complications.—

1. Early assumption of typhoid state.
2. Retention of urine.
3. Hyperpyrexia.
4. Gangrene of extremities, or bed-sores.
5. Broncho-pneumonia, most common complication, may become purulent.
6. Parotid bubo, and pyæmic abscesses.
7. Embolism of femoral vein, more common than in typhoid.

It should be noted that, notwithstanding the severity of the cerebral symptoms, meningitis and nephritis are rare; but albuminuria may be present.

Treatment.—*Special points to bear in mind.*—Whatever be the nature of the poison, oxygen has a remarkably destructive power over it; therefore, a plentiful supply of air is indicated in this, perhaps more than any other fever.

The nurse should be one who has either had the fever, or who has nursed "typhus" before. She should be specially instructed to note the tendency to "*retention of urine and bed-sores.*"

This fever seldom calls for lowering treatment, and frequently stimulants are required from the beginning. Employ the general principles detailed in opening chapter. A good mixture is the following—

℞ Acid Hydrobromic Dil. . ℥ss.
Quinæ. Sulph. . . . . ℥ss.
Aqua ad. . . . . ℥viij.
℥ss. 4 hor.

The contracted pupils and delirium suggest absence of excessive light, at least in the earlier stages. The food must be nourishing, and as plentiful as the assimilative powers will admit.

SMALL-POX.

A contagious, infectious disease, characterised by a rash, which usually appears on the third day, and runs through four stages, viz.:—(1) Papular; (2) Vesicular; (3) Pustular; (4) Drying up or Scabbing.

Ætiology.—Common to all ages and both sexes. Fœtus may be affected. Planted in a virgin soil it is very virulent. Its virulence is much modified by successful vaccination.

The contagion spreads by air, infected clothes, contact with people, and contact with contents of pustule. As the primary fever declines on the third day, when the rash appears, the

patients frequently walk about and apply at hospital for advice respecting a "rash," and they may thus infect all in the waiting-room. Of great importance to the State is the fact, that the virus may be given off from the dead.

Symptoms.—After twelve days' incubation, the malaise of onset comes on. Most notable features—*frontal headache, pains in back, vomiting.* Temperature runs up rapidly, with all the phenomena of "fever." On third day fever usually declines, and the rash appears as a papule. Soon, however, the symptoms become worse and more pronounced than before, the fever increases, and on the ninth day, when the rash becomes pustular, the so-called secondary fever occurs, due to absorption of pus. The fever then becomes septic in type. Temperature oscillates. There may be severe rigors and a rapid assumption of the typhoid state. There is great swelling of the face—the eyes may be even closed up—and too frequently the patient dies; or the fever may subside, and the scabs commence to dry, falling off finally on the eighteenth to twentieth day, leaving a more or less pitted appearance.

The Specific Rash first appears on third day of fever, on the face, forehead, and scalp, as slightly raised red papules; feels *shotty* beneath the skin.

Three days later (sixth day of fever) the "papules" become vesicular, at first clear and transparent, then turbid, depressed in the middle or *umbilicated.* Each vesicle is also *loculated*, that is, divided into compartments by delicate connective tissue partitions derived from proliferation of the cells of the rete Malpighii.

Usually about three days later (ninth day of disease), the vesicle becomes *purulent*, accompanied with an inflammatory ring round each vesicle, which causes great swelling and disfigurement of parts affected, and attended with exacerbation of symptoms due to absorption—*i.e.*, secondary fever; then the pustule begins to dry; a black brown scab forms and drops off, leaving a depressed scar as before mentioned.

Varieties.—

1. Modified, as seen occasionally after vaccination.
2. *Simple* or *discrete* = the pocks being few.
3. Confluent = where pocks run into one another.
4. Hæmorrhagic or malignant.
5. Corymbose, where pocks cluster together like grapes.

Other varieties have been tabulated, but they only confuse the student and serve no practical purpose.

How does the hæmorrhagic form influence the clinical course?

1. The patient may be so overwhelmed with the poison that he may die as early as the second day.
2. The hæmorrhage may appear later, and prevent the ordinary stages or course of the rash.
3. In any case the prognosis becomes more grave, and the patient may be carried off by hæmorrhages from mucous membrane, nose, lung, kidneys, etc.

Next to the hæmorrhagic, of course, the confluent is the most severe form —*fever does not fall on the third day.* The fever becomes more severe, the skin enormously swollen, and all the symptoms tend to a low type.

Complications.—

1. Pyæmic abscesses and albuminuria.
2. Suppurative keratitis, with ulceration of cornea.
3. Ulceration of pharynx, or larynx.
4. Septic pneumonia.
5. Hæmorrhage of *all* kinds.

Sequelæ.—

Otitis media.
Abscess of other bones.
Deep pitting.
Blindness.
Peripheral neuritis.

Pathology.—Tissues generally—like those of fever. (See page 4.)

1. *Rash.*—Papular stage.
   (1) Proliferation of cells of rete Malpighii.
   (2) Cell coagulation and central necrosis, due to micro-cocci.
   (3) Layer of leucocytes.
2. Formation of spaces containing—

   Serum.
   Fibrin.
   Leucocytes.
   } Vesicular stage.

   The umbilication corresponds to the area of primary necrosis. Often determined by the site of a hair follicle.
3. Degeneration into pus of vesicular contents—papillæ of true skin are now very swollen and infiltrated with leucocytes—pustular stage.

The blood shows no *specific* changes.

Initial Rashes—The specific rash may be preceded by other rashes (which may appear on second day)—

1. Hæmorrhagic—occupies lower half of abdomen.
   (1) Extends in triangular form, apex at Scarpa's triangle. Extensor aspects.
   (2) Purpura variolosa—constitutes a severe type of disease.
2. Erythematous form—similar to scarlet fever or measles. If this happen, the true rash when it occurs seems to spare the site of the initial rash.

Treatment. — General rules as laid down in introductory remarks.

Special Points are—

1. Use a darkened room when eyes are affected.

2. Prevent itching by smearing with carbolic ointment, or sponging with some antiseptic, non-irritating fluid.

3. Remember to thoroughly isolate, and

4. Bear in mind the good influence of successful vaccination amongst the inhabitants of the affected district.

VACCINATION IN MAN.

Jenner first inoculated cow-pox, with lymph obtained from pustules on the udders of cows. The contents of the pustule so set up may be used = arm-to-arm vaccination.

After inoculation (arm-to-arm)—

A papule forms second to third day.
Becomes vesicular on fifth to sixth day.
Becomes purulent on ninth day.
Dries up and scab falls end of third week leaving a permanent scar.

The neighbouring lymphatics become enlarged. After inoculation the arm usually swells, and more or less fever is occasioned.

Where lymph is used "direct" from the cow, the process is slightly slower.

British law requires all healthy children to be vaccinated before three months old.

SCARLET FEVER.

A contagious infectious disease, characterised by fever, sore throat, and a scarlet rash which freely desquamates.

Ætiology.—Epidemics prevail at all seasons. One attack usually prevents a future one. The poison is peculiarly persistent, and may be carried to a great distance by letters, etc.

Symptoms. — The principal features of the onset are—rheumatic pains, soreness of throat, and headache. Soon, however, the throat becomes very sore, the neck feels stiff, and the glands at the angle of jaw are swollen.

On the second day the rash comes out, first on the chest, then rapidly spreads over the face and body.

As the fever progresses, the throat symptoms become more severe; the tonsils approach the middle line, yellowish points appear. These may coalesce and form a "patch" resembling that of diphtheria. Other severe complications (see tabulated list) often arise and bring about a fatal issue; or the temperature may fall gradually with the fading of the rash, and a slow convalescence is gradually established.

The Tongue is at first covered with thick white fur, with papillæ projecting—*i.e.*, the white strawberry tongue; later, the fur peels off, leaving the typical red strawberry appearance.

The Rash appears on the second day, first as a scarlet blush, but if looked into carefully is seen to consist of small red spots, with red areolæ around them.

The severity of the rash varies, it may be absent or only present in the "flexures" of limbs as bright red lines. Diagnosis is then often difficult, but later on desquamation settles the question.

Desquamation begins as early as the sixth day; it may be only to the extent of roughness, or the epidermis may peel off in large flakes; time occupied = 4 to 8 weeks.

Complications.—

1. Scarlatinal arthritis (may end in suppuration).
2. Endocarditis and pneumonia.
3. Ear troubles are exceedingly common. The suppurative process going on in the middle ear may cause—
   (1) Perforation of the drum.
   (2) Permanent deafness, through suppuration extending to middle ear.

(3) Facial paralysis.
(4) Meningitis.

4. Extensive suppuration of glands.

5. *Scarlatina nephritis* is, however, more of a sequela, as it most often begins about the third week of illness, when the skin is freely desquamating. The symptoms are—

(1) High-coloured or smoky albuminous urine.
(2) Presence of tube casts.
(3) Dropsy of eyelids and ankles, but the dropsy may extend all over the body.

Varieties.—

1. Simple.

2. The Anginoma form, where throat symptoms are severe and a membranous exudation forms, or necrosis of the tissues of the throat, with intense fœtor. Extensive sloughing, causing perforation of the ascending pharyngeal artery or even the common carotid.

3. *Hæmorrhagic form*, in which extensive hæmorrhages occur, epistaxis and hæmaturia; and death takes place on the second or third day.

Special Points in Scarlet Fever.—The skin is peculiarly hot and pungent. Note the tendency to attack serous membranes.

The nephritis is usually late, the explanation being that the kidneys have been not only overworked, eliminating the products of the fever, but in addition are deprived of the support of the secretory function of the skin; and secondly, the new skin, being more sensitive to draughts and chills, causes internal congestion. Undoubtedly these are two powerful factors, but it is probable that the scarlet fever poison has a peculiarly irritating effect on the renal tubes. Scarlatina nephritis is said to affect the *glomeruli* more than ordinary nephritis.

Treatment.—Same general principles detailed in opening chapter.

Special Points are—

1. A minimum amount of *nitrogenous* food.

2. Danger of spreading the disease is greatest during the desquamative period; greater need, therefore, of prolonged isolation.

Complications.—The "Arthritis" demands warm applications. Wrap joints in cotton wool; salicylates internally. (If the kidneys are also inflamed, trust to alkalies internally and opium externally.) Elaterium, pulv. jalapæ co.

Throat.—Antiseptic steam. Painting with tannic acid and glycerine; plenty of hot milk.

For the severe types.—Pure carbolic acid to patches, or perchloride of iron, with free administration of ammonia; quinine, either alone or with perchloride of iron.

MEASLES.

An eruptive fever ushered in with coryza.

Ætiology.—Extremely infectious. Contagion is communicated by the nasal secretions and breath; also by fomites, or a third person. Epidemics occur often, and seem peculiarly associated with or related to whooping-cough, being often followed by the latter.

Symptoms.—*First*, are those of catarrh—eyes water, conjunctivæ become suffused, discharge from nose, and bronchitic symptoms. Often temperature falls on second day, but *rapidly rises with the eruption* of the rash on the fourth day. As the fever progresses the bronchial troubles increase, and the various complications arise.

The Rash.—First appears on *fourth* day at the roots of the hair, and on forehead and face. The rash consists of raised, dark-red spots, spreading in crescentic patches, and giving a velvety feel to the touch. The eruption begins as a hyperæmia around

the sebaceous follicles. There is usually much subcutaneous œdema, and the child presents a peculiar, blotchy, swollen appearance. The rash fades in about three days, leaving a slightly brown-stained appearance which soon passes away. There may be slight, branny desquamation, *but it is always slight.* The rash differs from that of scarlet fever by—

1. Appearing on the fourth instead of the second day.
2. Being darker in colour.
3. The velvety feel.
4. Manner of spreading, but diagnosis is sometimes difficult.
5. The subcutaneous œdema.

The rash may be slightly hæmorrhagic, even in non-severe cases.

Complications.—

1. The extension of bronchial catarrh, causing capillary bronchitis and collapse of the lung, or pneumonia either catarrhal or croupous in nature. Dr John Playfair notes a frequency of *croupous* pneumonia and bronchitis in the same lung. In children, laryngitis is often formidable.
2. Purulent ophthalmia, etc.
3. Otitis media, and swollen cervical glands.
4. Gangrene of skin, vulva.

The various sequelæ are very numerous.

Varieties.—

1. Simple.
2. Hæmorrhagic. Characterised like all other hæmorrhagic forms of fever by bleeding from mucous surfaces, hæmaturia, and an early assumption of the typhoid state.
3. Measles, complicated with diphtheria of the fauces.

Pathology.—Braidwood and Vacher found, in the exhaled breath, lungs, and liver, peculiar bright particles which did not stain with carmine. Nothing definitely is known of the real poison, as no organisms discovered meet the requirements of Koch's law to identify them as being the cause of the disease.

There is no special morbid anatomy except that of the various complications. The respiratory organs, however, always show marked catarrhal changes. Usually some subcutaneous œdema.

Treatment. — General principles hold good; but note the special claims of the respiratory organs, and the tardy convalescence. An expectorating saline mixture may be given early (avoiding squills), antiseptic inhalation, and glycerine applied to the fauces are indicated.

RÖTHELN—GERMAN MEASLES.

A highly infectious, contagious disease, ushered in with catarrh (often ill-marked) and an eruption.

Ætiology.—Was formerly thought to be a mixture of scarlet fever and measles, but is now held to be a separate disease.

Symptoms. — Those of bronchial catarrh, sore throat, and swollen glands; *rarely are there severe complications.* Many patients do not feel ill at all, but Cheadle reports some severe cases attended with albuminuria.

Rash. — Round or oval, slightly raised, pinkish-red spots, discrete usually, but sometimes confluent appear on the first, second, or third day.

Diagnosis.—From Measles by—

1. Short prodromal stage.
2. Absence of the dark colour and crescentic form of the measles rash.

From Scarlet Fever by—

1. Large size of spots.
2. Absence of severe symptoms and desquamation.

Treatment.—General principles.

DIPHTHERIA.

A specific contagious fever attended with grave throat symptoms, and the formation of a false membrane or fibrinous deposit on mucous and abraded surfaces.

Ætiology.—Is endemic in the larger towns, and becomes often epidemic at various seasons. It is highly contagious, and the poison is very concentrated in the *pharyngeal secretion*; it is very fatal to doctors and nurses. (Possibly the spluttering of secretion into the face whilst examining or swabbing out the *patient's* throat may account for this great tendency to attack the attendants.)

Recent evidence shows that most epidemics can be traced to spreading through—

1. Contamination of milk supply.
2. Defective plumbing in water-closets
3. Transmission of the poison from distant parts by the wind.
4. The prevalence of sore throat *prior to epidemic* — *i.e.*, formation of suitable soil for the germ.

Thorne denies it has ever been distinctly traced to polluted water.

Pathology. — *Specific Germ* — It is known as the Klebs-Loeffler bacillus. It is non-mobile, slightly bent and knobbed; and multiplies readily in milk. It is found in the false membrane associated with other germs, etc., strepto-cocci, and staphylo-cocci. The false membrane shows—

1. A heavy network of epithelium.
2. Fibrinous threads entangling masses of leucocytes, and proliferated connective tissue corpuscles.
3. Colonies of micro-cocci with the Klebs bacilli.
4. Granular debris.

Dr Oertel states: the poison first induces a necrosis of the cells with which it comes in contact; the superficial epithelium thus first disappears. The deeper cells become similarly affected, and a zone of inflammation forms around the dead cells; the membrane thus is really a mass of dead cells undergoing hyaline degeneration, and presents the peculiar laminated appearance considered characteristic. The neighbouring lymphatic glands become much enlarged.

The other visceral changes are similar to those of any other intense or malignant fever.

Symptoms. — After an incubation of two to seven days a general malaise sets in; there is *slight* fever, stiffness of neck, swelling of angles of jaws. The soft palate is deeply congested, and whitish patches soon appear; these patches coalesce, become "wash leather-like" in colour, and the false membrane is fully formed. It may extend *all over* the fauces, or may begin on the tonsils first and creeping forward surrounds the uvula like a finger of a glove. The membrane is at first easily stripped, but soon re-forms; then it becomes firmly adherent, and if torn away leaves a bleeding surface. If left alone it may slough off.

The glands in the neighbourhood enlarge, but do not usually suppurate.

The temperature varies, it may be 103° or higher, but is usually from 100° to 102°, and indeed often *subnormal*. This is peculiar, as most acute throat affections have high temperatures. The exudation may now extend in any direction, upwards into—

1. The pharynx, attended with regurgitation through the nose, epistaxis, and a nasal twang of voice.

2. The larynx — "diptheritic croup" — symptoms are stridor, brassy cough, great dyspnœa, and sucking in of the intercostal spaces. *(Some hold this to be the true croup.)*

3. The bronchi, with all the symptoms of severe capillary bronchitis. The membrane after extending to the first bifurcation of the bronchus speedily becomes purulent.

By this time the typhoid condition has been ushered in, and the patient may die at any time from the second to the sixth day from asthenia or from increasing asphyxia.

Albuminuria is frequently present from the earliest period. There is usually no dropsy.

Complications, beside those described, are—

1. Ulcerative endocarditis.
2. Meningitis.
3. Otitis media, etc.
4. Albuminuria with hæmaturia.

The most important sequela is *post-diptheritic paralysis.* This is a peripheral neuritis, and may vary much in its distribution and severity — usually coming on within three weeks of apparent recovery. The more constant symptoms are—

1. Anæsthesia and paralysis of soft palate.
2. Loss of accommodation, with squint or diplopia.
3. Loss of deep reflexes.

The paralysis may be much more extensive, and if the intercostal muscles, or vagi become affected, then the prognosis is very unfavourable. Usually, however, the paralysis passes off in time under suitable treatment.

Special Points.—Note—

1. The tendency for serous membranes to be involved.
2. The marked depression.
3. The red areolæ around the exudation.

Remember the membrane may be very localised, and not visible without a post-nasal or laryngoscopic examination.

Treatment.—

1. General principles, as in Scarlet Fever.
2. Hot antiseptic inhalations and emetics.
3. Painting with chromic acid solution, or pure carbolic acid.
4. Avoid *all* lowering measures.

The "P D" paralysis must be treated with careful hygiene, strychnine, massage, and nourishing diet.

Tracheotomy must always be kept in mind when the larynx is seriously involved.

TRUE CROUP.

An acute affection of the larynx, usually affecting children under five years of age, and characterised by the formation of a membrane, not contagious. Some authorities hold that all cases of *true* croup are diphtheritic in nature. It is true that the larynx when affected in diphtheria presents a most virulent type of croup, but there is also no doubt that we have a highly dangerous affection of the larynx caused by a "croupous inflammation" which is *not* diphtheritic; the membrane formed showing nothing pathognomonic beyond a fibrinous exudation, but the membrane may extend down to the most minute bronchi.

Symptoms. — After a short period of febrile disturbance, catarrh, and fretfulness, the cough becomes brassy, the cry hoarse and metallic, and the breathing much embarrassed.

The inspirations are peculiarly piping or hissing in character, and fatal asphyxia may rapidly supervene.

The Complications may be bronchitis and pneumonia, endocarditis, etc.

Treatment.—Hot bath, hot sponges to throat (some advise ice)—a brisk emetic of ipecac., alum, or mustard and water, followed by a saline mixture. Whitla advises small doses of the following mixture for a child one year old :—

℞	Vin. Antim. Tart. . .	℥iv.
	Vin. Ipecac.	℥iv.
	Syrupi Scillæ	℥iv.
	Aqua ad.	℥iij. misce.

A teaspoonful every fifteen minutes till vomiting occurs, then half a teaspoonful every two or three hours whilst the cough lasts.

Croup differs from diphtheria in—

1. Its onset—*i.e., no special soreness of the throat.*
2. Not being contagious.
3. There being no bleeding surface when membrane is torn off.
4. Absence of the "diphtheria bacillus."

In some cases the diagnosis may be difficult, in that case treat as if it were the graver disease.

Tracheotomy must be performed if dyspnœa becomes extreme, and emetics fail to give relief. Apparently hopeless cases have been saved by the operation.

CEREBRO-SPINAL FEVER.

An acute specific fever characterised by sudden invasion, and attended with painful contractions of muscles, a purpuric rash, and various grave nervous complications.

Ætiology.—According to Taylor, very little is known of this fever. It has appeared in severe forms in Germany and Ireland, but only in small epidemics in England. The disease does not seem to be contagious from man to man, but it is possible that the virus may be transmitted through the lower animals. It affects young adults chiefly.

Symptoms.—The invasion is sudden; there may be rigors, and usually intense vertigo, vomiting, intolerable headache, and noisy delirium. The face is pale, conjunctivæ red, *pupils contracted;* painful contraction of the muscles of the neck, causing *retraction of the head.* Herpes labiales and zoster are most constant symptoms. The sensibility may be so acute that the slightest movements are intolerable. This hypersensitiveness is most marked along the spine. As the fever progresses *tetanic convulsions may bring about a fatal issue from lock-jaw or asphyxia.* Frequently the typhoid state supervenes as soon as the fourth day, the pupils become dilated, anæsthesia takes the place of hyperæsthesia, and the patient quickly succumbs.

Rash.—Usually appears on second day; consists of small purpuric spots, not raised. They appear on the neck and extensor aspects, rarely on the face.

Complications—

1. Purulent effusions into joints.
2. Painful urticaria.
3. Optic neuritis.
4. Hyperpyrexia.

Special Points to note in this meningitis are—

The great tendency to opisthotonus.
The skin affections—herpes, purpura, erythema, etc.
The extremely rapid course.

Pathology.—Not to be regarded as a *simple* meningitis.

1. *Cord.*—Pia mater may be either intensely injected or totally disorganised, and the subarachnoidal space may be filled with pus, causing the dura mater to bulge out. Usually the pus collects most in the lumbar region.

2. *Brain.*—The meninges show intense inflammation, and the ventricles are distended with pus; the foramen of Majendie may be plugged with pus, or the veins of Galen become thrombosed, causing hydrocephalus.

Treatment.—General principles again hold good, but the excessive pain and convulsions demand special attention—absolute rest, antipyrin, chloral, and bromide; chloroform to subdue painful contractions. (In three cases I saw—chloroform during the convulsions, iodoform to shaved head, quinine, and free purging with calomel, seemed to do good.)

MALARIAL FEVERS.

An infectious fever characterised by being associated with certain soils, and the organism known as hæmatozoa of Laveran; also clinically, by paroxysms of (1) *intermittent fever* (true ague); (2) *continued* fever with well-marked *remissions* (jungle fever); (3) certain pernicious, rapidly fatal forms; and (4) a chronic cachexia with anæmia and enlarged spleen.

The Malarial Poison.

1. The Specific Germ.—Klebs' discovery of a bacillus is not yet confirmed (1893). If the blood of the patient be carefully

examined, there are always found colonies of bodies termed hæmatozoa. These are regarded as parasites, but nothing is known of their life history. They exist in six forms—all are found in the red corpuscles :—

(1) Unpigmented, active, hyaline bodies.
(2) Pigmented amœboid bodies.
(3) Segmented bodies.
(4) *Crescentic masses*, very distinctive.
(5) Flagellate organisms from the crescentic masses.
(6) Free flagella.

These parasites break up the hæmoglobin, thus causing excessive anæmia on the one hand, and give rise to products which probably are albumose in nature, and thus poison the nerve centres. Quinine has been observed experimentally to interfere greatly with the existence of these organisms.

2. The Soil.—The poison usually develops in low, marshy, or badly drained districts, rich in vegetable matter. A tropical climate is best for its production. The poison is contained in the emanations of the soil. It does not rise high, is modified by trees, especially the eucalyptus; but the beneficial effect produced by this tree is probably due more to its *drying influence* than to its antiseptic qualities.

The Attack.—After some days of premonitory symptoms of more or less general malaise, the typical attack comes on, and usually consists of three stages—cold, hot, and sweating stages.

Cold Stage.—Patient shivers violently, teeth chatter, skin is pale and blue, and papillæ are raised (goose-skin); there is a great tendency to collapse. The temperature though much lowered externally, is, however, raised in the rectum. As may be expected with such an extreme contraction of the superficial capillaries, the urine is pale, copious, and of low specific gravity.

This stage lasts from one-half to two hours. Towards the end of the stage the temperature may be 103° to 106°.

Hot Stage. — It may be gradual or sudden in its onset. The skin becomes hot and burning, and a patchy rash may form. There is vomiting or complete anorexia; throbbing of the carotids with intense headache, and there may be delirium. Often crops of herpes form at the mouth. This stage lasts from two to four hours. Urine is scanty, of high specific gravity, and contains a large quantity of *urates and urea.*

Sweating Stage.—The sweating commences at the roots of the hair, but soon becomes general and profuse. The pulse gets softer, the temperature falls gradually to the normal, and the patient is restored to the normal condition. During the sweating stage the urine is of high density, and scant in quantity; *urates* are more abundant than urea.

The Spleen.—The spleen enlarges during hot and cold stages, and repeated attacks finally induce the chronic, enlarged *spleen or ague-cake.* The organ is enlarged, the capsule is thickened and is often adherent to other organs. Internally it shows—

1. Fibrous stroma increased.
2. Infarcts.
3. Extravasations of blood.
4. Waxy degenerated patches.

The liver is often enlarged and blood-stained.

Malarial Cachexia is characterised by—

1. Anæmia, often intense, with a peculiar jaundiced or parchment look.
2. Pains in the joints and great lassitude.
3. Fœtid breath and anorexia.
4. Hæmorrhages. Pathologically, the typical "ague-cake" spleen, and pigment granules are to be found in the blood.

Varieties of Ague.

Quotidian = an attack every day.
Tertian = one day of health intervening.
Quartan = two days of health intervening between the attacks.

There are also compounds of these. When a next attack comes earlier than expected, it is said to be anticipated; and conversely, if the attack be delayed, it is said to be postponed.

The Pernicious Forms.—Sometimes an ague attack assumes a very virulent type, and death takes place rapidly from collapse, perforation of the gut, or a rupture of the enormously swollen spleen. The best recognised forms are—

1. Comatose form.
2. Choleræc or Algide.
3. Dysenteric.

REMITTENT AGUE.

(Jungle Fever.)

Differs from true Ague by having—

1. A badly marked cold stage.
2. A *well* marked hot stage.
3. A short sweating stage, and no complete intermission of fever.

The **Symptoms** are principally—

1. Chilliness (but not actually cold).
2. Intense headache and elevation of temperature.
3. Vomiting, first of stomach contents, then bile, and finally blood (black vomit).
4. Delirium often marked.

Symptoms usually abate somewhat in the morning, only to be followed by an exacerbation in the evening.

There is a great tendency to intense jaundice and the typhoid state.

Duration of attack—about twelve to fifteen days.

Treatment of *Malarial Fevers.*—During the attack, quinine with hydrobromic acid, antipyrin, jaborandi, etc. If collapse threatens, stimulants will be required. In the interval—

1. Removal from the malarial district.
2. Arsenic and quinine.
3. Careful diet and hygiene.
4. Hepatic purges.

Treat complications as they arise. Iron may be tried or Easton's syrup.

RELAPSING FEVER.

A specific contagious disease, occuring in epidemics, and characterised by terminating suddenly on the sixth or seventh day, but *followed by a relapse after an interval of a week.* The fever is always associated with specific organisms in the blood.

Ætiology.—Epidemics have a close connection with overcrowding and destitution. Like typhus, it occurs amongst the poor and filthy, but is more associated with *poverty and famine* than filth. It attacks all ages, but males more than females.

The Organism. — The spirillum or spirochæta consists of a coiled thread, varying from $\frac{1}{500}$ to $\frac{1}{1500}$ of an inch in length. It is in constant movement of a lashing character. The spirilla tend to adhere around the red corpuscles. They are absent during the non-febrile interval, but return again when the relapse occurs. Just as they disappear before the crisis, small glistening masses appear in the blood. It is not known yet whether these are spores or not.

Symptoms.—The fever is ushered in suddenly : rigors, frontal headache, backache, and rapid elevation of temperature which may reach, even on the first day, 104°. The pulse is very rapid, and respirations are also quickened. The tongue has a marked white fur, and there is much thirst. About the fifth night the symptoms become greatly aggravated and the temperature may reach 107° or 108° ; the delirium is increased, and a fatal termination seems imminent, when a profuse sweating takes place, the bad symptoms rapidly abate, and the crisis has taken place. In a few hours the patient feels comparatively well ; he is ravenously hungry, and has apparently fully recovered. Then in about a week, he is seized with similar symptoms as in the first attack—usually the second attack, however, *runs a less severe course, and shorter duration than the first attack,* but sometimes the exhaustion is so profound, that the "typhoid" condition rapidly supervenes, and a fatal issue results. Two, or even more, relapses have occurred. Convalesence is usually slow. It is much less fatal than typhus.

Complications.—

Pleurisy and Pneumonia.
Critical Diarrhœas.
Severe Jaundice.
Ophthalmia.
Hæmorrhages.

Special Points to note—

1. The Spirochæta.
2. Absence of Rash. (There may be petechial spots.)
3. The speedy, *apparent* recovery after the first attack.
4. The slow, *real* convalescence after a relapse.
5. Association of the fever with famine.
6. The Jaundice is not of the so-called obstructive kind.

Morbid Anatomy.—Beyond the presence of the organisms, nothing special is seen in the tissues. The spleen is, however, much distended, and presents numerous infarcts.

Treatment.—General principles.—Avoid lowering treatment. Quinine has no specific effect. During convalescence—generous diet, and fresh air.

DYSENTERY.

A contagious disease, characterised clinically by a peculiar diarrhœa, tenesmus, tormina, and cachexia. Pathologically, by an inflammation of the lower part of the small gut and large intestines, resulting in extensive ulceration and sloughing. The disease is always associated with amœbic organisms.

Ætiology. — Endemic and epidemic in tropical climates, where the epidemics are sometimes extremely fatal. In the temperate regions the epidemics are less severe, and sporadic cases are more common. Battlefields, dried up river beds, decayed vegetation, and dung heaps, form suitable soil for the poison, and a fitting nidus within the body is sometimes furnished by the ingestion of unripe fruit.

The contagion probably spreads through the stools, as in typhoid.

The Specific Germ. — Amœba is a unicellular organism, mobile, and shows—

1. Clear outer zone = ectosarc.
2. Granular inner zone = endosarc, containing vacuoles and nuclei.

Kartulis found them in the stools, intestines, and in the liver abscesses of dysenteric patients.

Symptoms of acute dysentery vary with the type, usually there is first abdominal pain, with general malaise and anorexia. Soon the characteristic diarrhœa sets in.

Each discharge is *scanty*, and may not be more than two drachms; the number of evacuations may be great; there is great straining (tenesmus), and severe griping pain around the navel (tormina).

The stools show—

1. Yellowish transparent mucus.
2. Blood.
3. Pus and epithelial debris.
4. Actual sloughed mucous membrane.
5. Triple phosphates and chloride of sodium in excess.

The general appearance is described as "boiled sago" or toad's "spawn."

The patient loses strength, and there is moderate fever, great thirst, dirty tongue, dizziness, and dry skin. *In the graver forms* the evacuations are not so numerous, but the general condition rapidly assumes a low or typhoid state, and a fatal result is only too often brought about. On *post-mortem* examination the mucous membrane is seen as a black, rotten, friable charred mass. (Rokitansky.)

Varieties—

1. Acute—
   (1) Catarrhal.
   (2) Amœbic.
   (3) Diphtheritic, divided into primary and secondary.
2. Chronic.

The **Morbid Anatomy**, obviously, will vary with the type of disease; but after all, the essential *lesions differ only in degree.* As a typical example, take—

1. *Catarrhal Form.* Site—lower part of ileum and large intestine, here we have to do with a severe inflammation of a particular mucous membrane; therefore we get—

   (1) Hyperæmia.
   (2) Exudation rapidly becoming purulent.
   (3) *Necrosis of exposed follicles.*
   (4) *Separation of sloughs and formation of dysenteric ulcer.*

Put into other words, the inflammatory process is very severe, and only differs from inflammation of any other mucous membrane, *because of the distinctive characters possessed by the gut involved.*

2. *Amœbic Variety. — Ulceration is more marked.* The whole of the large gut may be riddled with ulcers, and abscess of the liver much more frequently results.

3. *Diphtheritic Type.*—In the *Primary form* the "necrosis" is extremely rapid, and the "typhoid" state replaces the usual symptoms.

The Secondary type is peculiar as being associated with pneumonia, chronic Bright's disease, or cardiac disease; there is *more of a true diphtheritic membrane formed* than in the other forms; or putting it simply, *the croupous inflammation is associated with disease elsewhere.*

Dysenteric Ulcer—

1. Irregular undermined edges.
2. Base may be formed of any of the coats.
3. If they heal they cause much contraction.
4. Usually seen best marked on ridges of large intestine.

Complications.—As the blood from the intestines is returned by way of the liver, we naturally may expect trouble from products so carried, and usually we get—

1. Single or tropical abscess of liver, or (much less common)
2. Multiple pyæmic abscesses. Other complications are—
   (1) Hæmorrhages and perforation.
   (2) Peritonitis or pneumonia, etc.

Chronic Dysentery may be subacute from the beginning, or a result of an acute attack through persistent suppuration of the submucous abscesses.

Pathology.—

1. There are healing and healed ulcers.

2. The deep coats of the gut are much thickened and contracted, and sinuses are common.

3. The calibre of the bowel is much diminished.

Symptoms.—Slightly bloody, painful diarrhœa; the appetite is absent; tongue glazed and red; intense anæmia; jaundice; and patient has the shrunken look of malignant cachexia. The emaciation may be extreme, *but the spleen is not usually enlarged.*

Treatment.—

Acute.—Hygienic; careful dieting, or as Gull puts it, rest, warmth, and ipecac. Mag. sulph. largely diluted; minute doses of perchloride of mercury. Ipecac. gr. 20, followed by opium and bismuth. Try salol and opium if ipecac. fails.

Chronic.—Careful diet; plenty of fresh air; salol, quinine, bismuth, and Dover's powder; injections of silver nitrate, 20 grs. to 20 oz. of water; inject four pints. Arsenic and hypophosphates.

CHOLERA.

A specific infectious disease, occurring in epidemic form, and characterised by violent purging, pain, cramps in the legs, and intense collapse.

The mouth of the Ganges is claimed as the home of cholera, but epidemics have occurred in all parts of the world.

Ætiology.—Contagion is conveyed by stools and contaminated water. It breaks out principally in summer and autumn, and attacks all ages and both sexes alike.

The Germ is probably a spirochæta, known as the comma bacillus of Koch. It is about half the size of the tubercle bacillus and is curved, but may be spiral or shaped like an S. It is found in the dejecta and intestines of patients affected.

It grows in many media, but requires either a neutral or a slightly alkaline soil to flourish. It lives in fresh water, but does not multiply; multiplies rapidly in water containing dead vegetation.

Klein says the comma bacillus is found in abundance under other conditions than cholera.

Pettenkofer holds that "germs" develop in the subsoil during summer, and rise into the air as a "miasm."

Pathology.—That of a severe inflammation of the mucous membrane of the small intestines, the process being specially marked around Peyer's patches and solitary glands; usually there is no ulceration.

The blood is dark and thick, sometimes almost tarry through the drain of water from the system. The other organs show changes similar to those occurring in other virulent fevers. Note, however, that there *is rapid rigor mortis*, and sometimes *post-mortem rise of temperature.*

Symptoms.—After a short incubative period, a preliminary diarrhœa sets in, with more or less headache, vertigo, and nausea; then the characteristic diarrhœa commences. Most clinicians divide the disease into three stages—

1. Evacuative.
2. Algide or collapse.
3. Reaction.

Evacuative Stage. — Violent diarrhœa accompanied with intense pain, and cramps in calves of legs. The stools are

"rice water" in type, neutral or alkaline from ammonia. Specific gravity 1006 to 1012; show on standing—

1. Epithelial debris.
2. Threads of algæ.
3. Bacteria.
4. NaCl and triple phosphates.
5. Blood pigment and albumen.

Sometimes the evacuations which are extremely copious are quite painless. Then vomiting takes place, first of food, but finally matter similar to the stools; then the second, or

Algide Stage commences. The collapse becomes extreme; the features are shrunken, livid, or ashy grey; eyeballs sunk in; the skin is shrivelled and wrinkled, and covered with a cold, clammy sweat. Though the surface temperature is subnormal, the temperature rises in the rectum to 102° or more. The voice is husky, and the pulse small and flickering. The purging usually ceases, but the vomiting continues. There may be complete suppression of urine, coma, and death within a few hours; or patient passes into the third, or

Reaction Stage.—The temperature gradually rises, and a red glow replaces the ashy appearance. Erythema and urticaria are frequent. The patient gradually recovers, or the improvement is checked by the onset of—

1. Pneumonia.
2. Recurrence of severe diarrhœa.
3. Uræmia, coma, and death.

Treatment. — Do not disturb the stomach; remember its irritable condition and poor absorptive powers. Administer ice, brandy in small quantities, hypodermic injections of morphia, and apply hot bottles, etc., to feet and legs. Astringents are of little use. Starch and opium, or salol and enemata are useful.

The older writers advised calomel and opium; as a last resort try intra-venous injection of warm saline solution.

Prophylaxis.—The utmost sanitary vigilance, isolation, and quarantine; all soiled linen, etc., should be burnt; drinking water to be first boiled, and avoidance of all irritating food.

Protection by inoculation of cultivated virus, suggested and practised by Ferran, reports are too conflicting to be of value.

VARICELLA or CHICKEN-POX.

An eruptive fever, occurring principally amongst children, and characterised by a vesicular rash appearing on the first day.

Ætiology.—Epidemics are very common, and the disease once it starts usually spreads with great rapidity. Sporadic cases, however, are common. Nothing is known of the circumstances under which the fever arises.

The Rash consists of small raised vesicles, containing either transparent or turbid fluid—appearing first on the neck and chest, but quickly spreads over the entire body.

It differs from Small-Pox in—

1. Not being umbilicated or loculated.
2. Having no inflammatory areola around the vesicles.
3. Appearing on the first instead of the third day.
4. Being vesicular from the beginning.
5. The vesicles usually drying up as brownish scabs on the fourth day, leaving no scarring or pitting.

Symptoms are not severe. There is usually slight fever and fretfulness, with a very furred tongue and often vomiting.

Complications—

1. *Severe itching*, causing the child to scratch, producing deep scars, or even ulceration.
2. Gangrene in debilitated children around vesicles. (I have often seen this in children with congenital syphilis.)
3. Infantile paralysis (rare).

Treatment.—A gentle saline purge, careful dieting, and where there is much itching, soothing lotions on lint—morphine and lead.

MUMPS.

An infectious disease, characterised by inflammation of the parotid gland, with a tendency to metastatic inflammation of testes in males, or breasts in females.

Ætiology.—Is frequently contagious, especially in schools, and may be curiously localised in one district. Nothing is known of the virus, though it is highly probable that it is due to micro-organisms. Children from four to twelve years of age are most often attacked—others say the adolescent period is the favourite age.

Symptoms.—After a period of incubation, pain is felt under one ear, with stiffness or soreness of neck and jaw. The swelling first appears in the hollow between the angle of the jaw and the mastoid process, gradually extends, and may involve the sub-maxillary and sub-lingual glands. In about two days the *other side* undergoes the same changes—both swellings form a "collar," giving the child a ludicrous appearance. Deglutition and mastication are often very painful, the breath is foul, and tongue very furred. In about nine days the swelling resolves, and rapid improvement takes place. The glands seldom suppurate.

Complications are—

1. Orchitis (as the gland affection subsides).
2. Vulvo vaginitis in girls.
3. Meningitis (not common).

Sequelæ.—After severe cases the following have happened—

1. Permanent deafness.
2. Purulent Arthritis.

Treatment.—A simple saline purge, hot fomentations, and antiseptic wash for the mouth is all that is usually required. Treat complications as they arise.

WHOOPING-COUGH.

A specific contagious disease, affecting the respiratory organs, and attended with a peculiar paroxysmal cough and whoop.

Ætiology.—One attack procures future immunity. It is highly contagious from person to person; one single case will frequently cause the infection of a *whole* village or town. There can be little doubt about it being due to an organism, but its true nature has not yet been determined.

The Russian physicians claim to have discovered in the sputum of infected patients a short bacillus, which if cultivated and inoculated produces bronchial catarrh, but this, like the discovery of many other bacilli, requires confirmation.

Symptoms.—The disease is usually divided into three stages—

1. Invasion.
2. Paroxysmal.
3. Period of decline.

1. *Stage of Invasion.*—The symptoms are merely those of bronchial catarrh with coryza, and last about seven days or so.

2. *Paroxysmal or Whooping Stage.*—The paroxysm consists of a series of short coughs or expiratory puffs, with no intervening inspiration till about fifteen or more expulsive efforts have been made in about seven seconds. Then occurs a deep, prolonged inspiration, attended with the characteristic *whoop;* a second bout of short coughs succeeds with another whoop, and after three or four such sequences, a little plug of mucus is expelled, or more frequently vomiting takes place, and the child is all right until the next paroxysm.

During the severe cough the patient is perfectly helpless, and when paroxysms are very violent we may get—

1. Hæmorrhages from the nose, frænum of the tongue, sub-conjunctivæ, or even in the brain.
2. Collapse of the lung.
3. Convulsions in infants.
4. Fatal asphyxia (rare).

The appearance of the child soon becomes puffy or bloated. The whoop is due to partial closure of the glottis. The closure is considered by some to be "reflex spasm" in nature. Others say it is a mere passive approximation of the glottis. There may be as many as forty attacks in the twenty-four hours, but they range from four upwards; this stage lasts three to six weeks. Sometimes the "whoop" is delayed in its appearance; difficulty will be then experienced in diagnosis, and no general law can be laid down. Niemeyer says, "If a child has a violent *prolonged cough* attended with *vomiting*, suspect and treat as if it were whooping-cough." I am certain this is sound advice, borne out by experience of two epidemics I saw in Monmouthshire. Taylor says, "vomiting is not frequent." In the epidemics I witnessed, the bulk of the cases were attended with vomiting after each paroxysm. Osler states similarly.

3. *Period of Decline.*—This stage is marked by a gradual decrease in the number of paroxysms and a slow convalescence, usually lasting three months or longer.

Complications. — Besides those mentioned which might occur during paroxysm there might be—

1. Extensive capillary bronchitis, or
2. Broncho-pneumonia.
3. Cardiac strain ending in valvular disease.

Treatment. — Prompt isolation, a simple saline with paregoric. When the whoop develops we must try anti-spasmodics; bromides, belladonna, chloral, and hydrocyanic acid; others advise in addition—quinine, emetics, swabbing the throat with a 2 per cent. solution of resorcin, etc.

If recovery be tedious, *change of air*, cod liver oil, and Easton's syrup.

Special points to be noticed—

1. In *infants*, tendency to convulsions.
2. *Older children*, capillary bronchitis, causing—
   (1) Collapse of Lung.
   (2) Deformity of chest (pigeon shape).

YELLOW FEVER.

A contagious fever occurring in certain tropical districts, and characterised by two well-defined stages. It is at first endemic, then becomes contagious, and intensely malignant in its ravages.

Ætiology.—It originates in crowded cities, on the shores of large rivers, in ships, etc. Strangers landing in an infected district are nearly always attacked. The nature of the poison is supposed to be a miasm from decaying vegetable matter; but from its close resemblance to *remittent* ague it is more likely to be due to a special organism developed in such districts.

Symptoms—

The First Stage.—After a "*primary*" depression or sense of coldness, rigors occur, accompanied with elevation of temperature, which ranges even on the first day from 101° to 110°. The pulse is quick, full, and bounding, but a fact of great value is—*the pulse tends to decrease when the temperature is high, and may even become below the normal standard.* Intense headache and vomiting of blood-stained mucus. The tongue is at first red at the tip, and pointed, but thickly furred behind.

The *Second Stage* is marked by the advent of the typhoid state (see page 3), but with the following prominent symptoms in addition—

1. Black vomiting—*i.e.*, bile and blood.
2. Intense jaundice.
3. Excessive albuminuria,
4. Suppression of urine, uræmia, and only too often speedy death.

Pathology.—The blood shows marked changes.—

1. Marked diminution of fibrin.
2. Red corpuscles, crenated, and diminished in number.
3. Increase of fatty matter, and antecedents of urea in the blood.

After death the blood rapidly becomes acid.

The liver is extensively affected, deep golden yellow in colour. It is at first firmer than ordinary fatty degenerated tissue, but gradually approaches a condition similar to acute yellow atrophy. There is no *formed* bile, but plenty of bile pigment, also glycogen, but *no grape sugar.* Leucin and tyrosin are sometimes present.

The other organs show the same changes as other acute, malignant diseases.

Treatment—

1. Absolute rest in recumbent posture.
2. *Free Ventilation*, and light diet.
3. Antipyrin, quinine, ice, blood-letting, calomel.

Remember opium, iron, and mineral acids are badly borne, and are sometimes actually injurious. Later, champagne is the best stimulant.

THE SEPTIC STATES.

Under this heading we mean the clinical phenomena attending the introduction of noxious material into the blood. There are three types—

1. *Sapræmia — i.e.*, the introduction of bacterial products or substances which *cannot* multiply in the living tissues; the symptoms following being in proportion to the size of dose introduced.

2. *Septicæmia*—The introduction of living organisms which have the power of *living and multiplying in the blood.* The symptoms following *bear no relation* to the amount of bacteria introduced.

3. *Pyæmia* is now usually looked upon as a sub-acute septicæmia, attended with *metastatic abscesses.* Formerly it was held to mean the symptoms consequent on the introduction of pus, and pus only; but obviously, as pus is held to contain micrococci, and micro-cocci can set up thrombosis and metastatic abscesses, it seems idle to quibble over terms.

Much confusion has arisen through the term "Septic Intoxication," being used at one time to express "Sapræmia," and at another time "Septicæmia," but either ignore such an ambiguous term or relegate it to its proper position, viz., a convenient term to express the peculiar symptoms attending the septic state.

Symptoms of Acute Sepsis — *Rigors*, rapid rise of the temperature, flushed face, delirium, and rapid exhaustion; the typhoid condition may supervene.

Of the Sub-acute forms—frequent rigors; oscillating temperature; night sweats—*i.e.*, hectic fever; rapid loss of flesh; and general exhaustion are the cardinal symptoms.

Pyæmia is characterised by running a much more chronic course. Frequently periods of comparative convalescence seem established, but are interrupted by exacerbations of hectic fever and the formation of metastatic abscesses—often complicated with pneumonia, purulent pleurisy, etc.

Mode of Abscess formation—

1. The bacteria cluster together and form by themselves an embolus, which becomes arrested in the smaller blood-vessels, causing local thrombosis, with intense irritation, and formation of pus in a circumscribed area—*i.e.*, local abscess.
2. The bacteria may cause suppurative phlebitis.
3. The intense irritation leads to ordinary thrombosis, but the bacteria enter the clot, causing central necrosis and breaking down of the whole into a greenish purulent mass.

Treatment.—Sapræmia is generally due to absorption of products of putrefaction, such as decomposed placental remains, blood clots, etc.; or absorption of the microscopic layer of dead tissues killed in operations, even under antiseptic precautions. Obviously the treatment will be to stop the *local manufacture* of the poison by careful cleansing and rest; remember also a septic condition may begin as a sapræmia, and end as a true septicæmia, through the local condition of the first state acting as a favourable nidus for the production of the second condition.

Septicæmia, due to the introduction of *living* organisms, therefore, all we can do is to—

1. Stop the entrance of *more* organisms by using the utmost antiseptic precautions.
2. Support our patient, till the fight between bacteria and tissues is ended, by stimulants and *quinine*.

Antiseptics internally are advised by some, but it is very doubtful if we can kill those organisms already in the blood; probably, some substance will be discovered which will render the infected blood an unsuitable soil.

Pyæmia, as regards treatment, requires the same as advised under Septicæmia, but special care must be taken in opening abscesses *not* to *convert a sub-acute process into an acute one*, by causing putrefaction of the pus.

SYPHILIS.

By syphilis is now understood a *constitutional* affection, resulting from (1) impure venereal intercourse; (2) inoculation with the virus obtained from syphilitic lesions; (3) or transmission through diseased parent or parents — *i.e.*, congenital syphilis. Syphilis is such an extensive subject, and so highly important a factor in many diseases, that the reader is referred to a monograph on the subject.

Briefly put, the following are the more important facts of acquired syphilis—four periods are described—(1) Incubation; (2) Primary Sore; (3) Secondary Symptoms; and (4) Tertiary Stage.

1. *Incubation Period* = 6 weeks.

2. *Primary Sore*, or chancre, is a papule with an *indurated* base. The surface may or may not ulcerate, but is usually

depressed in the centre. The hardness or induration extends to the nearest lymphatic glands, and ultimately all the lymphatic glands may become involved. The chancre or "*hard sore*" finally disappears, leaving a cicatrix behind.

3. *Secondary Symptoms*—divided into two stages.

FIRST STAGE—

(1) Syphilitic fever, with transient hyperæmia of the skin, roseola, etc.

(2) Sore throat.

SECOND STAGE—

The second stage is marked by skin eruption, clinically distinguished by—

(1) Their symmetry.

(2) Copper colour, dull red at first, afterwards a reddish brown.

(3) A tendency to circular form of patches.

(4) The scales when present are light and small.

(5) The crusts are greenish and thick, adhering firmly.

(6) Ulceration is common—sharp edges and ashy-grey surfaces.

(7) The eruptions are often mixed, or poly-morphous. (Tilbury Fox.)

The other changes are—

Mucous tubercles or condylomata on rectum, mouth, etc.

Ulceration of throat.

Syphilitic Iritis.

Syphilitic Periostitis.

Anæmia.

Loss of hair—*not* followed usually by permanent baldness.

4. *Third or Tertiary Stage.*—The anæmia has become more marked, and the manifestations of the disease are those of serious malnutrition, with deep-seated organic changes due to the formation of peculiar growths termed "gummata." According to some, any chronic disease may be caused directly or indirectly by syphilis. Whilst no doubt syphilis is often a most powerful or even the chief factor in many diseases, and its importance cannot be over-estimated, still, probably, syphilis is often unjustly held responsible for many serious lesions.

Structure of Gummata.—They are at first hard, firm, nodular masses, fairly defined but *adherent to surrounding tissue.* Later, they become somewhat softer, and show from within outwards—

1. A central mass of fatty degenerated cells, sometimes granular, but may be homogeneous and of gum-like consistence.
2. A fibrillated structure, with leucocytes in the meshwork.
3. A more fibrous layer, with young epithelial cells.

They are met with in the brain (Jacksonian epilepsy), bones, testicle, and liver (syphilitic cirrhosis), etc., etc. The change *begins around* the small arteries.

Syphilitic virus is peculiar in—

1. Producing changes in the lumen of vessels apart from any external irritation, causing them to become narrowed—*i.e.*, arteries, larynx, intestine, etc.
2. In causing waxy disease without intervention of suppuration.

Tertiary syphilis is neither inocuable or transmissible to offspring.

Congenital syphilis may take place—(1) from father; (2) from mother; (3) from both. If the father alone be syphilitic, then the sequence may be—

1. Father.
2. Child.
3. Mother through child.

Symptoms—

1. The child is born with "snuffles."
2. Develops a peculiar "old man" appearance.
3. Mucous tubercles at the mouth or anus.
4. Red patches on buttocks, ankles, or hands.
5. Sub-acute onychia, fissures at lips, and later, notched "Hutchinson" teeth.

Specific Germ undoubtedly due to a special organism not yet cultivated successfully.

Treatment.—We have a choice of two great schools—

1. The mercurialists.
2. The anti-mercurialists.

The former say mercury cures, while the latter say mercury is not indicated, and even produces the worst features seen in syphilitic cases. Probably the truth lies between them. Let us review our data, and let each reader form his own opinion.

1. Syphilis is undoubtedly infectious, and, therefore, seems due to—

(1) A living organism capable of multiplying within the body; therefore

(2) The severity of the disease must vary with the soil; and

(3) From analogy with other bacterial diseases, syphilis must *tend* to cure itself.

2. It is extremely doubtful if we can kill the organism *directly*, but we can make the soil more resistant by *careful hygiene, diet, fresh air, and tonics.*

So much for the disease, now for the case of mercury.

The most sceptical must admit that in the skin eruptions and congenital syphilis a marked improvement takes place rapidly under the influence of any mercurial. *Mercury then, strongly influences demonstrable syphilitic affections.* Take the action of mercury in other diseases. We find it used in

general routine practice, in hepatic diseases, and it is a favourite drug in the treatment of the most simple ailments of children. It is also used to remove inflammatory products of inflamed serous membranes. In all these cases it is used in *small doses* with the best results. Then why deny the use of it in syphilis? However, men working amongst quicksilver become emaciated, their teeth are loosened, deep ulcerations form, peripheral neuritis or mercurial tremors supervene, and the man becomes a decayed wreck.

Here then are the *untoward effects of large doses of mercury.* Evidently the treatment of syphilis should consist of—

1. Small doses of mercury, alternating with
2. Tonics, fresh air, and careful diet,—these should be kept up for two years or more.

A good plan of treatment is the following:—Small doses, say two grains of pil. hydrarg. nightly for three months, then Easton's syrup for three months; and this alternation of mercury and tonic repeated for twelve months. Then an occasional *course* of—

℞ Pot. Iodid. . . . ℥i.
Liq. Hydrarg. Perch. . ℥i.
Dec. Cinchon Co. ad. . ℥viij.
Fiat mist., ℥ss ter in die.

No one is justified in *pushing* mercury into a debilitated constitution

TUBERCULOSIS.

A constitutional disease, produced by the pernicious action of the bacillus tuberculosa. There may be special foci, and when the disease is principally localised it receives definite names according to the part affected, viz.—

Lungs = Consumption—pulmonary phthisis.
Bones = Rarefying Caries.
Joints = White Swelling.

The Bacillus is small ($\frac{1}{3}$ size of a red corpuscle), motion less, and contains spores. The spores are more resistant to change than the mature bacillus, and requires temperature of body (98·4°) to multiply; sterilized serum jelly is the best medium for cultivation.

How to Stain—

1. Spread a portion of purulent sputum on a cover glass.
2. Press upon this another cover glass to get a clear film.
3. Heat gently till albumen is coagulated.
4. Place specimen (sputum downwards) in Neilson's fuchsin, and heat; then
5. Wash in water. Next transfer it to
6. A solution of—

Methylblue	. .	two grs.
Acid Sulph.	. .	20 cc.
Aqua dest.	. .	80 cc.

leave it for two minutes, then

7. Wash in water, and mount in balsam.

The "tubercles" formed by the bacilli are of two types—

1. The grey.
2. The yellow.

The latter is but an advanced stage of the former.

The grey tubercles are small transparent, grey gelatinous lumps, isolated or clustered in masses.

Microscopically, they show from within outwards—

1. The bacilli.
2. Giant cells, with branched processes forming a scaffolding for the next layer.
3. A layer of small epithelioid cells.
4. A zone of leucocytes.
5. Often a dense fibrous capsule.

Later.—The central zone undergoes fatty degeneration and bacillary liquefaction, or fatty degeneration with caseation and calcification, the nodule being converted into a fibrous calcified mass.

The changes which tubercular nodules undergo are essentially the same wherever the lesion is, though of course certain tissues will modify the rapidity of such changes. Take the lung for instance.

Always describe pathologically—

1. Preparation of the soil and invasion of the bacilli.
2. Inflammatory zone around bacilli, and formation of tubercle.
3. Fibrous zone around tubercle.
4. Fatty degeneration of tubercle and formation of—
    (1) Pus.
    (2) Cavities.
5. Bacillary liquefaction ; or pus may dry up and calcify.

Tuberculosis spreads through lymphatics and blood-vessels, from local foci.

Mode of Infection.

1. From phthisical patients' breath and sputum.
2. From tuberculous cattle, dogs, cats, etc.

Is tuberculosis hereditary? This is still a vexed question. It is usually taught, however, that it is not transmissible from parent, but that the children of tubercular parents are born with a highly favourable soil or predisposition.

Symptoms of tubercular disease vary according to—

1. Site.
2. Stage.

After the breaking down process has commenced, however, the symptoms are those of hectic fever.

1. Elevation of temperature, especially in the evening.
2. Night sweats.
3. Progressive emaciation.
4. Phthisical appearance is well known.

Treatment will be considered under the head of "Phthisis."

NERVOUS DISEASES.

The reader is strongly advised to digest the following brief account of the anatomy and physiology of the nervous system, before commencing the study of the various nervous diseases. I shall not attempt anything approaching an anatomical description, but content myself with the the briefest description of those facts *which form the foundation of a clear understanding of clinical phenomena attendant* upon pathological changes.

The Spinal Cord consists of a tubular prolongation of the brain enclosed in three membranes.—

1. Dura mater, externally.
2. Arachnoid, in the middle.
3. Pia mater.

Between the arachnoid and pia mater is the sub-arachnoidal space, filled with cerebro-spinal fluid. It should be noted that this space is continuous with the ventricles of the brain. The cord substance shows *white* matter externally, and grey matter internally. Like *all nervous tissue* the white and the grey matter have as a groundwork *neuroglia*, or nerve glue.

Neuroglia shows under the microscope—

1. Large and small cells.
2. Granules.
3. Fibres.
4. Interstitial cement substance.

Note that "sclerosis" means a great increase in neuroglia at the expense of the special nerve elements.

The *grey matter* differs from the white in—

1. Colour, and being more vascular.
2. Having special groups of nerve cells arranged—

Anteriorly Laterally Posteriorly	termed	Anterior vesicular column. Lateral vesicular column. *Posterior or column of Clarke.*

The *anterior cells* are multipolar and give origin to motor nerve roots; *they also act as trophic centres for motor fibres in the nerve* trunk.

The posterior cells (or column of Clarke) are very important through being implicated in the passage of sensory fibres, as will be seen further on.

The *white matter* though apparently homogeneous, is really mapped out into columns. Each column is specially concerned in transference of certain impulses up to, or from, the brain. Much that has been written about these columns has turned out untrue, and indeed, even now (1893) very little is known of the exact functions of any one column; but patient investigation has shown that there are at least two motor or *descending* paths, and four sensory or *ascending* paths.

THE TRACTS.

(See Diagram, p. 81.)

Motor Tracts—

1. Direct Pyramidal situated anteriorly.
2. Crossed Pyramidal situated laterally.

Sensory—

1. Comma Tract of Gowers. } Laterally.
2. Direct Cerebellar. }
3. Postero-External, or column of Burdach. } Posteriorly.
4. Postero-Internal, or column of Goll. }

Cerebro-Spinal Nerves.

An ordinary mixed nerve is made up of—

1. Anterior or motor root fibres.
2. Posterior or sensory root fibres.
3. Sympathetic fibres.

The *branches* of such a nerve may contain only (1) motor fibres, or (2) sensory, or (3) principally sympathetic.

Course of Motor Fibres. (See Diagram, page 81.)

As motor fibres proceed *from* the brain, we shall describe them from *above downwards.*

Starting from the cortex, principally around the *fissure of Rolando,* they proceed through—

1. Corona radiata.
2. Anterior two-thirds of *posterior* limb of internal capsule.
3. Crusta of Crus.
4. Pons Varolii.

5. *Medulla*, where the bulk of the fibres cross (decussation of pyramids) to the opposite side, and pass down the cord as the *crossed pyramidal tract*, thence to the multipolar cells of the anterior horn of grey matter, and finally out by the *anterior motor roots*. A few fibres pass down from the medulla without decussation, and so form the direct pyramidal tract.

Note here the trophic centre for the *motor tracts* is situated in the cortex, happily termed the first trophic realm by Wyllie. The trophic centres for the *motor nerves* are, as before stated, the multipolar cells in the anterior horn of grey matter, named by Wyllie the second *trophic realm*, or putting it briefly, a lesion of the motor cortex, is followed by degeneration of *motor tracts*. A lesion of the anterior grey horn is followed by degeneration of motor *nerves*, plus other changes.

Sensory Fibres. (See Diagram, page 81.)

Formerly they were supposed to enter the cord and immediately decussate over to opposite side. Recent researches show that little is really known about sensory nerve fibres, and still less about sensory tracts. The following will form a good *working* basis, and is at least as correct as the unproved statements of large text-books.

Sensory fibres enter the grey matter in two bundles—*i.e.*, Bundle *A* and Bundle *B*.

Bundle A passes first into column of Burdach, descends and ascends a little, then enters the column of Goll, passes straight up to the medulla, and there decussates to the opposite side.

Bundle B passes into the grey matter. A few fibres go to the anterior horn to form, with the motor cells, *a complete reflex loop*. The bulk of the fibres, however, cross to the column of Clarke, and thence to direct cerebellar and comma tract of Gowers.

Thus in the cord we have four distinct sensory railroads—viz., Burdach, Goll, Gowers, and direct cerebellar as high as the

medulla oblongata. It is not known definitely how they ascend from the medulla. For clinical purposes, however, we may say that sensory fibres are scattered throughout the brain substance, though possibly the larger number proceed as follows—

1. Direct cerebellar goes direct to cerebellum.
2. Gowers to the formatio reticularis.
3 & 4. Goll and Burdach—end as clavi in the medulla.

From each clavus fibres pass to the formatio reticularis. Thus Goll, Burdach, and Gower are amalgamated at a common junction. From this junction they pass upwards (behind the motor fibres) as *sensory fibres*, strengthened by co-ordinating fibres from the cerebellum, their course being through—

1. Medulla.
2. Pons.
3. Tegmentum of crus.
4. Posterior third, *posterior* limb of internal capsule.
5. Corona radiata.
6. Cortex, especially occipital lobe.

(A glance at the diagram will make this intelligible.) Some of the fibres stop, *en route*, at the corpora quadrigemina and optic thalamus.

WHAT IS THE INTERNAL CAPSULE?

It is the band of white matter between the basal ganglia, its boundaries being—

Externally = Lenticular nucleus.

Internally = { Caudate nucleus (anteriorly).
Optic thalamus (posteriorly).

In the middle of the internal capsule is a "bend" termed the "genu" or knee; the portion in front of the knee is

called the anterior limb; the portion behind, the posterior limb. The former contains various commissural fibres of various kinds, whilst the "*posterior*" limb contains, as we have already seen, the *sensory and motor fibres* from the spinal cord.

A lesion of the internal capsule involving the motor fibres must give motor paralysis of side of face and limbs of the opposite side of the body.

Sensation is not usually much affected as it will be readily seen that a lesion, extensive enough to cause both *sensory and motor* paralysis, would probably be fatal.

THE EXTERNAL CAPSULE.

The external capsule, as its name implies, is outside the internal capsule, and is that portion of the cerebrum lying between the—

Claustrum, externally;
Lenticular nucleus, internally.

It is in close proximity to the Island of Reil.

REFLEXES.

The student is frequently puzzled as to when reflexes should be lost, exaggerated, or impaired, but if he bears the following facts in mind there should be no difficulty.

A reflex act requires a continuity between an *afferent* nerve, and an *efferent* nerve. The parts usually involved are—

1. A receiving surface (as skin).
2. A sensory nerve (afferent fibre).
3. A receiving station (cells in cord).
4. A motor nerve (efferent fibre).

These together constitute *a reflex loop.* A break in *any* part of this loop must be attended with loss of reflex. What is the effect of the brain on reflexes? The *brain* exercises an inhibitive action; therefore, if the cord be cut off from its inhibitive influence, reflexes must be exaggerated.

Imagine the cord to be built up of segments, each segment giving off a pair of spinal nerves; then take a transverse lesion of the cord, what state of reflexes would we get?

1. At point of lesion—reflexes are lost (as the reflex loop is destroyed).

2. *Below* lesion—reflexes are exaggerated (as inhibitive action of brain is cut off).

3. *Above* the lesion—reflexes are normal.

Note, however, at the upper border of the lesion, the dead part would act as an irritant to the healthy portion of the cord, and consequently cause irritation of any sensory nerves coming off that area, causing a band of hyperæsthesia.

This painful zone, taken in conjunction with the condition of reflexes, often enables an accurate localisation of the lesion to be made.

The hyperæsthetic zone also explains the girdle pain present in many lesions of the cord.

DEGENERATIONS.

When nerve fibres are cut off from their trophic centres, they undergo disintegration, known as secondary degeneration —viz., in the case of sensory fibres = ascending degeneration, and in the case of motor fibres = descending degeneration.

R. D., or reaction of degeneration, is the common name in clinical medicine for such degenerations. Tested by electricity, the changes briefly are (on applying the electrodes to the *muscle*)—

1. Increased excitability to galvanic *and* faradic currents; this quickly passes off, and we get
2. *Decreased* to faradic, *but increased to galvanic;* next,
3. Decreased to *both* galvanic and faradic.
4. Lost to both.

These peculiar changes are accompanied by "polar" alterations.

In health we get in response to a minimum current—

1. K.C.C. = kathodal closing contraction is strongest; next,
2. A.O.C. = *anodal opening* contraction } equal.
3. A.C.C. = anodal closure contraction }
4. K.O.C. = kathodal opening contraction, the weakest.

But in disease—

A. O. C or anodal opening contraction may be stronger than the *K.C.C.* (kathodal closing contraction)—*i.e.*, a reversed condition to that seen in health. The main point, however, to remember is, "that a *muscle* cut off from its nerve, or supplied by a *diseased* nerve, undergoes—

1. Increased excitability.
2. Decreased excitability, and finally fails to respond to any electrical stimulus."

Pathology of R. D.—The nerve structure shows—

1. Breaking up of white substance of Schwann.
2. Nuclei, swollen and granular.
3. Axes cylinders broken, and nerve substance transformed into fibrous cords.

The Muscles structure shows—

1. Muscular fibres atrophied.
2. Disappearance of transverse striæ.
3. Granules.
4. Great increase of fibrous tissue.

HOW TO LEARN NERVOUS DISEASES.

Having mastered the foregoing brief but fundamental points, next consult diagrams (page 81), or better still, draw them for yourself; then mark with a blue pencil the sites of various lesions—you will then note what tracts, cells, or fibres are interrupted; and lastly, reason what *must happen* in any given lesion. For instance, in diagram (page 81), I have shown what happens in three typical lesions, affecting the anterior horn, lateral columns, and posterior horn and columns respectively. The advantages of such a method are obvious, for having mastered three typical sites, *all the others will be only mixtures or overflows* of one or more of these lesions, the symptoms of course varying according to the portions of cord attacked. We have already learnt how reflexes are altered, how atrophy begins, and why paralysis occurs; just one or two more points, then I think the reader will find no difficulty in mastering the essential features of the more common nerve diseases.

SPASM AND RIGIDITY.

When a motor nerve is irritated, the muscles supplied by that nerve contract. If the irritation be kept up, the contractions are more or less constant, and cause "spasm." Consequently, when the motor tracts are injured, let us say by a severe hæmorrhage in the brain, there is not only paralysis, but also "rigidity" of the limbs quickly supervening, *due to irritation of the motor fibres;* this is termed "early rigidity," and soon passes off.

Suppose the patient recovers from the fit of apoplexy, with, however, a *resulting hemiplegia*, it will be noted that later a second rigidity takes place. This is also due to irritation, but of a *destroying nature—i.e.*, secondary degeneration.

"Spastic paralysis" is an excellent example of irritative rigidity.

Irritation, then, gives rise to increased nerve action; and *compression*, on the other hand, causes paralysis.

CONVULSIONS.

Convulsions are of two kinds—

Tonic, when the muscular contractions are continuous.
Clonic, when relaxations alternate with contractions.

Convulsions may be the result of either direct irritation, or loss of inhibitive power, plus a hyper-excited condition of nerve cells (epilepsy).

CO-ORDINATION AND INCO-ORDINATION.

By *co-ordination* is meant the harmonious action of muscles involved in the carrying out of complicated movements.

Inco-ordination means failure of this harmony.

The cerebellum is said to be the *chief* centre for co-ordinated movements. Whether this be true or not, the cerebellum is an important centre, though there are subsidiary centres. The centres seem to act through afferent impressions derived from—

1. The sense of touch (columns of Goll and Burdach).
2. Sight (optic nerves).
3. Auditory organs (semi-circular canals).

Lesions interfering with these afferent fibres are attended with more or less inco-ordination. Inco-ordination may, therefore, be due to—

1. Disease of the *centres* rendering them powerless to emit the necessary influence.
2. Disease cutting off the means (afferent fibres) by which the centres are stimulated.

DEEP (SO-CALLED) REFLEXES.

Knee Jerk.—Present in health, and obtained by tapping the patella tendon.

It is not a true reflex, but depends on tone of the extensor muscles. As the muscles waste, however, if the nerves be diseased, so will the knee jerk disappear, as the reflex loop is then broken. But it is possible for the knee jerk to be lost *without* any break of the reflex loop, or *exaggerated* when the nerves are healthy if the muscles are fatigued.

Ankle Clonus.—Not present in health — means increased excitability of muscles, prior to the advanced pathological degeneration.

SCLEROSIS.

Sclerosis is a convenient term used to express the pathological changes which take place in *chronic nervous diseases.* We shall draw a picture of a typical "sclerosis," so as to avoid a constant repetition of words. Then in describing each particular disease, merely point out the *sites* of such lesions.

Sclerosis.—*Grey Matter shows*—

1. Increased fibrous tissue (neuroglia).
2. Atrophy of *proper nerve cells.*
3. *Blood-vessels thickened*, some obliterated.
4. Extravasations of pigment.
5. Small patches of fatty degeneration.

White Matter shows—

1. Discolouration.
2. Absence of Myelin.

3. Great increase of fibrous tissue.
4. Granular debris.

Stains deeply with carmine.
Chromic acid fails to stain.

Roots, when affected, exhibit similar changes.

DISEASE OF THE SPINAL MEMBRANES.

Spinal meningitis or inflammation of the meninges may be either acute or chronic. When the dura mater is principally affected it is termed pachy-meningitis; when the pia mater is most involved—lepto-meningitis. This distinction is convenient when we have to deal with a *slow* process, as in *chronic meningitis*. It should, however, be remembered in acute inflammation, though one or other membrane may be primarily affected, the disease quickly spreads, and involves the whole three membranes equally.

ACUTE SPINAL MENINGITIS.

The process might begin in the cellular tissue *outside* the dura mater—*i.e.*, external meningitis; or within the sheath, internal meningitis. The slight differences in the clinical symptoms will be easily understood if we consider the respective causes, and *recall* one or two physiological facts—

1. *Irritation* of nerve roots lead to severe radiating pains and increased reflexes.

2. *Compression* of the cord substance causes paralysis, and loss of reflexes in the area of the compression; so, consequently, we are prepared for a greater degree of local excitability when the inflammation is external, and a more extensive area of the lesion in the internal meningitis; the cord *substance* will also be involved more quickly.

External Meningitis.

Causes.—

Extension of contiguous disease, such as—

1. Caries of spine.
2. Fracture of spine.
3. Tumours.

Pathology.—

1. The dura mater is red and injected, with loss of lustre.
2. Exudation of lymph.
3. Accumulation of pus between *spine* and *membrane*. The pus frequently dries and forms a caseous mass.

Symptoms vary greatly.—

1. Severe cutaneous pains, followed by anæsthesia.
2. Localised pain in the back, worse on movement or pressure.
3. Exaggerated reflexes till pus is formed, then "paralysis" sets in, first in the legs, then ascends, and reflexes become abolished. Anæsthesia replaces hyperæsthesia; sphincters are paralysed; septic fever and lividity of the skin supervene; large bed-sores form, and the patient often succumbs from exhaustion.

Treatment.—

1. Remove cause, if possible, by trephining, or aspiration of abscess cavity, etc.
2. Absolute rest.
3. Counter irritation, cupping, actual cautery.
4. Drugs — bromides, chloral, antipyrin, etc.

Internal Meningitis.

Causes.—

1. Extension of external meningitis on the one hand, or of myelitis on the other.
2. Extension of *cerebral* meningitis.
3. Septic state of blood, as in fevers, etc.

Pathology.—

The membranes are involved to a much wider extent. The internal membranes suffer early. The arachnoid is frequently wholly disorganised, the pia mater deeply injected, and the dura mater bulged *outwards* from accumulation of pus; in severe cases, the *cord substance is semi-fluid.*

Symptoms.—

Are much the same, but of greater severity. There is more general excitement; opisthotonus, and retraction of the head are more marked; vaso motor centres are more deranged; and cerebral symptoms are much more common. Cheyne-Stokes' breathing is often typically seen. It may be impossible to distinguish this form from cerebro-spinal *fever*, or even acute myelitis.

Treatment.—

The same treatment, but Gowers advises in addition, the free application of oleate of mercury, and internal administration of *calomel.*

CHRONIC SPINAL MENINGITIS.

This is frequently the sequel to the acute affection, therefore, dependent on the same causes; but it is sometimes chronic from the beginning. In these cases, syphilis, alcoholism, certain poisons, or extension of any chronic disease of the cord are held as probable causes.

Pathology.—It varies much in different cases, from a mere thickening and cloudiness of the membrane with *increase* of cerebro-spinal fluid, to obliteration of the subarachnoidal space, by complete organisation of the inflammatory lymph, which matted the pia mater and dura mater together.

Nerve Roots.—At first are swollen and injected, but become fibrous and atrophied.

Spinal Cord suffers in proportion to—

1. The amount of compression from without.
2. The amount of thickening of the processes of the pia mater which *run into* the cord.

It should be noted when there is *much fibrous* formation between the *dura mater* and the *cord substance* it is termed Hypertrophic Internal Meningitis.

Symptoms are those of local irritation of the nerve roots, followed by paralysis; thus, from irritation of sensory fibres we get—

1. Pain in back with stiffness.
2. Sharp, darting, burning pains; occurring in various parts of the body, paroxysmal in character.
3. Spasm and rigidity.
4. After the cord has become invaded the symptoms will be those of local myelitis (which see).

Treatment.—Remove any cause if possible, then treat the myelitis.

MYELITIS.

Inflammation of the cord substance may be acute or chronic. Under the latter will be considered the various chronic spinal diseases; and under acute we shall consider—

1. A total transverse myelitis.
2. An unilateral myelitis (affecting one side only).

Causes—

(1) Extension of acute spinal or cerebral meningitis.
(2) Exposure to cold, etc.
(3) Occurring in the course of fevers.
(4) Concussion.

Symptoms again are due (1) to irritation of nerve roots, and (2) to paralysis from implication of cord substance. The following is a good table of the results of a total transverse lesion *after paralysis has become established*:—

Above the lesion—

1. Reflexes, etc., normal.
2. Hyperæsthesia at upper margin of lesion.

At the point of lesion—

1. Loss of reflexes.
2. Atrophy of muscles supplied by the implicated nerves.
3. Altered electrical conditions (*R.D.*).
4. Cutaneous insensibility.

Below the lesion—

1. Total loss of sensation.
2. Complete paralysis.
3. Muscular rigidity or spasm.
4. No alteration in trophic relations except from non-use.
5. *Vaso-motor dilatation.*

6. *Rectal Centre.* — Unconsciousness of need, and inability to prevent evacuation; therefore, we get constipation; and, *incontinence of fæces after an aperient.*

7. *Vesical Centre.* — Again unconsciousness of need, and inability to prevent micturition; so we get reflex evacuation in gushes at intervals.

8. *Sexual Centre.* — Absence of desire, but frequent reflex erections and emissions. (CHARLTON BASTIAN.)

9. Ordinary reflexes are exaggerated; the slightest touch of the bedclothes, catheter, etc. causing severe spasm.

It should be noted, however, that as time goes on the muscles from non-use may become flaccid and soft, with considerable wasting, and the reflexes also become exhausted.

Treatment.—As in acute meningitis.

UNILATERAL LESION.

At the point of lesion.—

1. On *the side of paralysis*, same as *transverse* lesion. (*vide supra*).
2. *On the opposite side*—anæsthesia without paralysis (?)

Below the Lesion.

SAME SIDE.	OPPOSITE SIDE.
Paralysis.	
Hyperæsthesia of skin. (?)	Anæsthesia (total ?)
Diminished muscular sense.	
Reflexes at first excited.	
Vaso-motor dilatation.	Other conditions normal. (?)
No atrophy.	

It will be seen, that as regards *unilateral* lesions, the queries placed show there is by no means an unanimous opinion. This is because, not only are clinical records conflicting, but physiological experiments are equally disappointing; indeed, in spite of much honest and hard work by experimenters, we are still far off from certainty regarding the spinal cord function.

INFANTILE PARALYSIS OR ANTERIOR POLIOMYELITIS ACUTA.

This, as the name implies, is an acute inflammation of the anterior horn of grey matter.

"Infantile" is not a good term, for though infants and young children under eight are most often affected, adults sometimes develop the affection. Women during the puerperal state sometimes become afflicted with this paralysis.

Causes.—Not known. "Chills" from exposure to wet and cold are usually blamed. Hæmorrhage into the cord, etc.

Pathology.—*At first*—

1. Anterior grey horn is red and swollen.
2. Minute extravasations of blood occur.
3. Cloudy swelling of anterior multipolar cells.

Later—

4. The motor nerve trunks show marked changes, being smaller and fewer in number.
5. The "neuroglia" becomes increased, and the grey horn as a *whole is shrunken.* (See Sclerosis, page 67.)

Muscles are pale and flabby; atrophy begins early, and is well marked.

Microscopically, the same changes take place (as before described) when muscles are cut off from trophic nerves.

1. Disappearance of striæ.
2. Atrophy of muscle cells, and
3. An increase of fibrous elements (see page 64).

Symptoms. — First, reason what we might expect. We have an interference of the reflex ~~roof~~, destruction of motor cells and motor trophic influence. The symptoms must be paralysis, absence of reflexes, and atrophy; the extent and severity of these symptoms will vary with the extent of the lesion. The onset is sudden, and after two or three days of rheumatic feverishness the paralysis becomes established. Usually the *lower* limbs are first affected, but all four limbs or only one, or only *a group of muscles* may be implicated. However, once the paralytic condition is established, atrophy, loss of reflexes, and reaction of degeneration, quickly supervene (see page 63). Note that "muscle wasting" may be concealed by fat.

Bladder and rectum usually escape. Sensation is but slightly affected, if at all.

Results—

1. Nutrition of bones, etc., is seriously affected, and the child may recover motor power, with a wasted limb.

2. Permanent deformities produced by—

   (1) Want of antagonism or unresisted contraction.

   (2) Weight of foot, etc., causing extension, and the occurrence of ankylosis in that position. (Talipes equinus.)

3. Even in most favourable cases there is some slight permanent deviation from a perfectly normal muscular apparatus.

Treatment.—Rest, salicylates, and warmth.

Later.—Strychnine, cod-liver oil, lacto-phosphate of iron and lime, electricity, and massage.

Gowers emphasises that complete recovery is impossible after a year, though a gradual improvement may to a certain extent take place from increased *movements* on the part of the patient.

CHRONIC DISEASES OF SPINAL CORD.

Having described the more common acute affections, we will now take up the more common chronic affections of the cord.

PROGRESSIVE MUSCULAR ATROPHY.

Progressive muscular atrophy, or chronic anterior poliomyelitis, is *the* example of a *chronic* affection of the anterior grey horn and motor roots; and again, from the pathological site, we are (as in the acute form) prepared for disturbances of reflexes, atrophy, and deficient muscular power; but the sclerotic changes being more localised and of slow growth, the clinical symptoms will vary.

Pathology.—Sclerosis of anterior horn, anterior roots and nerve trunks, with changes in groups of muscles.

1. Anterior grey horns are pale, but not altered much in shape.
2. Great increase of neuroglia, and
3. Obliteration of nerve cells.

Anterior nerve roots are markedly atrophied.

Nerve Trunks.—Changes are not so marked as in the roots, for the sensory fibres in the mixed nerve trunk are unaffected, and frequently *some* of the motor fibres escape.

Muscles.—Pale, flabby, etc., like in the acute affection; but sometimes fatty, vitreous, or waxy changes are also present. They exhibit the reaction of degeneration in proportion to the amount of wasting.

Symptoms.—Disease usually begins in the "*upper*" extremities with atrophy of—

1. Thenar and hypothenar eminences.
2. Interossei.

3. Shortening of extensors of the wrist, producing the claw-like hand—*i.e.*, the first phalanges are *hyper-extended*, the middle and distal phalanges are flexed on the first phalanges; next, the deltoid or biceps, and then the forearm become attacked. After this, the extension is extremely gradual, and years pass before both arms, both legs, intercostals, or diaphragm are attacked. The affected muscles exhibit the peculiar fibullar twitchings of dying or exhausted muscle. These twitches can be readily produced by a gentle tapping; they are often visible to the patient, lasting two or three minutes, and starting without any apparent stimulus. Probably no fatal case of *pure* anterior poliomyelitis chronica has occured. Gowers remarks he has always seen in fatal cases *evidence of a wider distribution of sclerosis.*

Treatment will be summed up under chronic nerve diseases.

Differential Diagnosis. (See Table.)

PRIMARY SPASTIC PARAPLEGIA.

(Primary Lateral Sclerosis.)

Primary spastic paraplegia is a paralysis attended with spasm and rigidity, resulting from sclerosis of the anterior pyramidal and crossed pyramidal tracts.

Let us just consider what we must expect from interruption of these two tracts.

1. *The brain being cut off from the motor nerves, two conditions are brought about:—*

(1) *Voluntary* motion must be imperfect, according to the extent of lesion.

(2) Reflexes must also be exaggerated, as they are cut off from the inhibitive influence of the brain, but the reflex loops are not interfered with. (See Diagram, page 81.)

2. *The motor tracts, being cut off from the first trophic realm in the cortex*, will undergo secondary degeneration, which causes at first *increased irritability*, which will excite *motor roots* and cause spasm and rigidity.

3. The anterior horn not being affected, there will be no interference with the second trophic realm, and consequently no atrophy of muscles.

Ætiology.—Most common in males about twenty. Children sometimes get the disease, through imperfect development of the tracts.

Symptoms—

1. Weakness and stiffness of the lower limbs.

2. Exaggerated knee jerk, and presence of ankle clonus.

3. Spasm and rigidity causing forcible *adduction* and extension of the limbs, rendering the gait characteristic. (See "Gaits.")

The rigidity is nearly continuous, and when relaxations take place the slightest stimulus causes spasm.

Though the legs are the limbs usually attacked, still the muscles of the trunk, and the arms occasionally, are first involved; but it is better to consider the latter as "amyotrophic paralysis" — *i.e.*, one of the "overflows" presently to be described.

The disease runs an extremely chronic course, as, until the disease becomes widely spread, the muscles remain plump, sensation unaffected, and sphincters unimpaired.

Differential Diagnosis.—(See Table.)

Treatment is considered under "Chronic Nervous Diseases."

Pathology. — Sclerosis of anterior and crossed pyramidal tracts (see page 67).

LOCOMOTOR ATAXIA.

Definition.—A nervous disease characterised by an indefinite onset; chronic, but progressive course; and attended with marked symptoms of inco-ordination, trophical changes, and disturbances of special sense.

This disease is the best example of disease affecting the posterior columns—though it is, as will be seen, associated with sclerotic changes in the cerebrum, etc.

Ætiology.—Most common in middle-aged men. Syphilis, exposure to severe weather, etc., sexual excesses are the usual causes. The last two are doubtful, but syphilis is most certainly a factor in producing this disease.

Pathology.—Sclerotic changes in (see page 67)—

1. Posterior nerve roots.
2. Posterior columns — Burdach first, then by secondary degeneration into Goll's column.
3. Restiform body (medulla).
4. Optic thalamus.
5. Certain nuclei of cranial nerves.
6. When the disease has advanced, it may attack any area.

Symptoms.—A careful digest of the foregoing pathological sites, will at once show that the symptoms will vary much in each case, but we are at once prepared for sensory changes, inco-ordination, and changes in the sight apparatus.

The disease is usually divided into three stages—

1. Pre-ataxic.
2. Ataxic, or stage of inco-ordination.
3. Stage of Paralysis.

PRE-ATAXIC STAGE.

Symptoms are most insidious. Taking a typical case we shall get—

1. *Changes due to irritation of sensory roots—*
   - (1) Lightning pains over the body: hot, burning, and tingling in character; lasting a few seconds.
   - (2) Girdle pains (from the upper margin of sclerosis), the patient complaining of constriction, as if an iron band was around him.
2. Early loss of knee jerk.
3. Symptoms in connection with the optical apparatus—
   - (1) Argyll-Robertson's pupil (pupil fails to react to light, but accommodation remains unaffected).
   - (2) Diplopia or double vision. } May occur early
   - (3) Optic neuritis. } or late.
4. Increased sexual desire, this is important in reference to charges of rape; *later, sexual desire is abolished.*

ATAXIC STAGE.

Often the first sign of inco-ordination is tumbling forward into the basin on closing the eyes during the morning wash, but once begun the inco-ordination often rapidly develops into the characteristic "Ataxic-gait." (See "Gaits.")

Soon anæsthesia of the soles of the feet comes on, the patient feels as if he were walking on wool, he feels unsteady on turning around or standing with his eyes shut, and fails to walk on a straight line, etc.

Both the tactile and muscular senses become markedly affected, and the patient fails to distinguish with his feet the difference between a hot and cold body, or between heavy and light weights, provided they are similar in appearance.

It should be noted particularly that the muscular *power* is not diminished, for the patient can resist movement or push away a heavy weight.

Often at this period, occur peculiar visceral disturbances, termed crises. The best known are—

1. Gastric crisis—intense epigastric pain, hyper-acidity, and vomiting.
2. Larnygeal crisis—noisy stridulous breathing, with great dyspnœa.
3. Vesical crisis—paroxysmal retention of urine.
4. Rectal crisis—tenesmus, etc.

The other most marked changes are the so-called "trophic" alterations :—

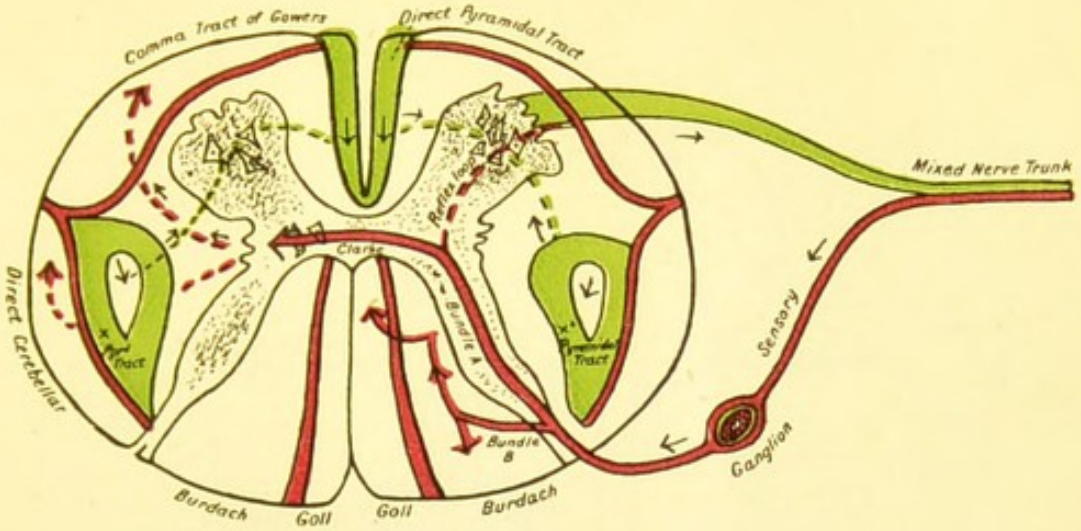
1. Skin becomes dry, or shiny and glossy with absence of hair; nails crack, etc.
2. *Joints.*—Charcot's disease—identical in its pathological changes with *rheumatoid arthritis* (which see).
3. Perforating ulcer of foot.
4. Ulceration of bones, cartilage, etc.

PARALYTIC STAGE.

The patient becomes bed-ridden, and liable to grave intercurrent diseases; and hemiplegia, pneumonia, or gangrene, etc., usher in a fatal ending.

It must be particularly noted that some cases run an extremely lengthened course, others very rapid, and though it is convenient to divide the disease into three stages, there is no hard-and-fast line or distinct margin between them.

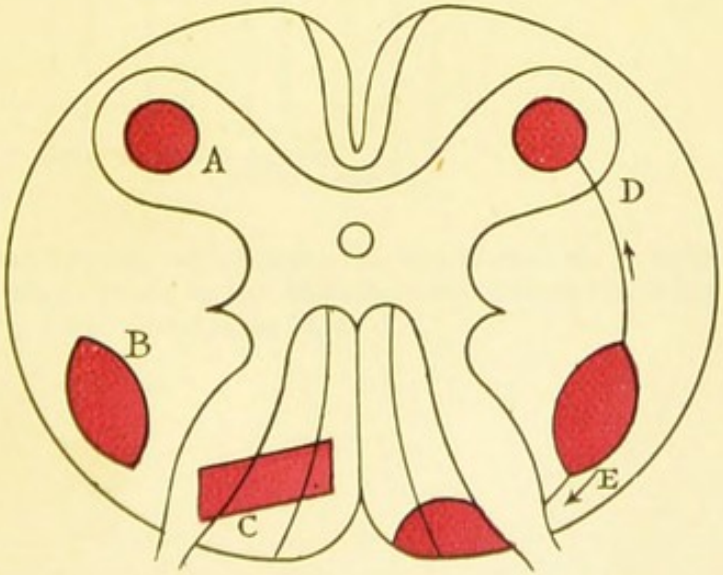
Treatment will be considered under "Chronic Nervous Diseases."



A DIAGRAM, designed by the author, to show the course of the motor fibres (*green*) and the sensory fibres (*red*) in the cord and nerve roots.

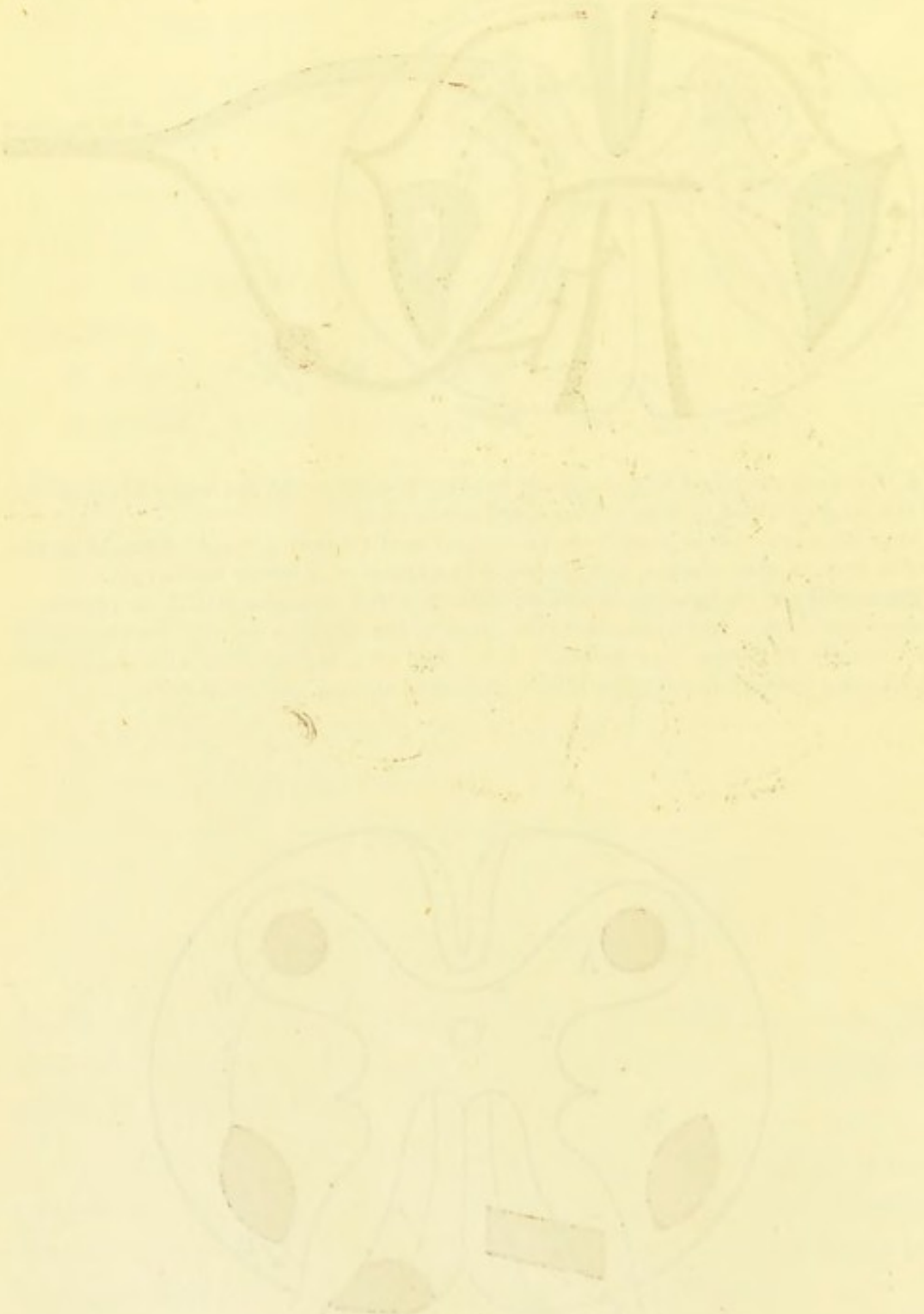
Note the motor fibres pass from the direct and crossed pyramidal tracts to the anterior horn of grey matter, and thence to the anterior or motor nerve root.

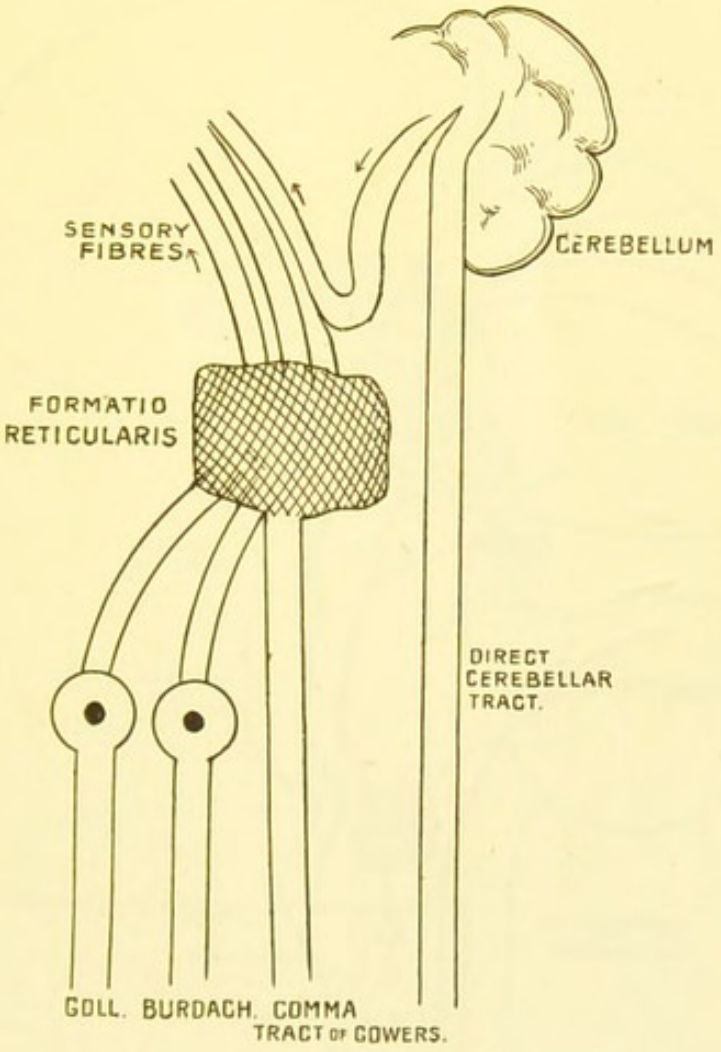
The sensory fibres pass in, in two bundles, A & B. Observe bundle A crosses to the opposite side, a few fibres however going to the anterior horn to form the reflex loop; bundle B passes into Burdach first, then at a higher level into Goll's tract (so that some sensory fibres cross to the other side at once, others do not).



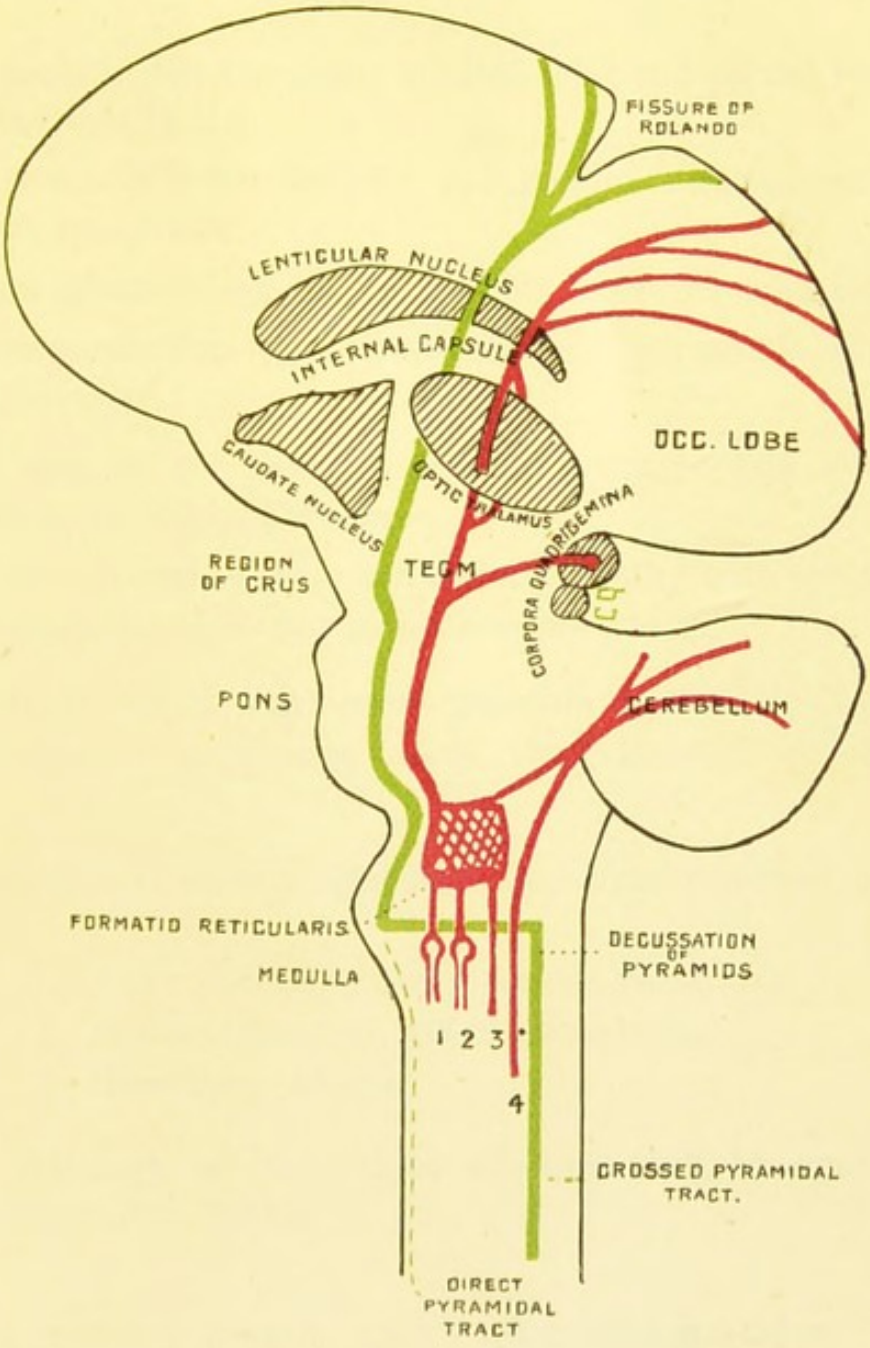
DIAGRAMMATIC DRAWING to show the fibres involved by lesions at A, B, C, D, & E.

A, Progressive Muscular Atrophy.
B, Spastic Paralysis.
C, Locomotor Ataxia.
D, Amytrophic Lateral Sclerosis.
E, Spastic Ataxia.





DIAGRAMMATIC SCHEME, by the writer, to show a possible re-arrangement of the Sensory Tracts in the Medulla before passing up to the Cortex as sensory fibres. (See pages 60, 61.)



Diagrammatic Drawing, designed by the writer, to show the course of the principal motor (*green*) and sensory fibres (*red*) between the medulla and cortex. (See pages 59–61.)

CHRONIC SPINAL DISEASES,

With mixed Lesions or "Overflows."

We have, in the foregoing, studied total and partial lesions; and, found that in the

Anterior affection—atrophy and loss of reflexes were the prominent symptoms.

Lateral affection—spasm and rigidity with increased reflexes.

Posterior affection—inco-ordination and anæsthesia were the marked features.

It is easy to see that we might get compounds of these; for instance, we might have an—

Antero-lateral lesion = incomplete atrophy with spasm; or
Postero-lateral = inco-ordination and spasm.

Indeed, it would seem more probable at first sight to get these "mixed" conditions rather than isolated or limited lesions.

We shall next describe the three best known mixed lesions, viz.:—

1. Antero-lateral or Amyotrophic paralysis.
2. Postero-lateral or Ataxic paraplegia.
3. Hereditary Ataxia.

The **Ætiology** of these three affections will be considered under "Diagnostic Tables."

AMYOTROPHIC LATERAL SCLEROSIS.

Pathology.—Sclerosis of—

1. Crossed pyramidal tracts.
2. Anterior horns of grey matter.

Symptoms.—

1. Weakness and wasting of the *upper* extremities coming on very slowly.

2. *Increase of reflexes with spasm* (until the wasting is profound; then, the anterior multipolar cells being extensively diseased, the reflexes *may be actually abolished*). The spasm and rigidity cause a peculiar deformity which is characteristic, viz. :—

Arm extended close to the body.
Forearm semi-flexed and pronated.
Wrist strongly flexed.
Fingers bent into the palms. (CHARCOT.)

3. In about twelve months, wasting and rigidity commences in the *lower extremities.*

4. Symptoms dependent on *extension of the disease to the medulla, tongue, face,* etc. Any of these may be involved and undergo atrophic changes, then the vital centres soon become involved, and the patient dies.

The disease runs a quicker course than the three chronic spinal diseases already described, and death usually occurs in from one to three years.

The beginner is frequently puzzled to understand why, in this disease where *the anterior horn* is involved, reflexes are increased instead of lost. The explanation is, that this affection *begins* in the "*lateral*" region but *extends* into the anterior horn, paralysing only a certain number of motor cells, and *irritating* others. Sensation is not impaired, and the sphincters are normal.

ATAXIC PARAPLEGIA.

Pathology.—Sclerosis of—

1. Lateral columns, (direct cerebellar and crossed pyramidal).
2. Posterior columns (but not the roots).

Symptoms.—Obscure in the early stages.

1. Patient complains of tiredness and weakness of the limbs (*no* lightning pains).

2. Inco-ordination, patient reels or sways if the eyes be closed, and he fails to walk on a straight chalked line.

3. *Increase* of knee jerk (ankle clonus is often present).

4. As the disease progresses the tendency is to become *more spastic,* and less ataxic in character—*i.e.*, inco-ordination does not increase, but *spasm and rigidity* become markedly increased.

It should be noted that there is no anæsthesia, lightning pains, or alterations of the optical apparatus; points which distinguish it from locomotor ataxia; and this is what we should expect, seeing there is no involvement of the posterior *nerve roots, or cranial nerves.* We might consider here an interesting form of ataxia, called—

FRIEDREICH'S DISEASE (Hereditary Ataxia).

Pathology.—Sclerosis involving principally the "neuroglia" element in—

1. Lateral columns—Gower's, direct cerebellar, and part of crossed pyramidal.
2. Posterior columns.
3. Clarke's columns in grey matter (to a slight degree).
4. Certain cranial nuclei.

Symptoms—

1. Inco-ordination of a jerky kind of the lower extremities.
2. Inco-ordination of the *arms* (somewhat later).
3. Hesitation in speaking, or a jerky manner in delivery of speech.
4. Irregular movements of the *head and eyeballs* (nystagmus).

5. Impairment of sensation—the anæsthesia is not marked in most cases.

6. Absence of knee jerk.

7. *Talipes equinus and other deformities*, especially curvatures of the spine.

Note *absence* of lightning pains, trophic changes, and the Argyll Robertson pupil to distinguish this disease from locomotor ataxia. The marked inco-ordination and nystagmus renders the diagnosis easy from that of spastic paralysis.

The most marked feature in the ætiology of this disease is, its *congenital* tendency, and its appearance in more than one member of a family. It is transmitted through the father, and is usually first noticed in the child about the age of eight years.

PSEUDO-HYPERTROPHIC PARALYSIS.

A nervous disease which manifests itself during the later developmental period of childhood, and characterised by a progressive change in size and diminution in the power of many muscles (GOWERS).

Ætiology—occurs principally amongst boys; and, in the few cases of which we have any authentic account, there seems a strange tendency to affect more than one male member in the same family. It is hereditary in a peculiar manner, as whilst it affects males, it is transmitted through the females—*i.e.*, in a certain family a son was affected; his sister married, and had two daughters and one son; the daughters escaped, but the *boy* was affected. The writer has a great friend, whom he had under his observation for four years; he is now twenty-six, and has been seen by Drs Byrom Bramwell and Gowers. In this case I can detect nothing which would suggest even a possible cause; indeed, after four years' steady and minute search of the literature of this disease, I fear there is absolutely nothing

known of the origin of this paralysis. All the suggestions thrown out are weak, and as devoid of any *real* explanation as Dr Gowers statement—"It is a perverted tendency of development inherent in the germinal tissue of the muscular system."

Symptoms are very characteristic when the disease is fully developed, but in the earlier periods they are somewhat obscure.

1. Impaired locomotion from muscular weakness, the little fellow lags behind his playfellows.
2. Hypertrophy of muscles with atrophy of others.
3. Certain deformities, through contraction of muscles unantagonised by the paralysed ones.
4. The characteristic gait and movements.

During walking the abdomen is thrown out, with a corresponding *hollowing of the back*; the legs are widely spread out, the whole effect being a *waddling gait.*

The getting up from a recumbent position is most characteristic, especially if there be no objects near, by which he can aid himself. He first gets on his hands and knees; then, extends his knees to the utmost; and lastly, by a climbing movement—*i.e.*, grasping the knees with his hands alternately, moves higher and higher up the thigh till the trunk is raised.

It is remarkable how easily some patients can manage these movements; later, the muscular weakness is such that they require the aid of near objects to grasp at, but finally failure is complete, the patient being helpless.

Pathology.—The essential changes seems to be in the muscular tissues alone. Changes in the cord are rare, and probably are only accidental complications. Microscopically there are seen in the affected muscles—

1. Increase of fibrous tissue.
2. Atrophy of muscle fibres.
3. Large deposit of fat.

The enlarged appearance of the muscles is due more to fat than the increase of fibrous tissue. The muscles mostly involved are as follows—

ENLARGED.	WASTED.
1. Calves.	1. Latissimus dorsi.
2. Glutei.	2. Teres major.
3. Infra spinatus.	3. Pectoral muscles.

The above table of course is only approximate, there seems to be no hard and fast line as to what muscles will show most changes.

The reaction of degeneration is in proportion to the atrophy; as long as any true muscular tissue is left there will be response to stimulation. The knee jerk is lost when the tonicity of the extensors fails.

Treatment.—Expect most benefit from a thorough system of continued massage, well planned gymnastic exercises, and careful hygiene.

Tonics such as strychnine and phosphorus may be given. The writer thinks there was decided improvement in two cases he attended, after *hypodermic injections of arsenic*, ij minims three times a week; but, untoward circumstances prevented an extended trial. The thinness of the chest walls renders the patient very prone to chest affections, which, should they occur, often carry off the patient, and are to be particularly guarded against.

DISEASES OF CRANIAL NERVES.

All have at least two origins—viz., deep and superficial; all behind the 4th nerve may be said to have their deep origin in the 4th ventricle. The nerves of the special senses—*i.e.*, 1st (smell), 2nd (sight), 8th (hearing), have in addition a special

connection with the so-called "centres," situated in the cortex of the brain. We have left out the sense of "taste" here, for no two authorities agree upon which is the special nerve, though there is increasing evidence in favour of the 5th being the nerve most implicated. For a special "sense" to be carried out properly, we can at once see that there must be a healthy continuity between *centre*, nerve *trunk*, and *special ending;* destruction of any of these will interfere with the carrying out of the function. We shall see when we proceed to study the brain, that there are in all probability, connections between all five senses (possibly through commissural fibres connecting the centres); thus, "taste is impaired when the smell is interfered with; hearing is sometimes more acute when blindness ensues, and so on."

OLFACTORY OR FIRST PAIR.

Anatomy.—They arise from the olfactory bulbs, pass through the ethmoidal plate, and are distributed to the mucous membrane in the upper part of the nose.

Physiology.—The nerves of smell. Pungency should be carefully distinguished from the sense of smell, or the detection of odours; pungency is indeed due to painful stimulation of the 5th nerve in the *lower* part of the nose.

Clinical.—Highly nervous or insane people often complain of—

1. Hallucinations of Smell.—They may complain of unpleasant odours, or that totally different odours are all alike—such as a rose and a herring having the same odour, which may be to them either pleasant or otherwise.

2. Increased Sensitiveness.—Perfumes become intolerable, or patients may tell by odour alone the different members of their own family.

3. *Total* loss of Smell.—This may be due to—

   (1) Affections of the *terminations* of the nerve (most often associated with nasal catarrh or polypi).

   (2) Lesions of the nerve *trunk.*

   (3) Lesions of the *centre*, in the uncinate gyrus.

Treatment.—Ferrier's snuff, douching with carbolic acid solution, after removal of any apparent cause, such as polypi, etc., are the remedies indicated.

OPTIC NERVES.

(See Diagram.)

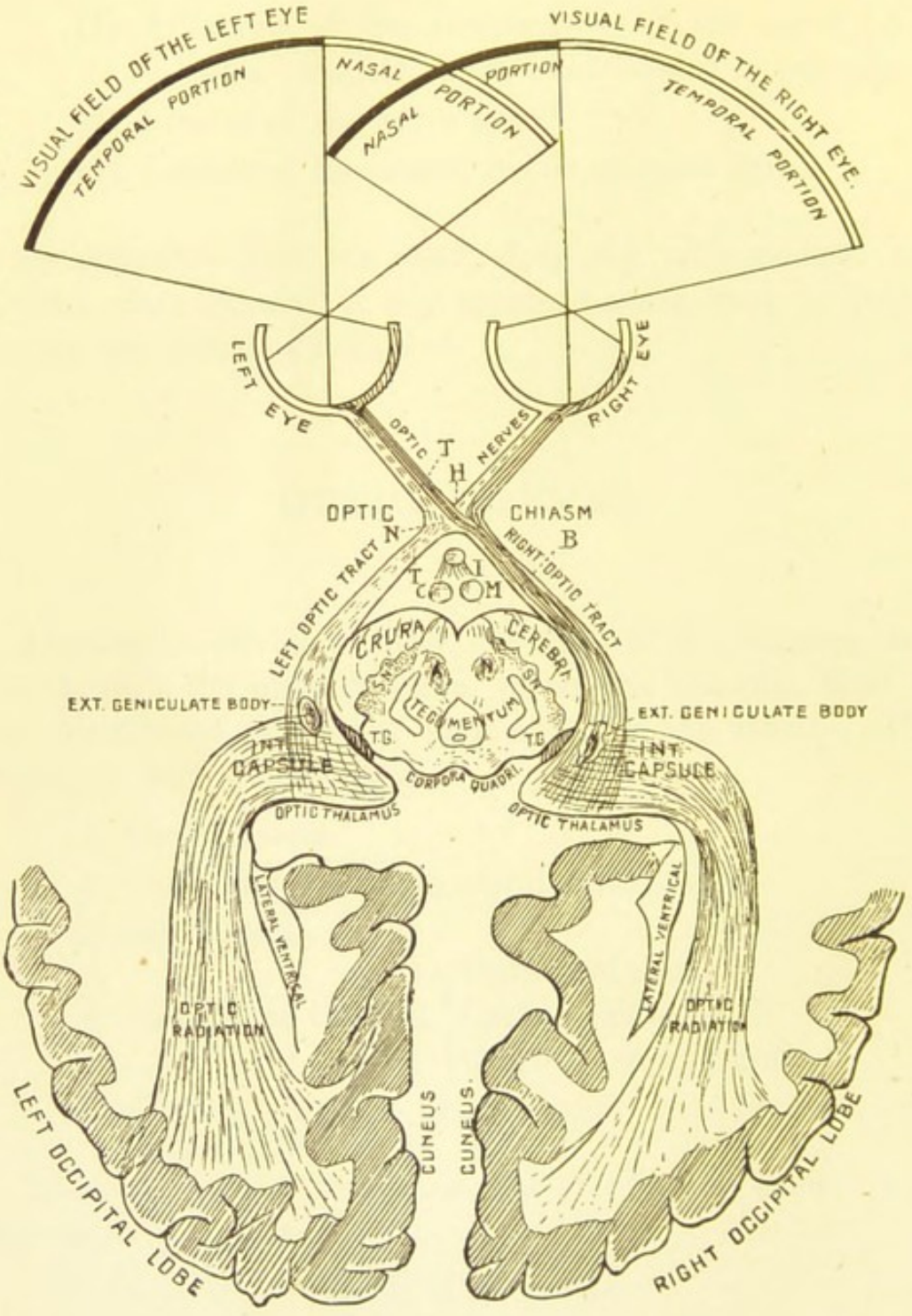
Anatomy.—Given off from the sides of the chiasma, they pass through the optic foramen, pierce the sclerotic, then become distributed as delicate filaments over the retina. Thus we get, on tracing backwards—

1. Optic Nerves.
2. Optic chiasma or *partial* decussation.
3. Optic tracts.
4. Ganglionic Centres. { Optic thalamus. / Corpora geniculata. / Corpora quadrigemina.
5. Cortical centres—*i.e.*, radiation in the occipital lobes.

There are no less than *three* sets of fibres, as shown in the diagram—

1. Outer set to eyeball of *same* side.
2. Middle fibres which decussate with the fibres of the opposite tract, forming the commissure.
3. Fibres having no real connection with vision (Ferrier).

Physiology.—Nerves of sight.



THE OPTIC AND VISUAL TRACTS (Starr).

N, Lesion causing nasal hemianopia. *T*, Lesions causing temporal hemianopia. *H*, Lesion causing bilateral heteronymous hemianopia. *B*, Lesion of tract causing homonymous hemianopia.

Clinical—

1. Affections of the *terminations* in the retina include—
   (*a*) Functional disturbances due to toxic effects, viz.—
      (1) Uræmia.
      (2) Jaundice.
      (3) Drugs—quinine, santonine, etc.
   (*b*) Hysterical amaurosis.
   (*c*) Tobacco amblyopia. The loss of sight is gradual, but, if the disorder persists, there may be *permanent* changes in the optic disc.
   (*d*) Night blindness—(nyctalopia). Objects are clearly seen by day, but not in the dusk; or (hemeralopia); objects are *easily* seen in the shade, but, in the sunshine, with difficulty.

2. Lesions of the Nerve.
   (*a*) Optic Neuritis.—The disc becomes blurred, swollen and red from congestion; there may be slight extravasations of blood; finally, there is an increase in the fibrous tissue, and atrophy of the nerve elements, causing often total blindness. It is common in Bright's disease, tumours of the brain and cerebellum, and other intercranial affections.

The blindness is preceded by changes in three directions—
   (1) Diminished acuity of vision.
   (2) Alteration in the *field* of vision.
   (3) Altered perception of colour (Gowers).

3. *Lesions of the Chiasma.*—If the lesion is confined to the central portion—*i.e.*, the decussation, the fibres passing to the *inner* or nasal portion of each retinæ will be involved, causing blindness of the *outer* half of each field—*temporal* hemianopia. If the lesion is more extensive there may be total blindness of the eye on same side, with temporal hemianopia of the opposite; or, a more extensive lesion still, may cause total blindness.

4. A Lesion *behind the Chiasma*, whether in optic tracts, optic thalamus, corpora quadrigemina or geniculate bodies, causes *lateral hemianopia*, that is, blindness of the *temporal* half on the same side, and the *nasal* on the opposite side. A lesion in the occipital—

5. *Cortex* also causes lateral hemianopia; but there is in addition, word blindness, *i.e.*, the patient *can see* but fails to *read letters.* (See Aphasia.)

THIRD NERVE.

Anatomy.—Arises from the floor of aqueduct of Sylvius, passes through tegmentum of crus on the inner side, and forwards to be distributed to—

1. The sphincter pupillæ.
2. Ciliary muscle.
3. All the muscles of the eyeball except superior oblique, and external rectus. *It also supplies levator palpebræ superioris.*

Physiology. — The nerve concerned in accommodation of vision and certain movements of the eyeballs.

PARALYSIS.	IRRITATION.
1. Ptosis or drooping of upper lid.	1. Convulsions or spasm of the the muscles.
2. Divergent squint, with inability to move the eye inwards, downwards, or upwards.	2. Convergent squint.
3. Loss of accommodation.	3. Contraction of pupil.
4. Paralysis of sphincter of iris = dilatation of pupil.	4. Great interference with field of vision.
5. Double vision.	

Clinical.—Symptoms due to paralysis, or irritation of the third nerve, are common in many intercranial affections; for instance, the former condition is usually well marked in

fractures of the base of the skull, the "*compression*" stage of meningitis, etc. ; whilst *irritation* of the nerve is often brought about by tumours, the *first stage* of meningitis, reflexly by stimulation of the sensory fibres through intestinal irritation, (worms, etc.)

It should be noted that only a *few* of the great number of fibres running in the third nerve may be affected, and the clinical phenomena will obviously vary with the number of fibres involved. We will consider the fibres to the iris first—

Paralysis of the Iris.—May be of three types—

1. Accommodative iridoplegia = failure to accommodate for near vision.

2. Reflex iridoplegia = failure of reaction to "light" stimulus, constituting the Argyll-Robertson pupil, a most important symptom in loco-motor ataxia. In this case, the lesion probably interrupts the fibres between the corpora quadrigemina and the aqueduct of Sylvius.

3. Loss of skin reflex—failure of the pupil to dilate when the skin of the forehead is pinched.

Affections due to Alterations in the Muscular Apparatus of the Eye—

1. Nystagmus, or clonic convulsions of both eye-balls.
2. Strabismus or squinting.

Nystagmus may be due to—

1. Irritation of *fibres* in connection with the third nerve nuclei, as seen in—
   (1) Lesions of the restiform body in the medulla.
   (2) Lesions interrupting the afferent fibres administrating to the functions of the cerebellum — *i.e.*, eighth nerve which conveys impulses from the semi-circular canals.

2. Tiredness or exhaustion of the ocular muscles after severe strain, common in miners after working with small Davy lamps; or after excessive glare, as seen in electric light employés.

Squinting or Strabismus.—In order to have perfect sight, there must be perfect harmony between all the ocular muscles; for instance, when we look at an object to the right, we turn *both* eyes in that direction, and thus we call into action the *external* rectus (sixth nerve) of the *right* eye, and the *internal* rectus (third nerve) of the *left* eye. In other words, on looking to the right side, we employ the services of two nerves, the third and fourth. Other movements will involve other combinations, such as the third and sixth nerves, third, fourth, and sixth, etc. Obviously then, there must be a centre for the harmonious or co-ordinated actions of the third, fourth, and sixth nerves. This centre is placed by physiologists under the corpora quadrigemina, or in the "iter." A failure of this harmonious action produces, on certain movements of the eyes, an alteration in vision termed *strabismus*, characterised by—

1. Diminished field of vision in certain directions.

2. Diplopia or double vision—*i.e.*, the appearance of a *true* and *false* image of the same object.

3. Erroneous projection—*i.e.*, errors in the judgment of the distance of objects (due to greater effort being required to focus for distant objects).

4. Secondary deviation—*i.e.*, if the affected eye be fixed on an object, it requires a greater effort than usual: but the muscle of the opposite eye, which acts in combination, will then be *unduly strained* and make a greater excursion than usual = secondary deviation.

Results of Paralysis of Special Ocular Muscles when the others are Sound.

Paralysis of Rectus Superior: inability to raise eyeball properly above horizontal level; pupil may diverge somewhat downwards, and a little outwards (from action of the rectus inferior and the obliqui).

Paralysis of Rectus Inferior: inability to lower eyeball properly below horizontal level; pupil may diverge somewhat upwards, and a little outwards (from action of the rectus superior and the obliqui).

Paralysis of Rectus Externus: inability to turn eyeball properly outwards; pupil diverges inwards (from action of rectus internus).

Paralysis of Rectus Internus: inability to turn eyeball properly inwards; pupil diverges outwards (from action of rectus externus).

Paralysis of Obliquus Superior: but little alteration in movements of eyeball; slight deviation of cornea upwards and inwards, or simply upwards.

Paralysis of Obliquus Inferior: but little alteration in movements of the eyeball; slight deviation of the cornea downwards and inwards. (Paralysis of the sphincter of the iris, giving rise to a moderate dilatation of the pupil, and paralysis of the accommodation, often accompany this form of paralysis; this depends on the branch to the lenticular ganglion being given off from that branch of the third nerve which goes to the inferior oblique muscle. Occasionally, however, the lenticular branch arises from the sixth nerve.)—FINLAYSON'S MANUAL.

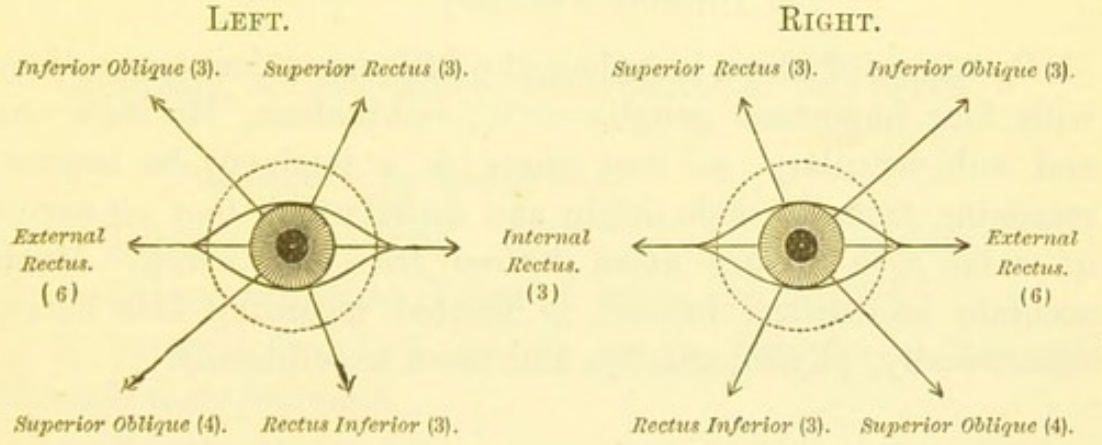


Diagram to illustrate the directions towards which the *pupil* is moved by the separate action of the six muscles of the eyeball. The eyes are turned inwards and outwards by the external and internal recti; the internal rectus of one side is the yoke-fellow of the external rectus of the opposite side in these conjugate movements. The eyes are turned upwards by the superior rectus and inferior oblique, downwards by the inferior rectus and superior oblique.

The eyes are represented facing the observer; *if it be imagined that the eyes are looked at from the back,* so that the diagram right-left becomes left-right, it will serve to illustrate the displacements in the field of vision caused by *paralysis* of individual muscles and nerves.—WALLER'S PHYSIOLOGY.

Strabismus is a most frequent cause of headache, etc., and probably many of the failures to treat hemi-crania successfully, etc., are due to the neglect of testing the visual apparatus, and thereby failing to rectify "eye-strain."

FIFTH CRANIAL NERVE.

Anatomy. — The fifth cranial nerve has a most extensive origin. The middle or motor root arises from the floor of the fourth ventricle; the two sensory (ascending and descending) arise from cells in the lower part of the medulla, and also from the outer wall of the aqueduct of Sylvius. The nerve in front of the Gasserian-ganglion divides into three branches—

1. Ophthalmic.
2. Superior Maxillary.
3. Inferior Maxillary.

By means of these branches, the fifth nerve has connections with four important ganglia—viz., ophthalmic, Meckel's otic and sub-maxillary, so that there is a tendency to assume, reasoning from its wide origin and distribution, *that all nerves after the fifth contain fibres derived from that nerve.* Much accurate knowledge, indeed, is wanted regarding this nerve, anatomically, physiologically, and more so, clinically.

Physiology.—It contains—

1. *Sensory fibres* to whole of head, except pharynx, Eustachian tube, tympanic cavity, and palate.

2. *Motor fibres* to temporal, masseter, both pterygoids, anterior belly of digastric, mylo-hyoid (muscles of mastication), and tensor tympani.

3. *Trophic fibres*, widely distributed (though disputed by some).

4. *Fibres* derived from—

   (1) Sympathetic = dilator pupillæ, sweat fibres, vasa dilators and constrictors.

   (2) Chorda Tympani from seventh = vasa dilator, and secretory to sub-maxillary gland, and taste (?)

   (3) Jacobson's nerve to parotid, from the ninth.

Paralysis of the *whole* nerve causes—

1. Loss of sensation to parts supplied.
2. Inflammatory changes in the cornea, due to—
   (1) Anæsthesia (irritating particles are not felt, therefore not removed).
   (2) Atrophic changes?
3. Impaired taste in anterior two-thirds of tongue. Gowers suggests that the *fifth* is *the* nerve of taste, and that the other nerves—viz., seventh and ninth—administer to taste *only in as much as they contain fibres from the fifth.*
4. Inability to detect pungent odours.

Clinically, we get various disturbances of this nerve.

Paralysis may be due to—

1. Disease of the pons.
2. Injury or disease at *base* of brain, medulla, etc.
3. Pressure from tumours, etc., on the branches as they pass through their foramina.
4. Peripheral neuritis.

A reference to the distribution of the nerve will give the symptoms attendant on paralysis of the whole or different branches.

Neuralgia may occur in all the branches of the fifth nerve. It is most often unilateral, or confined even to one twig. Caries of the teeth, or disease of the various bony canals, are most common causes.

Redness of the part affected is very characteristic; due to reflex disturbance of the sympathetic.

Trismus or spasm of the muscle of mastication may occur from irritation of the third division. This condition may be produced reflexly by stimulation of various nerves of the head.

Trophic changes may be confined to—

1. The cornea.
2. Hair roots (hair becoming grey).
3. Circumscribed areas of skin, affected with eruption of herpes. The pain is often intense, and may persist for years after the herpes have disappeared.
4. *Progressive unilateral atrophy* of the face. (Very rare.)

Treatment.—Paralysis.—(See general treatment on Nervous Disease.)

For Neuralgia.—Extract carious teeth; give quinine, gelseminum, tonga, croton-choral hydras, salycilates if there be a rheumatic history, or iodide of potassium and strychnine in syphilitic or broken down constitution. Morphia, hypodermically, may be required for continued suffering. Excision of the Gasserian ganglion is of doubtful value.

SEVENTH NERVE.

Anatomy.—This nerve consists of two parts, the facial part (portio dura), and the auditory part (portio mollis); but, in medicine, the facial portion only is meant when speaking of the seventh nerve.

The facial part arises from a groove between the pons and restiform body; its deep origin from the *dorsal* aspect of the pons and 4th ventricle. It is also connected by motor fibres with the cortex in the fissure of Rolando. Briefly, its distribution is as follows:—

1. *Motor* to—
   - (1) Muscles of expression.
   - (2) Stylo-hyoid.
   - (3) Posterior belly of digastric.
   - (4) Stapedius.
   - (5) Platysma Myoides.

2. *Secretory* to—
   (1) Sub-maxillary gland.
   (2) Sub-lingual gland.
   (3) Glands of mouth and tongue.
3. *Sensory.*—Nerve of taste, for anterior two-thirds of the tongue.

Paralysis Causes—

1. Motor changes—
   Face drawn to opposite side.
   Eye wide open, unwinking.
   Cheek puffs out with expiration.
   Food collects between cheek and teeth.
   Absence of wrinkles.
2. Sensory changes—
   Loss of taste in anterior two-thirds of the tongue.

Clinical.—Paralysis may be due to lesions affecting—

1. The cortical fibres—supra-nuclear paralysis.
2. The nucleus itself.
3. Nerve trunk in its tortuous course through the pons and bony canals.

The former (cortical) is associated with hemiplegia; and it should be at once noted that the paralysis will vary with the site of the lesion.

When it is a part of hemiplegia, the paralysis is on the *opposite side of the lesion*, that is, on the *same side* as the paralysed limbs; but the upper branches of the nerve escape, the orbicularis palpebrarum is *not* involved, thus *movements of the eyelids are not affected—i.e.*, winking is possible.

If the lesion be in the *lower* section of the pons, it will involve the facial fibres *between* the nucleus and their emergence in the pons. Thus we get "*crossed paralysis*"—*i.e.*, the face is paralysed on the *same* side as the lesion, the arms and legs (as before) on the opposite side.

Symptoms. — When the whole nerve is paralysed, the affected side is immobile, smooth, and expressionless. The eye cannot be shut, and usually waters freely. Smiling or whistling, etc., bring out the contrasts markedly. The food collects between the teeth, because of the buccinator paralysis. The paralysis of the stapedius (?) causes increased hearing to low sounds. The soft palate is not affected (being supplied by the spinal accessory nerve). The anterior part of the tongue is insensible to taste.

The electrical reactions are highly important. When there are *no polar* changes, *the case recovers rapidly.* If the reaction of degeneration be present, the prognosis is much more unfavourable. To sum up—

In the *nuclear* form of paralysis the whole of the one side of the face is involved, and typical "Bell's paralysis" results.

When of *cortical* origin it is—

1. Associated with hemiplegia.
2. *Upper part of face escapes.*
3. *Voluntary* movements are more affected than *emotional* movements.
4. The affected muscles seldom atrophy.

When in the lower portion of the *pons*, we get *crossed paralysis*, but of the nuclear type, as regards the muscles affected.

Causes—

Cortical Form.—Those of hemiplegia.

Peripheral. — Exposure to draughts, rheumatic neuritis, disease of the middle ear, etc.

Nuclear.—Extension of chronic spinal diseases, etc.

Treatment.—Remove if possible any pressure on the nerve by causing absorption of inflammatory products. Massage, electricity, and flying blisters are all useful. The peripheral type is usually very amenable to treatment.

EIGHTH OR AUDITORY NERVE.

Anatomy.— It arises from two nuclei in the floor of the fourth ventricle. Some of its fibres are connected with the cerebellum, and also with the cortical centre situated in the superior temporo-sphenoidal lobe.

Physiology.—The nerve fulfils two functions—

Firstly, it is the nerve of hearing, or that by which sound undulations are conducted from the labyrinth to the cortex, and there analysed.

Secondly, by means of its fibres in connection with the ampulla and semi-circular canals it transmits impulses from the oscillations of the endolymph in those regions, to the *cerebellum*, and thus assists the co-ordinating function in the maintenance of the equipoise of the body.

Paralysis causes total deafness, often attended by certain unpleasant subjective and objective symptoms.

Excitation of the auditory fibres cause unpleasant noises often accompanied with intense giddiness, nausea, and oscillations of the eyeballs.

It is highly important to remember the close sympathy and relation that exist between the optic, third, and auditory nerves; and again, between *these nerves and the cerebellum.*

Clinically we get—

1. Functional disturbances of the auditory nerve, including noises, buzzing, or "bell in the ear," and is often due to the accumulation of wax, altered tympanic pressure, etc.

2. Lesions affecting the cortical centre, causing word deafness—*i.e.*, spoken words are heard as noises, and not understood. (This condition may be associated with "word blindness," and "word aphasia," which see.)

3. Lesions of the nerve at the base of the brain, causing permanent deafness.

4. Disease of the labyrinth of the ear. The symptoms will depend on the extent of the lesion, there may be—

(1) Hyper-sensitiveness.
(2) Diminished hearing.
(3) Total deafness.

The subjective symptoms are typically brought out in—

5. *Ménière's Disease.*—An affection characterised by sudden and intense nausea, vomiting, vertigo, noises in the ear, and deafness of one ear. As we have already mentioned, nystagmus is frequently present. The attacks occur paroxysmally. The pathology is unknown, but it seems to be due to either—

1. Some affection of the semi-circular canals.
2. Disease affecting the cortical centre, or
3. Vaso-motor *neurosis* of the *vessels* of the labyrinth.

Treatment.—In any affection of this nerve first try to remove any discoverable cause. If associated with gout or syphilis treat these diseases primarily. Keep the external meatus clean, as deafness, giddiness, etc., are often caused by dried wax pressing on the drum. During an attack of Ménière's disease, the patient should be kept quiet in a darkened room; a brisk purge may be administered; and antipyrin, with an effervescing mixture containing caffeine, may be prescribed. When the nerve is affected through disease of the bony canal, the treatment is highly unsatisfactory.

DISEASES OF THE MEDULLA.

Much that has been said about the spinal cord may be also said about the medulla, or bulb. It is a conductor of impulses

to and from the brain, and it also contains the principal reflex centres. The more important points may be summed up as follows—

1. It contains the deep origin of all the cranial nerves after the fourth.

2. The *motor fibres* decussate to form the crossed and direct pyramidal tracts in the cord.

3. The *sensory* tracts become re-arranged as sensory fibres, prior to their course to the cortex.

4. It contains the following centres—

(*a*) *Centres essential to life.*

1. Respiratory centres.
2. Cardiac centres—motor and inhibitory.
3. Vaso-Motor centres.

(*b*) *Centres connected with the alimentary canal.*

1. Centre for sucking.
2. Centre for mastication.
3. Centre for vomiting.
4. Centre for deglutition.

(*c*) *Centres connected with the eye.*

1. Centre for winking.
2. Centre for dilator pupillæ.

(*d*) *Centres for secretion.*

1. Salivary centre.
2. Lachrymal centre.
3. Sweat centre.

It will be seen from the above table that even a small lesion may be attended by grave and diverse symptoms. An extensive lesion would be incompatible with life. Some authorities doubt if the medulla is ever diseased primarily without a speedily fatal issue; it is often, however, affected in the later stages of spinal basal disease, disseminated sclerosis, etc. Probably the best example of disease of the medulla is the so-called chronic progressive bulbar paralysis, often termed glosso-labial paralysis.

GLOSSO-LABIAL PARALYSIS.

An affection characterised by progressive paralysis and atrophy of the tongue, lips, etc., accompanied by difficult articulation and deglutition, ending in suffocation or inanition.

Ætiology.—It occurs most frequently in old people, but is sometimes seen in persons as early as thirty years of age. Men are more frequently attacked than women. Exposure to cold, blows and injuries to the neck, are usually put down as the principal causes. Often this disease is associated with chronic diseases of the spinal cord.

Pathology.—

1. *Sclerotic* changes are found in the—
   (1) Motor nerve *nuclei* of the medulla.
   (2) Motor nerve *roots* directly connected with the bulb.
2. Degenerative changes are found in the—
   (1) Trunks and motor endings of the glosso-pharyngeal, spinal accessory, and hypo-glossal nerves.
   (2) Atrophy of the tongue, lips, and muscles, supplied by the above nerves.

Authorities differ as to *how* the disease commences. Some say that the lesion first starts in the nerve *cells*; others say that the primary lesion is a subacute or chronic inflammation of the *neuroglia*, which ultimately causes atrophy by pressure. Bramwell believes both views are correct—*i.e.*, that cases beginning in either way are met with.

Symptoms. — Obviously, with so widespread a lesion, the symptoms must vary with the nuclei involved; also, whether associated or not with spinal or basal disease. Yet, in spite of

these facts, a fairly typical picture of glosso-labial paralysis may be presented. Tabulated, the principal symptoms are—

1. *Impairment of articulation* due at first to defective movements of the tongue, but later, to the atrophy of the lips; consequently, defective pronunciation of the letters involving the tip of the tongue is first most marked—*i.e.*, the letters T, E, D, or the exclamation SH!; later, OO, W, etc., or articulations which involve the lips become defective. Finally, *speech may be entirely abolished.*

2. *Difficulty in swallowing.*

3. *Symptoms indicative of paralysis of the palate* — *i.e.*, nasal twang of voice, regurgitation of fluids through the nose.

4. *Dribbling of saliva from the mouth.* This is a most marked symptom, and the patient is continually wiping the secretion away. Possibly more saliva is secreted than normally, but this symptom may be due to deficient deglutition, so that saliva is not *swallowed.*

5. *Symptoms indicative of paralysis of the larynx* are lowering of the pitch of voice, aphonia, etc. When the superior-laryngeal nerve is paralysed, particles of food get into the lung and excite a fatal pneumonia.

6. *Symptoms indicative of paralysis of the cardiac centre* are paroxysmal attacks of dyspnœa, a sensation of tightness across the chest, irregular action of heart, etc.

The patient has usually a most sad expression, which is a contrast to his often buoyant spirits. The condition of the tongue is very characteristic when the disease is well marked. Its muscular tissue is much atrophied, and the mucous membrane hangs in sack-like folds, wrinkled and covered with a dirty yellowish fur. It is tremulous, lies helpless in the mouth, and is only moved with great difficulty, hence the collecting of food between teeth and cheeks. The muscles of mastication may be also involved (showing implication of the fifth nerve).

Prognosis. — Invariably fatal. Death may occur from emaciation, but is more often due to the various complications that arise, especially pneumonia.

Treatment.—A mild course of galvanism, combined with careful hygiene are the only remedies likely to influence the duration of this disease.

Barium chloride 2 gr doses

Dr Byrom Bramwell's clinical atlas may be consulted with advantage for all that is known of this disease.

DISEASES OF THE BRAIN.

Before beginning the study of cerebral disease, we will first consider a few of the elementary anatomical and physiological facts; without a knowledge of such facts brain diseases are quite incomprehensible.

The brain proper, consists of two large hemispheres, partly separated from each other by the great longitudinal fissure; but, bound together below by various commissures.

Externally, each hemisphere is covered with grey matter, named the cortex; which is mapped out by furrows into a series of folds, termed convolutions. These convolutions are of the highest importance; absent in the lower type of animals, they become gradually more marked in the higher animal, well marked in monkeys, but only seen in perfection in the *highly-educated adult human brain.*

Of the *Fissures* of the hemispheres the largest and most evident subdivide the surface of the cerebrum into lobes, and may be called *interlobular*; the smaller fissures—*intralobular*—divide the lobes into convolutions, which, in most cases, have received definite designations.

The *Interlobular Fissures* are—

1. The *fissure of Sylvius.*
2. The *fissure of Rolando.*
3. The *parieto-occipital fissure.*

The *Lobes* of the cerebrum are five in number; four are bounded by the interlobular fissures, and take their names from the bones of the skull in relation to which they lie.

They are—

1. The *frontal.*
2. The *parietal.*
3. The *occipital.*
4. The *temporo-sphenoidal.*

The fifth lobe—the *central lobe*—(*insula or isle of Reil*) is not in contact with the bones of the skull, *but is hidden within the fissure of Sylvius*, the margins of which must be separated in order to see it.

The *external* convolutions of these lobes are—

1. Of the Frontal lobe—

   Ascending
   Superior
   Middle
   Inferior (or Brocas) } Convolutions.

2. Of the Parietal lobe—

   Ascending parietal
   Superior parietal } Convolutions.

3. Of the Occipital lobe—

   Superior
   Middle
   Inferior } Convolutions.

4. Of the Temporo-sphenoidal—

   Superior
   Middle
   Inferior } Convolutions.

The convolutions seen on the *mesial* surface of the brain are—

Gyrus fornicatus.
Marginal.
Hippocampal (uncinate).
Dentate.
Quadrate (Præcuneus).
Cuneus.
Paracentral lobule.

INTERIOR OF CEREBRUM.

Internally the cerebrum consists of white matter, and ganglionic masses of grey matter. The solid mass of white matter *above* the corpus callosum is termed the *centre ovale.*

Below the corpus callosum is an irregular and somewhat T shaped cavity, divided by septa into smaller spaces, termed ventricles. The ventricles communicate with each other by variously named canals — thus, the lateral ventricles communicate with each other and the third ventricle, by the foramen of Munro; the third and the fourth ventricles by the "iter" or aqueduct of Sylvius; the fourth ventricle and the sub-arachnoidal space by the foramen of Majendie.

Basal Ganglia are masses of grey matter situated at the base of the brain, viz.—

1. The *Corpora Striata*, consists of two portions—
   (1) Caudate nucleus = intra-ventricular portion.
   (2) Lenticular nucleus = extra-ventricular portion.

2. *Optic Thalami* contain sensory and optic fibres. The *upper* part of each thalamus appears in the lateral ventricles. The *under* surface rests upon the crura cerebri. (Between the lenticular nucleus externally, and the caudate nucleus and optic thalamus internally, lie the mass of white matter termed the internal capsule. See page 61.)

3. *Claustrum* is a narrow band of grey matter outside the lenticular nucleus. Function unknown. (See page 62.)

4. *Corpora Geniculata* are masses of grey matter in connection with the optic tracts.

5. *Corpora Quadrigemina* contain sensory fibres, and are also implicated in the movements of the eye.

6. *Pineal Gland.* Function unknown.

BLOOD SUPPLY OF THE BRAIN.

The arteries of the brain are derived from the two internal carotids, and the two vertebral arteries.

The branches are practically arranged in two sets, viz. :—

The Cortical group—(anterior, middle, and posterior cerebral arteries).

The Basal group—(comprising the circle of Willis, and the central arteries passing from it.

The circle of Willis may be represented in tabular form.

In front
ANTERIOR COMMUNICATING.

On each side.

ANTERIOR CEREBRAL.	ANTERIOR CEREBRAL.
INTERNAL CAROTID.	INTERNAL CAROTID.
POSTERIOR COMMUNICATING.	POSTERIOR COMMUNICATING.

Behind the
TWO POSTERIOR CEREBRALS,
BRANCHES of the
BASILAR.

So many pathological conditions result from cerebral hæmorrhage, that special attention should be directed to the course of the middle cerebral artery, branches of which so often rupture, that it is really *the* artery of hæmorrhage.

Middle Cerebral or Sylvian Arteries are the largest branches of the internal carotid, and pass upwards and outwards in the fissure of Sylvius till they reach the surface of the island of Reil, where they ramify in the pia mater, forming part of the cortical system of arteries; they anastomose freely with the anterior and posterior cerebral arteries. Other branches, furnished through the anterior perforated spot to the corpus striatum, are all terminal arteries and belong to the "ganglionic system" of branches. They are (1) the lenticular; (2) the lenticulo-striate; (3) the lenticulo-optic.

Cerebral Veins.—The veins do not accompany the arteries, but open into the various sinuses in the dura mater. They are arranged in two sets.

1. *The Superficial set*, which open into the superior longitudinal, the lateral, and cavernous sinuses.
2. *The Deep set*, which gather the blood from the *interior* of the brain, and empty into the straight sinus.

The special characters of the cerebral circulation are—

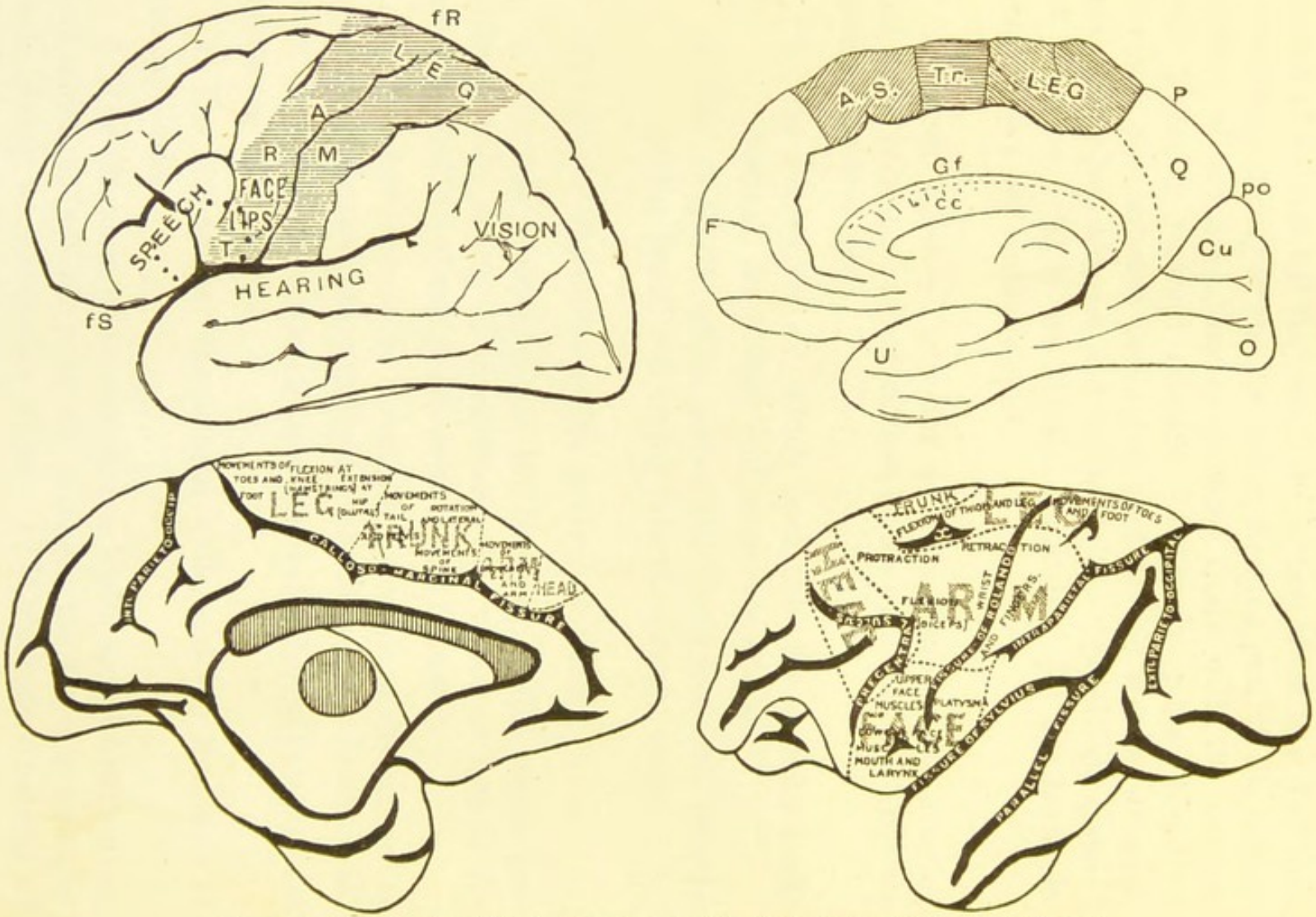
1. The free anastomosis at the circle of Willis, which provides a ready supply of blood from other vessels in case of the sudden blocking of any of the more direct channels.

2. The tortuous course through bony canals of the arteries as they enter the skull, thus mitigating the force of the heart's beat.

3. Their ramifications in the pia mater before entering the substance of the brain.

4. The thinness of the arterial walls, and the smallness of the capillaries.

5. The existence of venous sinuses which are without valves, and which do not run with the arteries; the larger arteries, in fact, having no companion veins. ("Whitaker's Anatomy of the Brain and Spinal Cord.")



Diagrams showing the Principal Centres in the Brain (after Horsley and Beevor).

SUMMARY OF THE FUNCTIONS OF THE BRAIN.

It has long been known that the cerebrum contains the highest nerve centres—viz., those centres whose activity is associated with volition, intelligence, thinking, consciousness, and analysis of sensation, etc.; but, it was not proved until recently (1882), that the cortex is sensible to *direct* excitation. Hitzig and Fritsch in Germany; Ferrier, Horsley, and others in England, have not only proved that the *cortex* itself is sensible to irritation, but, in addition they have shown that the brain does not act as a whole in all its various functions; indeed, on the contrary, certain parts have special duties allotted to them; in other words, "*Stimulation of certain areas cause definite and particular movements, sensations, etc.*"

The Localisation of such areas are briefly—

1. *The Frontal lobes* which are concerned in the higher psychical functions.

2. *The Rolandic area*—associated with motor functions.

3. *The Occipital lobes* which have to do principally with sensation.

Tabulated, we have the following centres—

1. The motor centres for the *legs*, *arms*, *face*, *lips*, and *tongue* (in order from above downwards), in the region of the ascending frontal and ascending parietal convolutions.

2. The centre for *speech* is in the *left* inferior frontal convolution. (If the person be *left* handed, the speech centre is in the *right* convolution.)

Sensory Centres.

3. The centres for *sight* are in the occipital lobe and angular gyrus (see "Optic Nerve.")

4. The centres for *smell* and *taste* are in the hippocampal region in the temporal lobes.

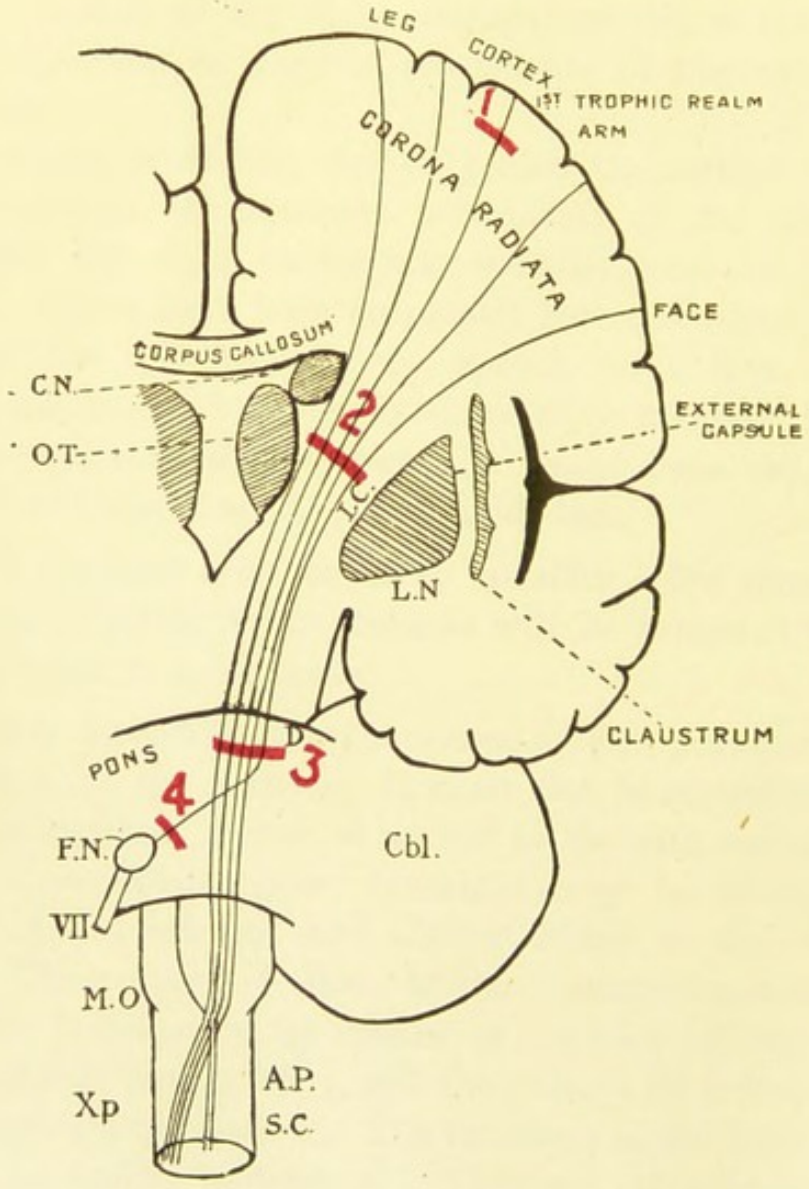
5. The centres for *hearing* are in the superior temporo-sphenoidal lobes.

The results of stimulation of these centres are seen on the *opposite* side of the body, though sometimes they are bilateral (especially as regards movements of the eyes, trunk, and mouth).

In the case of speech, not only does the cortical centre of one side govern the intricate movements of the mouth and throat, but the right centre may actually loose its power or function. Cases have been frequently recorded where, through disease of the *left* frontal lobe, speech *for a time* has been entirely abolished, but, regained in time *through the right centre re-acquiring its function*— in these cases the patients have had to learn language as a child does.

It only remains now for us to consider a few more simple facts, then, I think, brain diseases will be rendered fairly intelligible, even to a beginner.

Note that *irritation* causes increased action; *paralysis* abolish the functions of the centres. It must also be remembered that though the *cerebral cortex alone* has to do with sensation and voluntary movement, these functions may be abolished by interrupting the afferent and efferent fibres to and from the cortex. The cortex, in fact, is the commander-in-chief; it receives its information by means of inferior officers — basal ganglia, afferent nerves, etc.; and then issues its *orders through a similar efferent mechanism.* The functions of the basal ganglia will now be readily understood. They are subordinate centres conveying and receiving orders from the cerebrum, but they are also able to act as it were, on their own responsibility as regards certain complex reflexes and co-ordination of movements; thus, the optic thalami contain a large number of sensory fibres passing to the brain, and also a number of *filaments in connection with vision.* Lesions of the optic thalamus, or of the



DIAGRAMMATIC DRAWING intended to show the effect of lesions interrupting the motor fibres at various levels. Observe a lesion at 1 involves but few fibres; at 2, a much larger number. Note the relations of the facial nerve at 3 and 4. (See Hemiplegia and Bell's Paralysis.)

corpora striata do not produce *entire* loss of sensation, but only in *proportion to the number of sensory fibres interrupted* en route *to the brain.* Indeed, when a lesion in this neighbourhood is followed by loss of sensation, it is probably *due to implication of the posterior limb of the internal capsule.*

Summary of the effect of lesions (destructive and irritative) from the cortex to the spinal cord.

The Cerebral Cortex.—Destructive lesions produce paralysis of the opposite muscles of the body ; as the motor fibres are cut off from the first trophic realms secondary degeneration occurs, causing the paralysis to be of a *spastic nature. Note* that the fibres are spread out in a fan-like manner, so, consequently, the lesion may only affect a few fibres going to one or more groups of muscles.

Irritative lesions are described under "Jacksonian epilepsy."

Centrum Ovale.—A lesion will involve a larger number of fibres than in the cortex.

Internal Capsule.—The motor and sensory fibres here converge to the *posterior limb.* A lesion usually produces *typical* hemiplegia (which see).

Crura Cerebri.—A lesion produces a similar hemiplegia, but owing to its relations with the *third nerve*, the *ocular* muscles may be paralysed on the *same* side, *i.e.*, on the same side as the lesion.

Pons.—Note that here the sixth and seventh nerves may be paralysed in the resulting hemiplegia (see seventh nerve). Remember the crossed nature of the paralysis before-mentioned.

Clinical considerations—

Lesions of the cranial contents cause either *irritation* or *paralysis* of the nervous apparatus, motor, sensory, or reflex. Irritation of motor structures is shown by *muscular twitchings* or *spasms ;* irritation of sensory parts causes *pain* and *hyperæsthesia ;* irritation of reflex nerve centres leads to *increased reflex action.*

Motor paralysis is estimated by noticing the position of the limbs, the absence of all resistance to passive movements, and stertorous breathing, or flapping of the lips and cheeks with respiration.

Sensory paralysis is recognised by the insensibility of the patient to all external impressions, such as sound, light, pinching, pricking.

Reflex palsy is specially indicated by a fixed condition of the pupils, and the failure of contraction of the orbicularis palpebrarum when the conjunctiva is stimulated.

DIFFUSE IRRITATION.	COMPRESSION.	CONCUSSION.
1. Increased insensibility; patient shows great irritability of temper.	1. Total insensibility.	1. Insensibility, from which patient can usually be partly aroused.
2. Respiration may be quick.	2. Respiration stertorous, slow, and puffing.	2. Respiration feeble, like that of a person in a faint condition.
3. Pulse quick.	3. Pulse full, slow, laboured.	3. Pulse weak, irregular, and often frequent.
4. Pupils contracted.	4. Pupils widely dilated, or sometimes one dilated and the other normal or contracted.	4. Pupils variable, but usually sensitive to light.
5. Reflexes exaggerated.	5. Sphincters may be paralysed, but bowels are torpid.	5. Bowels relaxed, but sphincters not paralysed.
6. Increased irritability of organic reflexes.	6. Bladder paralysed. Consequent retention of urine.	6. Bladder can expel water.
7. Comes on some time after injury, or insidiously from disease.	7. Does not usually appear at moment of injury, but often preceded by signs of irritation.	7. Comes on instantaneously and passes off gradually after injury.
8. Vomiting common.	8. Vomiting *may* occur —not usually.	8. Vomiting as recovery is taking place.

We have thus seen that the brain is the initiator or starter of impulses on the one hand, and exercises a controlling or inhibitive power on the other hand. In other words—on the normal exercise of brain function depends the healthy activity of every organ and structure in the body; but the carrying out of this function *is dependent on a perfect maintenance of the anatomical and physiological relations of the component parts.* Lastly, remember that just as the commander-in-chief of an army may be temporarily absent without any great disadvantage, so

is it possible for life to be maintained, and, indeed, a fair standard of health to persist for a time, after the higher *centres have been destroyed or abolished by disease.* Such a condition, however, can only last for a limited period, for the inhibitive power being removed, the reflexes run riot, harmony is replaced by anarchy, premature decay supervenes, and often speedy death terminates a miserable existence. Paralysis of the insane furnishes an example of this.

CEREBRAL HÆMORRHAGE.

Ætiology.—It occurs most frequently in men in advanced life. Amongst the more important pre-disposing causes are—

1. A certain type of build—viz., stout, plethoric men, with short, thick necks.

2. Certain diathesis,—hæmophilia, gouty, etc.

3. Certain occuptions—viz. (1) butchers, publicans, by producing arterial changes through excessive consumption of nitrogenous food and alcohol; (2) carters, hammermen, etc., by producing vascular strain.

4. Certain blood diseases — leucocythæmia, pernicious anæmia, scurvy, Bright's disease, syphilis, etc.

5. Removal of the natural vascular *support*, as in the cerebral softening.

6. Injuries from without the cranium, etc.

The most important immediate factors, however, are—

1. Disease of the vascular walls.—
   - (1) Miliary and other aneurismal dilatations.
   - (2) Atheroma.
   - (3) Fatty degeneration.
   - (4) Waxy degeneration.
2. Vascular strain.

Site of the Hæmorrhage.—The most common form of cerebral hæmorrhage is *into the substance* of the brain, and is usually due to rupture of the lenticulo-striate branch of the middle cerebral artery which supplies (as we have seen) the basal ganglia. The explanation why this artery is so frequently ruptured, is probably due to the fact that it is the most direct branch of the middle cerebral, which is in turn the most *direct* branch of the internal carotid.

Anatomical Changes.—If the hæmorrhage be severe it tears up the brain tissue, or it may destroy the basal ganglia, and bursting into the lateral ventricles distend them, and flow through the aqueduct of Sylvius into the fourth ventricle. The hæmorrhage, however, may be small. The subsequent changes in the effused blood are of the utmost importance, viz.:—

1. *Changes in the clot*—
   (1) Retraction and exudation of serum (which is partially absorbed).
   (2) Discolouration of the clot through infiltration of leucocytes, and partial breaking-down of red corpuscles.
   (3) Formation of hæmatoidin crystals. Hæmatoidin is a form of reduced hæmatin which contains no *iron*. (It cannot be formed artificially.)
   (4) Formation of a fibrous scar.

2. *Changes in the tissues around*—
   (1) Irritation causing hyperæmia.
   (2) Diapedesis of leucocytes, and proliferation of the tissue cells.
   (3) Increased fibrous formation, forming a capsule around the clot.

The above are favourable terminations; but, too frequently the inflammatory reaction is intense, the vessels become

thrombosed, and the brain substance thus cut off from nutriment, rapidly breaks down (red softening); or the process may be less acute, but also attended with purulent degeneration (white softening), in such a case a "central abscess" is often thus produced.

3. *Changes in the fibres* interrupted by the hæmorrhage, and thus cut off from the first trophic realm (cortex). Secondary descending degeneration occurs of the motor tracts on the affected side.

Summary.—After cerebral hæmorrhage, if the patient survives the primary shock (the apoplectic fit), he has to run the gauntlet of—

1. Reaction. (Inflammation of brain substance, softening, and abscess formation *may* occur.)
2. Resulting motor-paralysis (hemiplegia).
3. Secondary degeneration of motor fibres, and possible involvement of important nerve nuclei.

Symptoms.—There may be premonitory warnings of the attack, such as giddiness, headache, etc. The attack may occur when the patient is resting or asleep, but much more frequently is it directly attributable to an over-loaded stomach, a severe strain, coughing, or the exertion in running to catch a train. Apoplexy is not synonymous with cerebral hæmorrhage; still, the condition which supervenes on such a lesion is generally summed up in the description of an—

Apoplectic Fit.—The patient is suddenly seized with severe pain in the head, feels faint, giddy, and quickly falls into a state of collapse, which in a short time passes into a comatose condition. The face is flushed, the pulse full and tense, breathing stertorous, and cheeks puffed out. The snoring noise is due to the paralysed tongue and palate falling back, and impeding the entrance of air. The pupils are dilated or irregular. The limbs at first are flaccid, but soon become somewhat rigid (so-called *early* rigidity). The skin reflexes are lost, but the deep

reflexes are increased. The head and eyes are usually turned to one side—*i.e.*, patient looks towards the lesion *away* from the paralysed side (conjugate deviation). When the case is going to end fatally, the coma increases, temperature falls, and Cheyne-Stokes' respirations usher in the fatal end. In favourable cases the primary disturbance is not so grave, and the patient may regain consciousness in a few hours, or even after two or three days. Recovery, however, is rarely complete, but usually with more or less paralysis or typical hemiplegia.

HEMIPLEGIA.

By hemiplegia is meant paralysis of the face, arm, and leg of the opposite side. The reason why the trunk and eyes are not usually affected, according to Broadbent, is briefly as follows:—"That when groups of muscles are usually associated together, there must be commissural fibres running between the centres in either hemisphere. These fibres are so well drilled by constant stimulation, that if one centre be destroyed, the opposite centre is equal to the task of commanding the well disciplined fibres on both sides, and consequently both sets of muscles act harmoniously."

Causes. — Though most commonly the result of a free hæmorrhage, embolism, or thrombosis, hemiplegia may be caused by depressed fractures, tumours, etc., involving the motor fibres anywhere between the cortex and the medulla; the symptoms will also vary with the sites of such lesions.

Symptoms.—When the hemiplegia is not preceded by an apoplectic fit, the patient often first becomes aware of anything wrong by finding on awakening from sleep—

1. Loss of power on one side, or
2. Difficulty in speaking, or even loss of speech.

Unconsciousness may develop and pass into complete coma, or, instead of unconsciousness there may be only a feeling of cloudiness or bewilderment, which may or may not develop into coma.

The arm is usually paralysed to a greater degree than the leg. The tongue is not put out readily, and deviates towards the *paralysed* side. Sensation is rarely affected to a marked extent. The facial paralysis has already been described (page 98), but we may again note it implicates the *lower* part of the side affected, thus differing from true Bell's paralysis. As time wears on, certain important symptoms appear, as a result of the secondary degeneration of the motor fibres cut off from their trophic centres. The limbs before flaccid, now become rigid. The arm is flexed at the elbow, and resists extension; frequently the wrist and fingers are also flexed. Atrophy does not usually occur, but there may be wasting consequent on non-use. The reflexes are exaggerated, and ankle clonus is often obtained.

The later changes are—peculiar tremors of the affected limbs, enlargement of the joints, and inability to maintain one position of the fingers and toes (athetosis), post hemiplegic chorea, etc. Remember the peculiarities before mentioned, when the lesion is in the crus (third nerve), or pons (sixth and seventh nerves).

Diagnosis must be made from hysterical paralysis, which often simulates very closely the organic lesion.

EMBOLISM AND THROMBOSIS.

Instead of a free extravasation of blood we may get blocking of blood-vessels through detached clots (embolism), or a local clotting of blood (thrombosis). The following table shows the more important points.

Diagnostic Table.

CEREBRAL EMBOLISM.	CEREBRAL THROMBOSIS.
Causes.— Most commonly associated with valvular lesions of the heart, aneurisms, or a suppurating thrombus.	*Causes.*— 1. Atheroma and morbid states of the arteries, as seen in chronic Bright's disease, syphilis, pyæmia, etc. 2. Pressure upon the veins or arteries.
Sex.— Most frequent in females.	*Sex.*— Equally frequent in both sexes.
Onset.— Sudden — no prodromal symptoms.	*Onset.*— Gradual—with prodromal symptoms.
Convulsions.— Rare.	*Convulsions.*— Convulsive attacks are common, and may exist for some time in *venous* thrombosis.
Paralysis.— A sudden hemiplegia of the right side, *with aphasia.*	*Paralysis.*— Paralysis may occur suddenly in arterial thrombosis, but aphasia is not so common.
Consciousness.— Not *usually entirely* abolished.	*Consciousness.*— Profound coma supervenes.

Lastly, we must remember that hæmorrhage, embolism, or thrombosis, may be confined to the small vessels of the cortex alone.

In cortical hæmorrhage—

1. Consciousness is seldom completely lost at the onset of paralysis.
2. The resulting paralysis is of a "monoplegic" type.
3. The paralysis is often transitory.
4. Rigidity of affected muscles comes on earlier.
5. Prognosis is much more favourable.

Anatomical Changes following thrombosis and embolism will vary according to the site and size of blood-vessels affected. Briefly, the ultimate results are degeneration and softening of the parts supplied by the affected artery. Blocking of a terminal artery causes the affected area to look slightly paler at first; then extravasation from the engorged veins or collateral circulation may take place and cause an infiltration of blood. The resulting softening may be of the red, yellow, or white types, and the clot undergoes the changes before described. Two things the student must remember—

1. If the clot be of a *septic* nature, an abscess is likely to form.

2. If *not* septic, it is surprising how slow the symptoms dependent on softening develop.

Blocking of particular arteries.

1. *Vertebral.*—Rare, left more common than right. The symptoms are those of acute bulbar paralysis.

2. *Basilar.*—Bilateral paralysis with *rise of temperature.*

3. *Posterior Cerebral.* — Affections of vision and hemianæsthesia (posterior portion of internal capsule).

4. *Middle Cerebral.*—Hemiplegia with aphasia.

5. *Anterior Cerebral.*—Grave interference with the higher intellectual faculties.

Treatment of Cerebral Hæmorrhage.—Place the head high, and put ice to head, and warmth to feet; administer an enema, calomel grains v. on the tongue, or a minim of croton oil. Venesection should be performed if the pulse is full and of high tension; if, on the other hand, the pulse be weak, or we are fairly certain the symptoms are due to thrombosis or embolism, it is best not to resort to bleeding. The resulting paralysis must be treated on the general principles detailed in the chapter on Chronic Nerve Lesions.

APHASIA.

Aphasia is a morbid condition consequent on a cortical lesion whereby speech, writing, or reading may become impaired either from (1) an inability to co-ordinate the necessary movements involved in speech; or (2) from a defective interpretation of sounds or visual impressions.

To understand the various forms of aphasia it is necessary first to consider the factors employed in expressing our thoughts. Language is *gradually* learned. For instance, a child has first to learn the movements necessary to articulate the word. Having learned the word, he then associates it with a particular something he has seen or heard. Later, he learns the alphabet—that is, he invests certain letters with definite sounds and meanings, and as his vocabulary increases he can express ideas in speech, and finally spell and write. Thus, intelligent and rational speech, though apparently simple, involves many complex processes. It requires—

1. The aid of memory to *co-ordinate the necessary ideas* formed by incoming impressions derived through hearing, seeing, and in the case of the blind, through the sense of touch.

2. A mechanism by which these ideas can be spoken and written.

In short, speech involves a healthy continuity between—

The chief motor centre in the left inferior frontal lobe, and the visual and acoustic centres on the one hand, and—

The chief motor centre and the muscles employed in speech, writing, or reading aloud.

Lesions of the occipital lobe cause *word blindness* — *i.e.*, the patient can see print, but cannot read it.

Lesions of temporo-sphenoidal lobe cause *word deafness* — the patient hears, but does not understand words.

A large number of forms of aphasia are described, but they cannot be gone into here. Two forms will only be described—*i.e.*, sensory and motor aphasia :—

MOTOR APHASIA.	SENSORY APHASIA.
Patient is almost speechless, but may manage words like oaths, yes, or no.	Has a slightly greater command over words in a purely automatic manner, and may make the same stilled remark to all questions.
Understands what is said to him.	Cannot understand what is said to him (word deafness).
Cannot repeat words.	Cannot repeat words.
Cannot write words *but can copy.*	
Is aware of his errors—he can recall words but cannot utter them.	Is unaware of his mistakes—he cannot recall to his mind the meaning of words.

TUMOURS IN THE BRAIN.

The most important are—

1. *Tubercle.*—They may vary in size from a millet seed to a hazel nut. Usually they are multiple, and most common in the cerebellum and about the base of the brain. Histological characters are the same as already described under tuberculosis.

2. *Glioma, or neuro-glioma.*—They are usually dense, firm tumours, but *may be soft and vascular.* Histological characters consist of round or oval nucleated cells, with branched processes, forming a network. *Often enormous, single-nucleated, spindle-shaped cells are present.* They are usually innocent tumours, and may remain for years without creating very marked symptoms.

3. *Sarcoma*—attacks the membranes and pons—may attain a large size.

4. *Sarco-glioma*—found chiefly in the retina. They differ from simple glioma in a tendency to assume a malignant character.

5. *Carcinoma*—rare—secondary to cancer elsewhere.

6. *Cysts*—may be the result of—

(1) Hæmorrhage.
(2) Congenital defects.
(3) Hydatids.

Symptoms. — They vary according to the site, size, and nature of the tumour. The more constant symptoms are—

1. *Headache.*—May be diffuse, and dull aching in character, but is much more often of an acute stabbing nature, persistent, and localised.

2. *Vertigo.*—A most marked symptom, especially when the tumour affects the cerebellar region.

3. *Vomiting.*—Very persistent, and occurs whether food be taken or not.

4. *Double Optic Neuritis.*—Choked disc (is almost pathognomonic).

5. *More or Less Mental Disturbance.*—Sometimes absent, but the patient is nearly always either highly emotional or dull and apathetic.

6. *Motor Disturbance.*—There may be slight paralysis of a monoplegic type, but much more frequently convulsions of a Jacksonian type occur.

Whilst the above are the cardinal symptoms, it is easy to see that the symptoms may be very numerous, and on the other hand, very obscure. Inco-ordination, spastic rigidity, nystagmus, and hydrocephalus are frequently present.

Diagnosis.—Having well considered the various symptoms, if they are *pronounced*, little difficulty will be experienced on approximating the site of the tumour; but, when *obscure*, the diagnosis will have to be made from cerebral softening, cerebral abscess, uræmia, and hysterical paralysis, etc.

Treatment—

Medical. — If syphilitic, iodide of potassium in heroic doses; other forms are practically unamenable, at least to any known medicinal treatment.

Surgical.—Trephine and remove if possible, but few cases are available for this treatment. Consult a surgical work for details.

Though so many cases cannot be *permanently* benefited by treatment, everything should be done to make the patient's life bearable, by relieving pain, etc., with opiates.

MENINGITIS.

Inflammation of the meninges of the brain is of two types, *i.e.*—

1. The *simple or traumatic form*, attacking most frequently the convexity of the brain.

2. The *tubercular form*, which principally affects the base of the brain, and is usually part of a *general* tuberculosis.

THE SIMPLE FORM.

Causes.—Injuries of all kinds to the skull, extension of inflammation from other parts—*i.e.*, disease of the middle ear, irritation from tumours, minute hæmorrhages, etc., or as a complication in fevers, syphilis, etc. The inflammation may be most marked in one membrane, and thus, when the dura mater is principally involved, it is called pachymeningitis; when the arachnoid and pia mater are most affected, it is termed lepto-meningitis. Such distinction is of doubtful value in the acute form, though it is well to remember such forms in the chronic variety. Bear in mind also that the dura mater acts as the periosteum to the inner table, so therefore it is easy to see lepto-meningitis is most common when the cause is from within the cranium; pachymeningitis when the causes are from without.

Symptoms.—They are divided into three stages.—

1. *Premonitory.*—Headache, more or less constant; vomiting and restlessness.

2. *Stage of Irritation* is marked by febrile symptoms, great intolerance of light, contracted pupils, exaggerated reflexes, and convulsions. The patient lies curled up in a characteristic manner.

3. *Stage of Compression.*—Temperature tends to fall, irritability is replaced by stupor, and convulsions by paralysis. Pupils now become uneven or dilated. Optic neuritis may rapidly develop. The peculiar cephalic cry is a constant symptom. Cheyne-Stokes' breathing, involuntary escape of fæces and urine, and stertorous breathing, usher in death, which often takes place very soon after the onset of grave symptoms.

Pathology.—

1. Hyperæmia of the meninges which become swollen and injected.
2. Exudation of lymph.
3. Effusion, which rapidly becomes purulent.

THE TUBERCULAR FORM.

The symptoms of the tubercular form of meningitis are also divided into three stages—

1. *Prodromal.*—The child usually shows more or less definite symptoms of the tubercular diathesis, such as, emaciation, want of appetite, constipation, alternating with diarrhœa; irritability of temper, and headache, perhaps form the more common features, until *definite* symptoms of the meningeal affection arises.

2. *Irritative Stage.*—The symptoms are similar to those described under the simple variety, but the head is usually

more retracted and neck more rigid; abdomen is hollowed out or boat-shaped, temperature oscillates, internal strabismus, and often marked vaso-motor paralysis (*tache cérébrale*).

3. *Compression Stage.*—The symptoms that accompany coma develop, and death may take place in from ten days to six weeks from the onset of acute symptoms.

Pathology.—The inflammatory conditions associated with the presence of tubercle, viz.—

1. Invasion of the bacilli into the meninges (pia mater principally), and formation of tubercles.

2. Irritation set up by the tubercles. The tubercles may then go through the stages of (1) softening, (2) bacillary liquefaction—(formation of a greenish pus,) or (1) fatty degeneration, (2) caseation, with very little lymph, (3) calcification (rare).

The down-grade process seems to commence in the perivascular tissue of the Sylvian and other arteries at the base of the brain, hence the *ventricles are often distended*, and the brain substance flattened up against the skull. Hydrocephalus has been produced by thus obstructing the veins of Galen.

Treatment—

Simple Meningitis.—Remove any obvious cause; darken the room; shave the head and apply ice; administer a calomel purge, bromides, hyoscyamus, and potass. iodid. Support the patient with fluid nourishment, and prevent bed-sores and over distension of bladder during the later stages.

Tubercular Meningitis.—This form needs no different treatment after symptoms are developed, but during the prodromal stage, everything should be done to combat the tubercular condition by careful hygiene, diet, etc., with the administration of cod-liver oil, fresh air, etc.

DIAGNOSTIC TABLE.

SIMPLE.	TUBERCULAR.
Age.—Any age.	*Age.*—Young children.
Cause.— Injury or local causes, fevers, etc.	*Cause.*— No local cause, but symptoms of tubercle elsewhere.
Course.— Short.	*Course.*— Longer than simple, especially the prodromal stage.
Convulsions.— They may be present.	*Convulsions.*— *Common*, even during the compression stage, often precedes death.
Abdomen.—Nothing marked.	*Abdomen.*—Markedly retracted.
Pathology.— 1. That of simple or suppurative inflammation.	*Pathology.*— 1. That which is associated with the presence of tubercle, and formation of peculiar greenish pus.
2. Attacks convexity of brain.	2. Attacks the base of brain.
3. Ventricles not distended.	3. Ventricles are distended, and may cause hydrocephalus.
Prognosis.— Almost hopeless.	*Prognosis.*— Depends on cause and extent.

DISSEMINATED SCLEROSIS.

Disseminated sclerosis, or the so-called "insular" paralysis (want of insulation of the axis-cylinders being referred to as a cause of this peculiar affection,) is a disease characterised pathologically by scattered patches of sclerosis throughout the central nervous system. The condition may be most marked in the brain, or in the spinal cord; but the *cerebro-spinal* variety is much more common.

Ætiology occurs in youth or middle age; both sexes are liable to the affection. Exposure to cold, injuries, and mental worry are possible exciting causes.

Pathology.—Patches of sclerosis, sharply defined, and varying in colour from pink to ashy grey; some hard and leathery, others soft, are found scattered throughout the white matter of the cord and surfaces of the medulla and cerebrum.

Symptoms.—With such wide spread pathological changes, it is practically impossible to say what symptoms may, or may not be present, as the symptoms will of course depend on the site and extent of the sclerosed areas. Taking a typical case however, we usually find the following symptoms more or less marked :—

1. Impaired Speech.—The person has a slow, monotonous way of pronouncing every syllable distinct, like the staccato delivery of music.

2. A peculiar tremor of muscles, which become very marked when voluntary movements are attempted; the movements of the hands are specially embarrassed by the tremor, and directed with difficulty.

3. Nystagmus, worse on trying to fix the eyes.

4. Impaired sensibility.

5. Impaired intellectual functions, as loss of memory, etc.

6. More or less paresis with spasm.

7. Peculiar Gait.—There is marked inco-ordination of not only the legs, but *arms, trnnk, and head.* When walking, the patient shoots suddenly forward or to one side, and may fall, or bruise himself by bumping against various obstacles.

8. Occurence of peculiar apoplectic attacks, from which the patient recovers in one or two days.

Diagnosis.—The peculiar gait and speech usually renders the diagnosis easy, but remember that the dissemination of the lesion might be due to an extension of a hitherto *local* lesion.

PARALYSIS OF THE INSANE.

(G. P.)

A condition marked by progressive derangement of the mind accompanied by paralysis.

Ætiology.—The disease seldom occurs before twenty years of age—most frequently about forty. It affects the rich more than the poor. The most important causes are excessive study, drinking, and syphilis.

Pathology. — The post-mortem appearances vary greatly. In some cases little anatomical change has been discovered; but, usually the following conditions exist :—

1. Thickening of the inner table of the skull, which is marked by Pacchionian bodies.
2. Thickening of the membranes.
3. Degeneration of cerebral blood-vessels.
4. Patches of sclerosis throughout the white matter of the cord and brain.
5. Atrophy of the frontal lobes.
6. Distension of the ventricles by fluid.

Symptoms. — It is impossible to give a typical clinical description, as individual cases vary so much. Perhaps it is best to describe three stages :—

First Period marked by changed mental conditions such as—

1. Irritability of temper, jealousy, hallucinations.
2. Loss of memory.
3. Defective speech—patient is unable to repeat similar or many syllabled words.
4. Exaltation of ideas, and loss of judgment. Patient may fancy he is rich, or may buy a stud of horses, order a banquet, etc.

Second or Epileptiform Period.—

1. Patient has a series of convulsions which render his mental condition worse than before.
2. Symptoms of paralysis appear ; first, in the execution of fine movements, such as writing, etc.; later, the paralysis involves the face and tongue, or paresis of the limbs and common sensibility develops.

Third or Paralytic Stage.—

The mind finally totters, even to complete dementia. The rectum and bladder act involuntary. Bed sores form, and grave interference with the vital centres, or pulmonary affections soon put an end to a pitiful existence.

Whilst we have described three stages and sketched a clinical course, cases occur where the symptoms are chiefly ataxic, *plus* mental impairment ; or mental, *plus* symptoms of disseminated sclerosis ; but differential diagnosis or enumeration of the various types cannot be entered into here. Remember, however, that the optic discs *are not often affected*, and *wasting is rare ;* the patient often being fat, even to the end.

Treatment.—Put patient under asylum treatment—*i.e.*, use common-sense principles, and by careful supervision, hygiene, and diet, try to prevent the disease progressing. *Rest, and firm, but gentle, control,* do more good than drugs.

HYDROCEPHALUS.

Acute hydrocephalus has already been described under tubercular meningitis. Under the term *chronic* hydrocephalus is meant that form arising from—

1. Congenital malformations.
2. A low type of inflammation attacking the lining of the ventricles during fœtal life or early infancy.

Morbid Anatomy.—

Skull.—Sutures fail to unite, and the skull as a whole does not ossify as in health. A characteristic deformity thus develops —viz., overhanging brow, great increase of the circumference of the cranium *and its disproportion to the size of the face*, open fontanelles.

Ventricles.—They are distended—one, two, or more ventricles may be affected. The lining is granular, and the ependyma thickened. Fluid contains albumen, chloride of sodium, traces of urea, cholesterine, flakes of lymph, etc. The quantity varies —may reach fifteen pints or more.

Brain Substance—Is much compressed, convolutions flattened out, and cortex much thinner.

Cranial Nerves.—Certain nerves may at first be inflamed, and subsequently atrophy, especially the *optic* nerves.

Symptoms. — The size and shape of the head sufficiently indicate the nature of the disease, even at an early period; later, the symptoms that develop are—

1. Arrest of development generally.
2. Impaired digestive functions—distended abdomen, etc.
3. Walking power is slowly gained, or not at all.
4. Mental deficiency, sometimes complete idiocy.
5. Convulsions.
6. Condition of apathy, coma, and death, usually in from five to seven years. Cases have been known to attain the age of thirty years.

Treatment.—Very unsatisfactory, if not hopeless :—

1. Pressure by strips of plaster or elastic bands.
2. Application of iodoform to scalp.
3. Puncturing at various intervals, and drawing off the fluid.

FUNCTIONAL DISEASES OF NERVOUS SYSTEM.

PARALYSIS AGITANS.

Shaking palsy or Parkinson's disease is an affection characterised by rhythmical contractions of certain muscles, progressive weakness and deformity.

Ætiology.—It affects men more than women, and is rarely found in patients under forty years of age. Hereditary; excesses in alcohol and tobacco; gout, etc., are put down as the predisposing causes.

Symptoms.—They are very insidious; insomnia, unnatural irritability and weakness of the limbs generally precede the more characteristic symptoms, which are,—

1. *Rhythmical contraction* of certain muscles of the fingers and arms; the fingers are flexed with the thumb resting against the forefinger, the alternating flexion and extension causes movements like rolling pills. The tremors usually spread to the leg of the same side, and finally trunk and face become affected. The movements at first are checked by voluntary effort or support, but later (like chorea) the movements are actually increased on attempting any voluntary restraint. They cease during sleep.

2. *Weakness of the affected muscles.*

3. *Rigidity and contraction*, causing deformity and peculiar gait. The body is bent forwards, head held stiffly, the "vertebra prominens" stands out in bold relief, and the elbows are flexed at right angles and stand out from the sides. The gait is very characteristic, the first steps are hesitating and slow, but become quick, and the patient appears to trot rather than walk. If the patient be gently pushed

forwards or backwards he is unable to stop himself. The movements forward are called "*festination*," and backwards, retropulsion.

4. *Defect of Speech.*—When the tongue is affected the speech is at first quick, but becomes slow.

5. *Certain subjective symptoms*, hot flushes, rheumatic pains, etc.

The disease runs a very chronic course, and is fatal only through some intercurrent diseases.

Pathology.—Unknown; probably it is an advanced and premature senile change in the cerebral cortex, rendering certain motor points highly irritable, and diminishing at the same time the inhibitive power of the brain.

Treatment.—Unsatisfactory. Arsenic, chloride of barium, sedatives and attendance to hygiene and diet, avoidance of alcohol, etc., are the chief lines to go upon.

CHOREA OR ST VITUS'S DANCE.

Ætiology.—It is most frequently seen amongst female children of the lower classes, between five and ten years of age, and also during the adolescent period. Emotional or hysterical children are prone to this affection, a point which is of great importance when discussing the pathology of the disease. Clinical records show a more or less constant relationship between chorea, endocarditis, and rheumatism. However, there are cases in which neither a history of rheumatism nor endocarditis can be traced.

The **Causes** briefly are—

1. Disturbed mental conditions, brought about by hereditary weakness, excessive study, school strain, and *astigmatism*, etc.

2. Imitating choreic movements (very doubtful).
3. Endocarditis.
4. Rheumatism.
5. Traumatisms.

Pathology.—No constant lesion is found in chorea. Many observers have noticed punctiform hæmorrhages in the cortex, and also embolic plugging of the small capillaries; but such conditions are not invariably found. The embolic condition fits in well with the theory, "That chorea is caused by detached bits of endocardial vegetations which become arrested in the cortical vessels, and there set up irritative changes." At present, however, the pathology may be summed up as follows:—"*An altered condition of the cerebral cortex, in virtue of which, explosions of energy are discharged from the motor area, either through want of inhibitive or controlling power, or as a result of undue irritation and anæmia of the motor cells caused by minute emboli.*"

Symptoms.—

1. Are irregular and purposeless convulsive movements of hands, facial muscles, etc., rendered worse on attempting to execute voluntary acts, or fine movements—such as picking up pins, conveying a cup to the lips, writing, or touching a particular point, etc. Dr Sturges sums up the symptoms "as an extremely exaggerated fidgetiness." The movements cease as a rule during sleep.

2. Altered mental conditions are sometimes most marked; the patient laughing or crying in turns on being spoken to. When these symptoms are present they point strongly towards the "functional character of the disorder."

3. Characteristic attitude when in bed—child lies extended in bed, twisted to one side (pleurosthotonos), and then changes suddenly to an exactly opposite condition.

4. Irregular and rapid action of the heart.

5. Soft systolic murmurs at the apex of the heart.

Treatment.—

1. Dr Goodhart insists upon *rest in bed* as the first and essential point of treatment.

2. Careful attention to the bowels, in order to obviate any reflex irritation from that quarter.

3. Arsenic and cod-liver oil.

As soon as convalescence seems established, allow the patient plenty of fresh air and gymnastic exercises.

EPILEPSY.

A paroxysmal affection characterised by sudden attacks of unconsciousness, with or without convulsions. Two forms will be described under this heading—

1. *Petit mal.*
2. *Grand mal.*

Jacksonian epilepsy differs so much in ætiology, treatment, and pathology, that it will be dealt with by itself.

Ætiology. — The disease most frequently begins in early childhood; probably one-fourth of the cases begin before the child is ten years of age, and *three-fourths* between ten and twenty years of age. *When it begins in adults* look for a *local* cause; probably all such cases are either of a *Jacksonian or hysteroid type.*

Heredity is a most important factor, in so far as children of families in which insanity, hysteria, alcoholism, neuralgic affections, etc., are prone to this affection.

Exciting Causes.—Those put down are—

1. Excessive worry.
2. Puberty.
3. Masturbation.
4. Fright.

It will be seen that these "*causes*" are similar to those of chorea, and such a list is probably the result of anything but

careful analysis. Given, however, a person *born with a marked and distinctly neurotic temperament*, it needs little skill to come to the conclusion, that any undue cause of nervous irritation, ranging from a tight foreskin to absolute debauchery, must be potent factors in producing epilepsy.

Pathology.—At present it is entirely speculative. Reasoning from what we know through direct experiments on the cortex, the great symptoms of epilepsy—*i.e.*, loss of consciousness, convulsions, and coma, point to a disturbance of the higher centres of the cortex; but the explanations given of such disturbances are diverse and many. They are—

1. Diminished *resistance* on the part of the grey matter (Gowers).
2. A paroxysmal nerve storm, consequent on an unstable condition of the motor cells (Hughlings Jackson).
3. Vasa motor changes in the medulla (Van der Kolk).
4. Disturbances of the basal ganglia (Fagge).

Symptoms.—

Grand mal—divided into three stages.—

1. *Aura* or warning, peculiar sensations felt in the stomach, toes; palpitation, or vertigo, etc.
2. *Fit.*—

(*a*) The patient drops down suddenly after uttering a piercing shriek (epileptic cry).

(*b*) *Tonic stage.*—The head is *turned to one side*, the hands clenched, and the muscles of the chest by their contraction interfere with the respirations, so that the preliminary pallor is succeeded by lividity. The pupils are dilated, eyes fixed, and sensibility is abolished. This stage lasts from thirty to forty seconds, and then passes into the—

(*c*) *Clonic Stage.*—Convulsions of the face quickly extend to all the muscles of the body; tongue may be pushed between

the teeth and bitten severely; and the foam at the mouth is then coloured with blood. The face assumes a purplish hue, and the eyes seem to protrude from their sockets. Urine, semen, or fæces may escape involuntarily, and the pulse is often much embarrassed by the muscular contractions. This stage lasts about four minutes or so, and then the convulsions cease. The breathing gradually becomes easier, features regain their natural colour, and consciousness may be quickly recovered; or, what is more common, pass into the fourth stage—namely—

3. *Post-epileptic condition.*—Patient falls into a comatose stage, succeeded by a natural sleep. Rarely in this form are there post-epileptic maniacal manifestations.

Petit mal, or minor epilepsy.—

In this form the patient is suddenly seized with unconsciousness, the eyes become fixed, speech incoherent, but there *are no convulsions.* The attacks vary much in severity. Sometimes they are so slight as not to be noticed by others than the patient. This form of epilepsy must be remembered, for though the fits are often trifling, yet, the "post-epileptic" condition is sometimes of a very serious nature: two such forms are described—

Post-epileptic conditions—

1. A condition in which the patient performs automatic actions, of which he is then and *afterwards entirely unconscious.* A father may kill his child; well-to-do patients may steal; etc.

2. A condition characterised by maniacal manifestations, intense passion, etc.

Prognosis.—Frequency of epileptic attacks vary considerably. Females are more frequently attacked during a menstrual period. Epileptics may be fairly strong between the attacks, and finally recover completely; but too often some mental weakness shows itself in a permanent form. Imbecility has developed after a few fits in children. The dangers during the fit must not be forgotten—fatal falls, burns, or other injuries are common.

Diagnosis.—Must be distinguished from hysteroid convulsions, uræmia, malingering, etc.

	EPILEPSY.	HYSTERIA.	MALINGERERS.	URÆMIC CONVULSIONS.
Consciousness.—	Lost.	May be partially lost.	Normal.	Lost in later stages; coma is prolonged and deep.
Pupils.—	Dilated during fit.	Normal.	Normal.	First contraction, followed by dilatation.
Tongue.—	Often bitten.	Normal.	May be bitten to simulate real fit.	Normal.
Restraint.—	Necessary to prevent accident.	Necessary to control violence.	Not necessary.	Not necessary.
Onset.—	Rapid and sudden, patient falls unconscious.	Usually after some mental excitement, patient may fall into a "dazed" condition.	Always under conditions where the fraud may hope to gain sympathy. He falls in a business-like manner, taking care not to sustain painful injuries.	Preceded by alterations in health, urine, etc. Patient is usually in bed before convulsions come on.
Recovery.—	Moderately rapid.	Very variable.	Very rapid after object has been gained.	Slow, if not fatal.

Treatment.—Try to discover any cause of reflex irritation and remove it if possible. Try to keep the general health as perfect as possible. Of drugs, bromides stand first; no permanent ill effects have been definitely traced to a very prolonged use of this drug. Some authorities advise an occasional course of bromides and arsenic. Careful attention must be paid to the state of the bowels, menses, etc.

During the Fit.—Loosen the collar, corset, etc., and put something between the teeth to prevent the tongue being bitten; inhalation of nitrate of amyl; dash cold water on face, etc.; and prevent patient from injury through his convulsive movements.

JACKSONIAN EPILEPSY.

Jacksonian epilepsy is an affection characterised by epileptiform convulsions, not attended with loss of consciousness and dependent on a coarse lesion of the cerebral cortex.

Ætiology.—It is usually associated with syphilis, and therefore most frequently seen amongst adult men; but other tumours besides gummata are held responsible, also traumatisms, blood clot, etc.

Symptoms.—First twitchings of a muscle or group of muscles in the arm, leg, or face. The spasms may be distinctly localised, and the patient being conscious, often watches the progress of the march, and thus affords most important information, which often enables the physician to localise the lesion. The convulsions very rarely become general. They may be followed by a transitory paralysis of the affected muscles.

Diagnosis.—

1. The slow onset.
2. Absence of unconsciousness.
3. Localised type of the convulsions.

Sufficiently distinguish it from ordinary epilepsy. *The part which first shows rigidity during the convulsion* points toward the motor centre for *that part* as the *seat of the greatest irritation.*

Treatment.—

1. If syphilis be the cause, give heroic doses of iodide of potassium.

2. If not syphilitic, then trephine, explore, and remove if possible any discoverable source of irritation.

INFANTILE CONVULSIONS.

Causes.—

1. Reflex irritation, especially from gastro-intestinal irritation, and teething; worms, otitis media, and phimosis are also occasional reflex causes.
2. Infectious fevers.
3. Cerebral congestion, well seen in severe cases of whooping cough, and after exposure to the sun in a perambulator, etc.
4. Rickets.
5. Cranio-tabes induced by syphilis.
6. Hydrocephalus.

Treatment.—During the fit. Seek cause, and if associated with a full stomach give an emetic, if with dentition lance gums, and so on. A few whiffs of chloroform, warm bath, ice to head, small doses of chloral, etc. The treatment after the fit must be conducted on general principles.

HYSTERIA.

Hysteria is a form of functional disturbance of the nervous system, characterised by an abnormal susceptibility to external impressions, and a deficient power of the will to restrain its manifestations.

Ætiology.—It is seen principally amongst women, and has long been considered symptomatic of disorders of the ovaries or uterine functions. It may be the result of nerve exhaustion from any cause.

Symptoms.—They are too varied to tabulate. Indeed, it is difficult to say what some authorities consider *to be* hysterical symptoms. The more common phenomena are—

1. Disturbances of the sensory apparatus.—
   (1) Hyperæsthesia of localised areas.
   (2) Anæsthesia, generally of left side.
   (3) Loss of muscular sense.
   (4) Neuralgias.
2. Disturbances of the motor apparatus.—
   (1) Hystero-epilepsy.
   (2) Tonic spasms. Example—vaginismus.
   (3) Clonic spasms. Examples—globus hystericus, uncontrollable laughter, hiccough, etc.
   (4) *Motor* paralysis—aphonia, simulated hemiplegia, etc.
3. Visceral Disturbances.—
   (1) Palpitation.
   (2) Vicarious menstruation.
   (3) Vomiting, belching, etc.
4. Physical disturbances.—
   Morbid desire for sympathy.
   Apathy and obstinacy.
   Sudden transition from the joyous to the sad.
   Melancholia.
   Somnambulism.
   Nympho-mania.
   Catalepsy—may simulate death.
5. Vaso-motor disturbances.

It will be seen from the above table, that it would not be easy to mention what symptoms may not crop up in various forms of hysteria. So closely does hysteria simulate real disease that the best diagnosticians have been deceived. When grave symptoms appear, even after a careful consideration of the history, etc., of the patient, *a provisional diagnosis* should be made until the patient has been for some time under treatment. (Dr Ranny's Lectures).

Treatment of hysteria depends on the type. All forms, however, require *firm*, kind, and judicious treatment. The

physician must first gain the patient's full confidence, and having done this he may use bromides, tonics, massage, and "Weir-Mitchell's" treatment. The various special symptoms must be treated on general principles. It is doubtful if such drastic measures as excision of the clitoris, removal of the ovaries, or applications of the actual cautery are admissible, though they have been practised by eminent authorities.

NEURASTHENIA.

By neurasthenia is meant the manifestations of nerve exhaustion. Hysteria may therefore be considered a symptom *per se* of neurasthenia, but there are some important differences.

NEURASTHENIA.	HYSTERIA.
1. Occurs most often in men.	1. Women most frequently.
2. Usually directly attributable to over work.	2. Often seen amongst the indolent and the rich.
3. No desire for sympathy.	3. Great desire for sympathy.
4. Usually wasting is present.	4. Often plump or fat.
5. Very amenable to proper treatment.	5. Anything but amenable to treatment.

The chief reasons why so much prominence has been given to neurasthenia as an independent condition are as follows :—

1. The tendency of the age is towards brain work.
2. Brain workers have enormous competition in life's fight.
3. The neurotic element is largely increased thereby.
4. This neurotic element renders its possessor more susceptible to external impressions.
5. Abuse of stimulants, alcohol and tobacco especially being taken at too frequent intervals, in order to lash up the flagging energy.
6. Modern cooking.

Treatment. — The causes and definition of the disorder makes it sufficiently obvious what line of treatment should be employed ; indeed, it may be summed up in four words— complete change, rest, tonics.

THOMSEN'S DISEASE.

It is a condition too rare to require a lengthened description in such a work as this.

The main features are a tendency of the muscles to tonic spasm during attempts at voluntary movements.

Ætiology.—Probably it is always associated with some congenital defect. Heredity is a strong and important factor, and more than one member of a family may be affected.

Pathology unknown. Possibly it is a *muscular* disease, and not a *nervous* lesion at all. The muscles are usually well developed as far as appearance goes; indeed, they often appear hypertrophied.

Electrical Reactions.—There is an entire upset in that relationship, which exists in normal muscles between C. C. C. and A.C.C. This tempts one to think the disease may be due to local metabolic changes, setting up an irritability of the muscular tissue, such irritability being specially brought out on attempts at movements; but *why* such a condition occurs is wrapped up in that obscurity, associated with congenital defects.

Symptoms.—

1. After *rest* the patient experiences tension and stiffness of the muscles on attempting to rise, etc.
2. Difficulty in *relaxing* the muscles.
3. The muscles of the tongue, face, and eyes, may also be similarly affected.

Note that continued movements and warmth decrease the spasm; mental worry and cold usually aggravate it.

Treatment.—Massage, gymnastic exercises, electricity, and warm baths.

TETANY.

Tetany is another rare disease which I only mention as it is so often confounded with tetanus.

Symptoms.—Paroxysmal tonic muscular spasms. When the upper limbs are attacked, the forearms are *flexed* and drawn across the chest, and the wrist and fingers are also usually flexed. If the trunk be affected, there may be oposthotonos or pleurosthotonos. When the lower limbs are implicated, the thighs are adducted; big toe brought *under* the other, and leg extended.

Trousseau lays stress upon the fact that the attacks can be induced at will by pressing upon either the arteries or nerve trunks. The great point is to diagnose this disease from tetanus, which may be done by remembering that in *tetanus* the *muscles of the jaw* are mostly implicated, and there is more pain and *general* rigidity; also a traumatic history and a more acute onset and rapid course.

Diagnostic Table No. 1.

	Locomotor Ataxia.	Spastic Ataxia.	Freidrich's Ataxia.
Age—	Middle-aged men.	Middle life.	Early life.
Causes—	Syphilis, intemperance.	Exposure to cold, traumatisms, etc.	Hereditary. Often more than one in same family.
Argyll-Robertson pupil—	Present.	Absent.	Absent.
Lightning Pains—	A prominent symptom.	Absent.	May be present, but more often absent.
Knee Jerk—	Lost.	Increased.	Lost.
Nystagmus—	Absent.	Absent.	Present.
Speech—	Not involved.	Rarely involved.	Often markedly involved.
Inco-ordination—	Marked as regards "gait" and lower limbs.	Marked, but attended by spasm. Spasm *tends to increase*, but *inco-ordination does not.*	Marked, but irregular and jerky; affects upper limbs as well.

DIAGNOSTIC TABLE No. 2.

	PROGRESSIVE MUSCULAR ATROPHY.	AMYO-TROPHIC PARALYSIS.	PRIMARY SPASTIC PARAPLEGIA.
Limbs most affected—	Upper — atrophy begins in thenar and hypo-thenar eminencies. Unilateral at first.	Upper — atrophy may begin in muscles of fore-arm or deltoid. Unilateral.	Lower—*no atrophy;* but rigidity and spasm are present. Bilateral.
Deformity—	The "claw-like" hand.	Flexion of elbow, pronation of hand, flexion of wrists, and fingers into palms.	Adduction of legs. They may cross each other.
Tendon reflexes (knee)—	Unaffected.	Unaffected.	Exaggerated on both sides.
Electrical changes—	Reaction of degeneration.	Partial.	Normal as a rule.

GAITS.

Locomotor Ataxia.—Feet are brought down with extended heels, creating a stamping gait. Greatest difficulty is experienced in turning around, or standing with feet close together. Patient's eyes are usually steadily fixed on the floor to *guide* movements.

Cerebellar Disease.—Here the inco-ordination is of the type of a drunken man. The greatest difficulty is experienced on trying to walk along a *straight* line. On standing, note the movements of the toes as if they were digging into the carpet. The patient may sway to the front, backwards, or laterally.

Disseminated Sclerosis.—Ataxia gait is accompanied with marked inco-ordination of the trunk. The patient shoots suddenly forward to one side. Note jerky movements of the head.

Spastic.—Feet are turned inwards, and are scraped along the ground; later, the patient may "hop" (when the muscles of the calves are implicated). The back is arched and the chest pushed forward. The feet, by forcible adduction, often cross each other.

DISEASES OF THE NERVES.

In the opening remarks on nervous diseases in general, we considered the functions and structure of peripheral nerves. We also considered briefly, the more important changes in their structure and function when they became diseased or cut off from their trophic centres. In order to save time and space, we shall not repeat the details of those minute pathological changes; but, just add that the morbid conditions may be most marked in—

1. The nerve sheath.
2. Nerve fibres.
3. Interstitial substance.

And it will be readily understood that sclerosis, whether in sheath or interstitial matter, must powerfully influence the function of that nerve. By "multiple sclerosis" is meant more or less scattered sclerosis of many peripheral nerves. The symptoms of such a condition are very many; when tabulated they are:—

1. *Sensory Symptoms.*—Patches of hyperæsthesia and anæsthesia, especially below the elbows and knees. Tenderness over the affected nerves and muscles. *Delayed transmission* of pain sensations and temperature sensations.

2. *Motor Symptoms.*—

(1) Progressive muscular weakness and a sense of fatigue, which in a few weeks develops into paralysis. The extensors of hands and feet are most frequently attacked.

(2) Atrophy of the muscles.

(3) Deformities due partly to the weight of the hands and feet not being supported by the paralysed muscles.

(4) Tendon reflexes are abolished.

3. *Trophic Symptoms.*—

(1) Glossy skin is usually very persistent.

(2) Œdema of the feet and hands, often present in the early stage.

(3) Excessive perspiration or marked dryness of the hands and feet.

ALCOHOLIC PARALYSIS.

Alcoholic paralysis is one of the best examples of multiple neuritis.

Ætiology.—It usually occurs amongst *secret* drinkers, and for that reason women are more often attacked. Most cases occur after thirty-five years of age. Club-men and brewers furnish a number of cases.

Symptoms.—We must remember that the manifestations of chronic alcoholism differ much in individuals; there are at least four well marked types.

1. That in which tremor predominates.
2. The anæsthetic type.
3. The paralytic type.
4. The ataxic form.

First there is tingling like pins and needles in the muscles generally, then numbness of the fingers and toes; vasa-motor spasm causing dead fingers, and muscular cramps. Progressive paralysis compels the patient to take to his bed; the extensors are more implicated than the flexors, and consequently the patient lies with dropped feet and dropped wrists (the feet generally first). Other muscles are gradually involved, and phination may be lost or much weakened. Atrophy of the

affected muscles rapidly supervenes, being especially well marked in the extensors below the knee and elbow. The atrophied muscles are often exquisitely tender.

The mental state often suffers: the memory is almost lost, and often delusions are marked features. The reaction of degeneration is present to a partial extent. Rarely, if ever, is there complete failure of response to faradic stimulation. The ataxic form resembles tabetic ataxy in the numbness of the feet, absence of knee jerk, locomotion, and non-wasting of the anterior tibial muscles; but, in such cases the gait is different from the true ataxic walk, as the patient not only throws *out his feet brusquely, but steps high in order to clear his toes of the ground.* (Dr Ferrier.)

Treatment.—Cut off the supply of alcohol entirely. Perfect rest. To ease the pain — iodide of sodium, antipyrin, and croton-chloral may be given. Afterwards—good feeding, massage, and electrical stimulation as a rule, will, if persevered in, cure.

SPECIAL NERVES.

After what has been said about the *cranial nerves* and *multiple neuritis*, there is no need to devote time to special descriptions of affections of special peripheral nerves. A knowledge of their anatomical distribution and function should enable us to reason what to expect when such nerves are injured.

DISEASES of the RESPIRATORY SYSTEM.

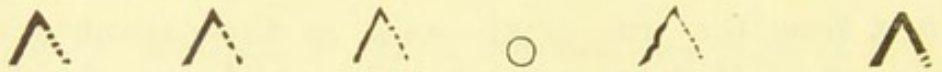
Dr John Wyllie's Notes on Examination of the Respiratory System.

AUSCULTATION.

Listen for—(1) *Type of Breathing*, (2) *Accompaniments*, (3) *Vocal Resonance.*

1. Types of Breathing.

(1) VESICULAR or RUSHING—



Puerile. Normal in Adult. Feeble. None. Interrupted. Harsh, with expir. prolonged.

Transition Type—



(Broncho-vesicular or "indeterminate" breathing.)

(2) BRONCHIAL or BLOWING—



High-pitched or Tubular. Medium-pitched. Low-pitched or Cavernous.

(3) AMPHORIC—



High-pitched. Medium-pitched. Low-pitched.

NOTES.—(1) *Vesicular Breath-sounds.*—In the auscultation of normal Vesicular Breathing, the *Inspiratory sound*, represented in the diagrams by a single line, is a fine, continuous, rushing sound, soft in the adult and loud in the child, and audible from beginning to end of the act. The *Expiratory sound*, on the other hand, is thin in quality and of short duration, being audible only during the earlier part of the Expiratory act. It is generally believed that in normal Vesicular Breathing the Inspiratory sound passes, as represented in the diagram, directly into the Expiratory without a break. Having, however, paid special attention to this point, Dr W. believes that there is often, in perfectly normal Vesicular Breathing, a distinct break between the two sounds. When the breathing is quiet and easy, the Expiratory sound is often totally inaudible,

even in children; but in such cases it can usually be brought out by causing the patient to breathe deeply. The term "prolonged expiration" is used to signify not a prolongation of the *act* of Expiration, but only a prolongation of the expiratory *sound*, resulting from the encroachment of the audible upon the inaudible part of the act. There is indeed one form of breathing, common to advanced Emphysema and Asthma, in which the act itself is really prolonged, being often much longer than the Inspiration. In such "Asthmatic" breathing, the type of respiration, primarily Vesicular, is as a rule totally masked by the loud wheezing accompaniments of both Inspiration and Expiration.

(2) *Bronchial Breathing.*—The auscultatory sound of Bronchial Breathing, indicated in the diagrams by a double line, can be imitated, as pointed out by Skoda, by holding the tongue in the position for the pronunciation of the guttural *ch* sound (as in the German word *Ach*, or the Scotch word *Loch*), and causing the air to pass inwards and outwards over it. The blowing sound thus produced can be made to represent the various pitches indicated in the diagram. There is always in Bronchial Breathing a distinct break between the sounds of Inspiration and Expiration, and the two sounds closely resemble each other. The higher-pitched varieties of Bronchial Breathing should be associated in the mind with conditions of consolidation of Lung Substance, such as that of Pneumonia, and the Low-pitched or Cavernous variety with Excavation, as in Phthisical cavity. *Bronchial Breathing is never produced by Bronchitis.*

(3) The *Amphoric* type of breath-sound can be well imitated by whistling with the mouth. Inspiratory and Expiratory sounds can thus be produced by causing the air to pass inwards and outwards, and the pitch can be varied according to the variety of Amphoric Breathing that is being imitated. Amphoric Breathing is best developed in Pneumothorax, but is also sometimes met with *in very large* Phthisical cavities.

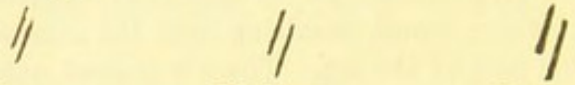
(4) In the *Healthy Chest* the respiratory sounds are purely Vesicular (without harshness of quality or prolongation of expiration) over the whole surface of the lungs, except (*a*) opposite the *Roots of the Lungs*, at level of third dorsal vertebra behind, and lower part of manubrium sterni in front, where the proximity of the large Bronchi generally renders the breathing *Broncho-vesicular*, by the addition of a Blowing or Bronchial element, most distinct during expiration; (*b*) over the *Apex of the Right Lung*, especially above the Clavicle and Spine of Scapula, where, in health, from causes as yet imperfectly ascertained, the Vesicular Breath-sound has very generally a more or less prolonged, and often harsh or even somewhat blowing, expiration.

The only example of purely *Bronchial* Breathing that can be heard on auscultating the healthy subject, is the "*Tracheal*" Breathing, to be obtained by placing the stethoscope over the Larynx or Trachea. This is low in pitch, and if heard over the apex of the Lung would be termed "Cavernous."

[Unfortunately some of the great original authorities on Auscultation applied the term "Bronchial" to the type of breathing heard over the roots of the Lungs, but, as this is partly of Bronchial and partly of Vesicular origin, the term "Broncho-vesicular" is much more appropriate.]

2. Accompaniments.

(1) Friction (in Pleurisy)—



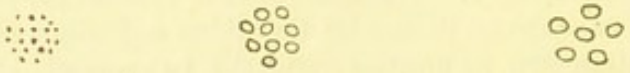
Fine. Medium. Coarse.

(2) Dry Sounds, or Rhonchi (in Bronchitis)—



High-pitched. (Cooing, Wheezing, etc., called "Sibilant.") Medium-pitched. Low-pitched. (Sonorous.)

(3) Moist Râles or Crepitations (1 and 2 in Pneumonia; 2 in Bronchitis; 2 and 3 in Phthisis)—



1. Fine. 2. Medium. 3. Coarse (Bubbling).

May be *Consonating* in three degrees—tough, metallic, tinkling. (Tinkling rales are specially important in connection with Pneumothorax.)

3. Vocal Resonance.

(1) Simple Increase—

(*a*) Slight, comparative.
(*b*) Marked (Bronchophony).
(*c*) Very marked (Pectoriloquy).

(The chief conditions which cause Increase of Vocal Resonance are Consolidation and Excavation of the Lung substance.)

(2) Simple Decrease—

(*a*) Slight, comparative.
(*b*) Marked decrease.
(*c*) Total absence.

(Decrease of Vocal Resonance is most frequently due to Thickening of the Pleura or to Pleuritic Effusion.)

(3) Qualitative Alterations—

(*a*) Aegophony (Nasal timbre).
(*b*) With metallic echo (Amphoric Resonance or Nach-klang).

(Aegophony occurs in Pleurisy with Effusion when the layer of fluid is thin. Metallic echo is one of the signs of Pneumothorax.)

PERCUSSION.

1. Hyper-Resonance—

(1) Slight.
(2) Marked.

(3) Very marked (Tympanites).
  (*a*) High-pitched.
  (*b*) Medium-pitched.
  (*c*) Low-pitched.

(Emphysema causes slight Hyper-Resonance. Relaxation of Lung substance, as in the Superior lobe when the Inferior is solid from Pneumonia or compressed by Pleuritic Effusion, is a cause of marked Hyper-resonance. Pneumothorax is the commonest cause of true Tympanites.)

2. Deficient Resonance—
  (1) Slight, comparative dulness.
  (2) Marked dulness.
  (3) Absolute dulness.

(Consolidation of Lung substance, thickening of the Pleura, and Pleuritic Effusions are the chief causes of Deficient Resonance.)

3. Mixture of Dulness and Resonance—*i.e.*, a Wooden or Boxy Note.

(This is one of the most important signs of a Phthisical Cavity.)

4. Special Quality—
  (1) Cracked-pot sound.

(Another very important sign of a Phthisical Cavity.)

  (2) Bell sound, got with two coins and stethoscope.

(One of the signs of Pneumothorax.)

SUCCUSSION.

Called "Hippocratic;" used in Hydro- and Pyo- pneumothorax.

INSPECTION, PALPATION, AND MENSURATION.

1. Form and Size of Chest—
  (1) Circumference of Chest at line of nipples.
  (2) General Form (flat, barrel-shaped, etc.)
  (3) Local alterations in Form (local flattening, bulging, etc.)

2. Movements of Chest—
  (1) Number of respirations per minute.
  (2) General type of movement (thoracico-abdominal, abdominal, thoracic).
  (3) Rhythm and Volume of respirations; and their Special Character, as in the "Cheyne-Stokes'" type, etc.
  (4) Local Movements (sucking in of intercostal spaces, etc.)
  (5) Deficient Expansion (over one apex, over one side, etc.)

3. Parts outside Chest—
  (1) Box of Larynx (its upward and downward movement).
  (2) Alae Nasi (their action in difficult breathing).
  (3) Bulging of Apices in neck on coughing.
  (4) Action of the Scaleni and other Extraordinary Muscles of Respiration.

4. Palpate for the Vocal Fremitus. (Note increase or diminution.)

EXTRA AUSCULTATION.

1. Of the Sounds of *Obstruction* in Respiratory Passages.

(1) Obstruction to Breathing in Nose	(*a*) From hardened or fluid Mucus.	Nasal Bubbling	
	(*b*) From Paralysis of Alae.	and Sniffling.	
(2) Obstruction in Back of Throat	(*a*) Nasal Snore.	Stertorous	
	(*b*) Oral Snore.	Breathing.	
(3) Obstruction in Larynx	(*a*) Swelling of Cords.	Stridulous	
	(*b*) Paralysis or Spasm of Glottis.	Breathing.	
(4) Obstruction in Trachea	(*a*) From Aneurism (Leopard-growl).		
	(*b*) Death Rattle.		
(5) Obstruction in Bronchi	(*a*) Musical Sounds (Wheezing, etc.)		
	(*b*) Crepitant Sounds.		

2. Of the *Cough*.

The following are some of the more common varieties of Cough :—

(1) *Cough of the ordinary type*, such as is met with in—(*a*) Common Colds ; (*b*) Some conditions of mere nervousness ; (*c*) Irritation of certain peripheral nerves, as those of stomach, ear, etc.; (*d*) Early Phthisis, when, however, it is apt to be specially hacking and irritating ; (*e*) Later Phthisis, when it may vary much in severity, from moderate, though frequent, to severely paroxysmal. Phthisical cough is often succeeded by vomiting.

(2) *The frequent, prolonged, paroxysmal, and often wheezing Cough* that is characteristic of severe Bronchitis.

(3) *The frequent, short, "suppressed" Cough* of Pneumonia with associated Pleurisy, and of simple acute Pleurisy. It is "suppressed" because it excites pain in the side.

(4) *The husky and sometimes stridulous or "croupy" Cough* that is characteristic of Laryngitis.

(5) *The peculiarly "brassy" or ringing Cough* (like the cry of a gander) that is met with in many cases of Aortic Aneurism or Mediastinal Tumour with pressure on the Trachea.

(6) *The prolonged paroxysm of fully developed Whooping-Cough*, with its rapid and long-continued succession of short, sharp coughs, and its final long-drawn stridulous inspiration. A succession of these paroxysms is very often succeeded by vomiting.

(7) *The loud Barking Cough* met with in some cases of Hysteria.

Note.—The *Sputum* should be carefully examined and described. Its more common types are—

(*a*) Viscid, mucous, and often pigmented, in Common Catarrh; (*b*) Bloody, in Hæmoptysis ; (*c*) "Nummular," in advanced Phthisis; (*d*) Copious, frothy, mucous or muco-purulent, in Bronchitis ; (*e*) Sticky, gelatinous, and rusty, in Pneumonia ; and (*f*) extremely fœtid in Gangrene of the Lung and some cases of Bronchiectasis.

In special cases a report should be made of the Microscopic characters of the Sputum.

When, with cough, there is no expectoration, *its absence should be noted.*

BRONCHITIS.

INFLAMMATION OF THE BRONCHI.

Ætiology. — Occurs most frequently in winter amongst elderly people, but the capillary variety is more often seen amongst young children, especially in connection with whooping-cough and measles; insufficient food, scanty clothing, or on the other hand, excessive confinement in warm rooms, and too warm wrapping up, are the great predisposing causes.

Exciting Causes—

1. Spreading of nasal catarrh.
2. Foreign bodies in larynx, etc.
3. Certain infectious diseases, *e.g.*, influenza, measles, diphtheria, etc.
4. Extension of inflammation from other parts.

Pathology.—That of a typical inflammation of a mucous membrane, *i.e.*—

1. Hyperæmia.—The membrane is injected, dry, and secretion scanty.

2. Exudation of lymph with increase of mucus—secretion more profuse.

3. Purulent degeneration of the exudation—muco-purulent phlegm.

4. Changes in the tissues beneath, slight at first, but repeated attacks cause grave changes, viz. :—

(1) Epithelium becomes to a large extent destroyed.
(2) Muscular coats are fibrosed and rigid.
(3) Tubes themselves become dilated or varicose, causing (as we shall see further on) bronchiectasis.

(*a*) *Acute Bronchitis.*—Ushered in with coryza and pains about the chest, and cough. The pain is burning in character, and situated behind the sternum. The expectoration at first

is frothy and scant, but soon becomes abundant and purulent, and often is expectorated in greenish jelly-like masses; later, it is more muco-purulent. The breathing is much embarrassed, and noisy or whistling in character; the temperature is not high as a rule, and the skin is moist. The urine is of a febrile type, scanty and high coloured. After a few days the more acute symptoms subside, and convalescence becomes rather slowly established. Death is rare in uncomplicated cases, but is often fatal through extension to the capillaries or air cells.

(*b*) *Capillary Bronchitis.* — This variety more frequently affects children, and is characterised by great dyspnœa, *sucking in* of the intercostal spaces, high temperature, quick feeble pulse, a tendency to drowsiness or coma, and collapse of the lung.

Physical Examination.—Rhonchi and râles are heard all over the chest. Percussion yields a slight hyper-resonant note, except where there are collapsed portions of lung; then there is, of course, impaired resonance and absence of breath sounds.

Treatment.—In the first stage employ the bronchitic kettle, containing a solution of eucalyptus or pinol; administer a brisk saline purge, and a diaphoretic mixture such as—

℞	Pot. Citras . . .	ʒiij.
	Vin. Antim Tart. .	ʒiij.
	Spt. Æther Nit. .	ʒiij.
	Spt. Chloroformi .	ʒij.
	Aq. ad	℥viij.

Fiat mist. A tablespoonful every three hours for an adult.

Later—ammonia, with senega and ipecac.; or ammonia and pot. iodid., with the compound tincture of camphor.

In the capillary form lowering measures are seldom called for. The two great dangers are—

1. Collapse of the lung.
2. Early heart failure.

If the former threatens, as evidenced by increasing *lividity and dyspnœa*, a brisk emetic should be given, followed at once by a stimulant, such as the compound spirit of æther with ammon. carb. *Sedatives are nearly always contra-indicated.*

CHRONIC BRONCHITIS.

(Winter Cough.)

Chronic bronchitis may be the result of the acute form; but it more often results from a series of sub-acute attacks in old people, whereby the bronchial tubes are "devitalised" and prone to inflame on slight provocation, especially in winter time.

Symptoms are dyspnœa, harsh paroxysmal cough, and copious expectoration of phlegm, which varies much in character, from being mucoid and ærated, to non-ærated muco-purulent, or even pus alone. Chronic bronchitis is too frequently looked upon as a *mere bronchial affection*, forgetting the wide spread and grave changes induced, as the following table shows:—

Pathological Changes.	*Physical Examination.*	*Clinical Symptoms.*
1. In the tubes themselves—rigidity of walls and subsequent dilatation.	1. Abundance of râles. Signs of indistinct cavities.	1. Copious expectoration at intervals of fœtid, pus-like secretion.
2. Dilatation of the air vesicles = emphysema.	2. Altered shape of chest, hyper-resonance, decreased breath sounds	2. Dyspnœa through imperfect æration, increased on exertion.
3. Hypertrophy of right side of the heart, (quickly masked by great *dilatation*).	3. Increase of cardiac dulness. Epigastric pulsation. Later, signs of tricuspid leakage—*i. e.*, general venous congestion.	3. Feeble pulse — œdema — perhaps anasarca; in fact all the troubles consequent on general venous congestion of the viscera.

Treatment.—Hygienic measures rank first in the treatment; proper clothing, careful dieting and change of air do more good than drugs. Of drugs, the iodides with ammonia are useful, terebine is praised; digitalis and strychnine are extremely beneficial when the pulmonary circulation is more sluggish than usual.

BRONCHIECTASIS.

Dilatation of the bronchi has already been referred to, as a complication or result of bronchial disease. We must now consider more fully how the condition is produced.

1. It may be a congenital defect, or produced by—
2. Primary disease of the bronchial walls.
3. Contraction of lung tissue in phthisis, etc.

Two forms occur :—

(1) Cylindrical or fusiform.
(2) Saccular.

The former is more often produced by strain from within, such as violent coughing, the rigid walls yielding to the expiratory inter-pulmonary pressure.

The saccular form is most often produced by—

1. Contraction of new fibrous tissue outside the tubes.
2. Breaking down of the lung substance, causing diminished support, *and yielding of the walls at that point.*

The dilated portion is usually smooth, until ulceration from retained secretion occurs. The walls are very thin, the muscular and elastic tissue being much atrophied.

Symptoms.—Physical examination may reveal all the signs of a cavity. The expectoration is copious and fœtid, and if

allowed to stand it separates into three layers, the lower layer being almost pure pus; next, a more granular zone; and a clear frothy layer on top of this.

Microscopically it shows—

1. Pus cells and epithelial debris.
2. Crystals of fatty acids.
3. Elastic fibres (may be absent).

The horrible odour is due to valerianic and butyric acids, H_2S, etc. The mode of expectoration is characteristic, patient usually brings up a huge quantity in the morning, or on moving after resting in an horizontal position for some time.

Treatment.—Antiseptic inhalations, capsules of creosote, etc. Terebine is warmly praised by some.

PNEUMONIA.

By pneumonia is meant inflammation of the lung substance. Three types are usually described, viz.:—(1) Croupous, lobar or acute pneumonia; (2) Catarrhal or lobular pneumonia; (3) Chronic or interstitial pneumonia. We shall first describe

CROUPOUS OR ACUTE PNEUMONIA.

This form is characterised—pathologically: by an inflammation giving rise to an exudation, *rich in fibrin, and showing colonies of special organisms—i.e., pneumo-cocci;*—clinically: by its abrupt onset, by running a definite course, and ending by crisis. It usually begins at the base, and may involve one or more lobes.

Ætiology.—Croupous pneumonia is a most common affection, occurs most frequently in winter and spring, and attacks all ages, though adults, perhaps, are more often affected than children. Amongst the more common exciting causes are draughts, intemperance, exposure to inclement weather, or irritating gases.

Frequently this kind of pneumonia occurs in epidemic form; indeed, there is an increasing tendency to look upon this form as a "*specific fever with marked lung symptoms.*"

Pathology.—It is convenient to describe four stages, viz. :—

1. Hyperæmia or engorgement.
2. Red hepatization.
3. Grey hepatization.
4. Resolution.

It is more than doubtful whether grey hepatization occurs, except in *fatal cases;* indeed, the condition is looked upon by many as a *post-mortem change*, and not a pneumonic process.

1st Stage.—The lung is injected, heavy, and more friable; on pressure, there exudes a frothy serum tinged with blood and slightly ærated.

2nd Stage or Red Hepatization.—The part involved is solid, and presents a granular or red granite appearance. (Dr Alex. Bruce denies the appearance being "liver like" or uniformly red.) The alveoli are filled with a coagulated exudation, which shows under the microscope—

1. Fibrin.
2. Proliferated cells.
3. Leucocytes.
4. Red corpuscles.
5. Granules and cell debris.
6. *Pneumo-cocci.*

3rd Stage or Grey Hepatization.—The lobe has now the appearance of grey granite, the lung substance is softer and more friable; on pressure, a dirty purulent fluid exudes. The grey appearance is due to four factors—

1. Decolourization of the red blood corpuscles.
2. Obliteration of the alveoli blood vessels from pressure.
3. Fatty degeneration of the coagulated material.
4. Great infiltration of leucocytes.

It will be seen at a glance that such an advanced change is hardly compatible with life.

4th Stage or Resolution.—Means resolution of the inflammatory exudation, principally by absorption, but partly by liquefaction and expectoration.

The Germ or diplo-coccus pneumoniæ of Fränkel, consists (in cultures) of *cells arranged in pairs, and sometimes in chains.* In the tissues the microbes become lancet-shaped. They retain the aniline stain when treated by Gram's method. Further evidence is wanted to prove that these are *the* organisms which cause acute pneumonia.

Physical Examination.

1st Stage.—*Percussion* yields a slight dullness, but sometimes even a slight hyper-resonance may be present. *Auscultation* reveals the characteristic fine crepitations, compared to the sound produced by the rubbing together of hair between the fingers. Many explanations of these sounds are offered, but the writer thinks the fine crepitations are due to the crackling of the *elastic tissue* of the alveoli in the inflamed condition.

2nd Stage.—Gives the signs of consolidation, viz.—

Inspection shows diminished movement.

Palpation confirms this, and also demonstrates *increased* vocal fremitus.

Auscultation reveals the absence of vesicular breathing, but, presence of typical tubular breathing. *Vocal resonance* is increased to the pitch of broncophony, or even whispering pectriloquy.

Percussion yields a dull note (not absolutely so flat as in pleuritic effusion).

3rd Stage.—This stage of grey hepatization can scarcely be detected by physical examination.

4th Stage or *Resolution.*—Here we have a speedy clearing up of the exudation, and a return to the normal condition. Small râles or *redux coarse* crepitations are heard, the dulness is less marked, and the movement increased.

Symptoms.—Croupous pneumonia may be ushered in with one or more rigors, *rapid* rise of temperature, and localised pain, plus the ordinary accompaniments of the febrile state. As the disease progresses, several characteristic features develop. The pain becomes less marked, but there is greater dyspnœa, and a marked disproportion *between respiration and pulse.* The former may be from thirty to seventy, and the pulse perhaps only 110. The cough becomes either hacking or paroxysmal in character, and there is expectoration of the *rusty, viscid phlegm, parthognomonic of this condition.* The pneumonic countenance develops—*i.e.*, flushed face, malar lividity, dilated pupils, and crops of herpes round the mouth. The urine is highly febrile, *chlorides are markedly diminished* and urates increased. Between the fifth and eighth day the symptoms most frequently *abate quite suddenly*, and rapid recovery takes place; but often, instead of this happy termination, the temperature increases, or perhaps falls to sub-normal, the *pulse becomes more rapid*, tongue dry and brown, the sputum less viscid and prune coloured, the patient quickly falls into the typhoid state, and death takes place most frequently from heart failure. Sometimes, prolonged exhaustion, or œdema of the sound lung brings about a fatal issue.

Special Points to Note—

1. The *viscid, rusty phlegm* (if it becomes prune-coloured, is an extremely bad sign).
2. Marked disproportion between pulse and respiration.
3. Diminished chlorides in the urine.

As failure of the right side of the heart is the more common mode of death, special attention should be paid to the state of the pulse, and condition of the second sound of the heart.

Treatment depends entirely on the type of case, and condition of the patient. In no disease has there been more serious blundering; and, routine treatment is the worst of all treatments. Answer the following questions before prescribing. Is the patient full blooded, and is there a full bounding pulse? Is the pulse feeble, irregular, or intermittent?

In the first case we may proceed cautiously with depressants, such as $\frac{1}{6}$ grain of tartar emetic, in combination with nitric æther and citrate of potash.

In the latter case we can hope for nothing from a depressing treatment, so stimulants must be resorted to, such as alcohol, ammon. carb., egg and brandy mixture, quinine, æther, etc. When the sputum is very bloody, I have seen nothing act as well as twenty minims of tinct. ferri perchlor., with five grains of quinine every four hours or even oftener. *Chloral* should be avoided.

Remember also that narcotics are not well borne in respiratory embarrassment as a rule, but *if pain be excessive* a hypodermic injection of morphia does *more good* than harm, notwithstanding that theoretically morphia is contra-indicated. Poultices may be applied to the chest, but they are of doubtful use where they are carelessly made. Some authorities advocate the local application of ice.

CATARRHAL-PNEUMONIA.

(Broncho-Pneumonia.)

Catarrhal Pneumonia as the name implies, is an extension of inflammation of the bronchioles into the air vesicles. *Frequently the process is set up in collapsed lobules.*

Ætiology.—It is most frequently seen amongst children, old people, and those who have to assume the recumbent position. Perhaps this, more than any other affection brings about a fatal issue in specific fevers. It is an almost constant accompaniment of whooping cough and measles.

Causes.—The more common are—

1. Chills, and exposure to inclement weather.
2. Extension of bronchial affections.
3. Inhalation of irritating vapours, gases, etc.
4. As a part of the tuberculous process.
5. As a sequence of infectious fevers.

Symptoms.—At first merely those of bronchial catarrh, the temperature rapidly becomes high, and markedly oscillating, or irregularly remittent in character. The dyspnœa becomes marked; the pulse feeble and irregular; and the cough harsh, short, and painful; the sputum is scanty, *never rusty*, but may be streaked with blood. The disease ends by lysis, and frequently the exacerbations of fever after the temperature has become normal, are most numerous. Loss of flesh is marked and difficulty might be experienced in diagnosing this affection from acute tuberculosis. Indeed, some authorities look upon this affection as an acute phthisis. *Undoubtedly catarrhal pneumonia is often followed by phthisis.* Brunton's explanation is: "That the delayed resolution of the inflammatory products form a suitable nidus for the tubercle bacillus," and he further suggests the use of arsenic to hasten fatty degeneration and absorption. Death often takes place from asthenia; or recovery is followed by a rather tardy convalescence.

Pathology.—We find consolidated patches, indefinite or sometimes sharply defined. The *air vesicles* in the consolidated area are—

1. Usually collapsed, with congested walls and swollen epithelial lining. There are—
2. Proliferated epithelial cells in abundance.
3. Leucocytes.
4. Mucus, and a slight amount of fibrin.

The bronchioles are inflamed, frequently plugged, and their walls infiltrated with small cells.

The surrounding lobules are somewhat congested, and emphysematous (compensatory emphysema).

Physical Signs are very uncertain. The chief signs over the consolidated areas are, increased vocal resonance and fremitus, *slight* tubular breathing *with small râles.* The intercostal spaces are often sucked in over the collapsed areas.

Treatment.—*Avoid lowering treatment.* Give ammonia, senega, and ipecac. Quinine, whisky, egg mixture, and antipyrin, are all useful; also the use of antiseptic inhalations. *Great care should be taken during convalescence.* Woollen clothing, cod-liver oil, hypophosphites, and malt extract, etc., must not be forgotten.

PLEURISY.

An acute inflammation of the lining membrane of the lungs and thorax.

Causes—

1. Exposure to cold.
2. Extension of inflammation from other parts, as pneumonia, hepatitis, tuberculosis.
3. Irritation from tumours—*i.e.*, aneurism of aorta, tumours of lung, mediastinal glands, etc.
4. Injuries from without.
5. In the course of other fevers.

Pathology.—That of a typical serous membrane, viz.—

1. *Hyperæmia*—loss of lustre, membrane is dry and red.

2. *Exudation of lymph*, which coagulates, and gives a shaggy appearance to the membranes.

3. *Effusion of Fluid* which is sero-fibrinous, yellowish-green in colour, with floating flakes of lymph. Specific gravity 1010 to 1020; coagulates on boiling (from amount of albumen); it also contains a large amount of fibrin.

4. *Resolution*, with more or less permanent *adhesions.*

(See Inflammation of Serous Membranes.)

The process, however, may stop at the second stage, and the exudation speedily resolve—*i.e.*, *dry pleurisy*; or the effusion may become purulent, and constitute an empyema.

Effects of the Effusion.—If large, it produces grave symptoms by the pressure on the lung tissue and viscera near.

1. It causes collapse of a portion, or sometimes the whole of the lung. (The lung naturally *tends* to collapse, by virtue of its own elasticity.)

2. The lung may be actually *pushed* across the middle line.

3. In extreme cases the heart, great vessels, and mediastinum are pushed to the *opposite side.*

The thorax bulges *forward*, and the liver or spleen get displaced *downwards.* The intercostal spaces may be slightly distended, and the diaphragm much embarrassed.

Absorption should occur in from 9 to 21 days. If it is unusually slow in absorbing, suspect empyema.

Are there any absolute signs of the fluid being purulent? No; but there are usually *some* signs, such as—

1. Period of absolute dulness persisting.
2. Temperature becoming septic in character.
3. Night sweats. (Patient often complains that his night clothes are wet through.)
4. Intercostal spaces are said to "*bulge*" more. (?)

The only certain way of ascertaining the presence of pus, however, is to use a hypodermic needle as an aspirator.

Symptoms.—Pleurisy is ushered in by—

1. Chills or rigors.
2. Rapid ascent of temperature (which is not so high as in pneumonia.)
3. Lancinating, tearing pain in the side, rendered worse by *any respiratory act.*

As the disease advances, and effusion takes place, the severe pain becomes replaced by dyspnœa. The patient lies on the

affected side to give the sound lung more freedom. The respirations are hurried, pulse quickened, and cough is hacking in character; the sputum is, however, slight in amount. Death may occur from collapse of the lung, or through the advent of some—

Complications such as—

Empyema.
Pneumonia.
Meningitis.
Heart failure.

Physical Signs.—*Vary with the stage.*

1. *Stage of Exudation.*
   (1) Auscultation reveals the characteristic friction rub. This sound, synchronous with the chest movements, is usually leathery-creaking in character, but is sometimes quite musical, like rubbing a pane of glass with a moist finger.
   (2) Palpation yields friction fremitus.
2. *State of Effusion.*—The physical signs over the affected area are—
   (1) Absence of breath sounds.
   (2) Absence of vocal resonance and fremitus.
   (3) Marked dulness on percussion.
   (4) Diminished movement.
3. *Above the line of Effusion.*

Just above is a small area where the voice is transmitted through—

(1) Slightly *condensed* lung;
(2) A *thin* layer of effusion; consequently giving rise to a peculiar modification of the voice, termed œgophony —compared by some writers to the bleating of a goat.

I have never found the resemblance. Perhaps the older writers' goats were different to those of the present generation.

4. Over the *lung* above the effusion.

The signs will depend on the amount of pressure. Usually, we get on—

Inspection—Diminished movement.

Palpation—Vocal fremitus increased. Vocal resonance increased to the extent of broncophony.

Percussion—"Skodic resonance." If heavily percussed, we get almost a "cracked-pot" sound.

Sometimes in the effusion stage, over the fluid, the breath sounds are *replaced by slight tubular breathing* (Osler). This, however, must be due to collapse of lung tissue around *patent* tubes, or a *patch of pneumonia;* and cannot, therefore, be looked upon as a *sign of effusion,* but of a *complication.* I earnestly ask the student to get a thorough understanding of (1) how breath sounds *are produced,* and (2) what the necessary conditions are *to modify those sounds—then,* reason for himself what "ought to occur under certain conditions." If we speak of possibilities of what *might* be heard in effusion, it would fill a book, and surely "*typical*" signs are enough for a junior student to get up intelligently.

Signs of displaced organs—

1. Altered position of "apex beat."
2. Ensiform cartilage pushed aside.
3. Liver pushed downwards.

Empyema—special points—

1. Whilst an empyema is frequently due to the *pleuritic effusion* becoming purulent, it may be *primary—i.e.,* purulent from the beginning.

2. Note that when "pleurisy" is a *sequela or complication of the zymotic fevers or pyæmic state, it nearly always becomes purulent.*

3. If pleurisy is a result of pneumonia, cancer, or tubercle, it is usually *purulent from the start.*

4. Dr Cathcart has called attention to the peculiar state of the ribs in *old* empyæmas, viz.—

(1) A crowding together of the lower ribs.
(2) Absorption of a portion of rib (atrophy).
(3) With a deposit of new bone (hypertrophy).
(4) Giving the rib on section a △ appearance—*i.e.*, atrophy at apex, and hypertrophy at base.

5. The prognosis is much more unfavourable, and demands surgical interference, as *pus may burrow anywhere.*

Treatment *of pleurisy*

Onset.—General principles of "fever." Relieve the intense pain by—

1. Poultices or blisters.
2. Hypodermic injection of morphia, or
3. Fifteen grains of pulv. ipecac. co.
4. Strapping the affected side (to diminish movement).
5. Leeches.

During Effusion. — Salines, quinine or antipyrin, digitalis and ammonia.

To Promote Absorption. — Pot. iodid., ammonia carb., ammonia chloride, arsenic and iron.

During Convalescence.—If slow, nourishing diet, cod liver oil, *arsenic and quinine, or phosphorus.*

Empyema.—

1. Evacuate pus—by—

(1) Aspiration.
(2) Resection and free drainage.

2. Tonics; remember the tendency of this complication to be associated with tuberculosis.

Diagnostic Table from Dr F. Roberts' Hand-book.

(MODIFIED.)

	BRONCHITIS.	CROUPOUS PNEUMONIA.	CATARRHAL PNEUMONIA.	PLEURISY.	ACUTE PHTHISIS.
1. Mode of invasion.	Coryza, and other symptoms of 'cold.' No marked rigors, but only slight and repeated chills, if any.	One or more severe rigors.	Generally after bronchitis, or collapse, and without distinct rigors.	Frequently none, but sometimes several; not severe rigors.	Follows pneumonia, bronchitis, or catarrhal pneumonia; or begins with severe rigors, often repeated.
2. Sensations about the chest.	Soreness, heat, etc., behind the sternum. Muscular pains from cough.	Pain in the side at first, not stitch-like, but more dull and diffused.	Pains about the chest, but not specially localised.	Severe, stitch-like pain in side, increased on respiratory movements.	Generally pains in various parts of the chest.
3. Cough.	In paroxysms, often severe.	Hacking, or in paroxysms.	Short, hacking, and painful.	Slight, and patient tries to repress it.	Frequent and violent fits.
4. Expectoration.	Abundant mucus, muco-purulent etc., changing its characters as the case progresses.	Considerable; viscid, tenacious, "rusty."	Often less than before; not "rusty."	Absent, or very slight, and of no special characters.	Abundant, and purulent, and numulated; often streaked with blood.
5. Disturbance of breathing.	Sense of dyspnœa, in proportion to the extent of the disease; may be extreme. Pulse-respiration ratio not proportionately altered.	Very rapid breathing, and much perversion of pulse-respiration ratio, but not proportionate feeling of dyspnœa until the later stages, when dyspnœa is marked.	Rapidity of breathing increased when it occurs in bronchitis; dyspnœa may be marked.	Quick, shallow breathing at first, but less disturbance of pulse-respiration ratio than in pneumonia. Later on, more or less actual dyspnœa according to amount of pressure.	Great dyspnœa, and very hurried breathing.

6. Degree of pyrexia.	Often absent or slight, and temperature rarely above 100° to 102°. Skin moist.	Considerable; temperature usually high, 103°, 104°, 105° or more, and runs a regular course. Skin peculiarly hot and dry.	Temperature high, but there are considerable remissions, at irregular intervals.	No regularity in course of temperature. Skin not acridly hot.	Often very high, but no regularity in temperature, *i. e.*, oscillates, but usually higher in evening.
7. Aspect of patient, & general condition.	Tendency to cyanosis, if the disease be extensive. In some cases adynamic symptoms.	Marked flushing of face, often unilateral. *Malar* cyanosis. Herpes round mouth.	Face is flushed. Often much anxiety and restlessness, with loss of flesh and strength.	Nothing special. No particular prostration, or tendency to cyanosis unless dyspnœa becomes marked.	Severe prostration and weakness, with profuse night sweats.
8. Physical signs.	Various râles and rhonchal fremitus. Signs of obstruction of bronchial tubes. More or less bilateral.	At first, fine crepatations, followed by signs of consolidation, viz., diminished movement, increased vocal fremitus, dulness, bronchial or tubular breathing, increased vocal resonance, etc. Usually one base is affected. The side is not notably enlarged, nor is there displacement of organs.	There may be signs of consolidation in scattered spots, with râles. Both lungs are usually involved in irregularly scattered patches. When it follows extensive collapse, there may be a peculiar pyramidal form of dulness.	At first friction-sound or fremitus, succeeded by signs of fluid, viz., side often enlarged, movements interfered with, diminished vocal fremitus, dulness occasionally moveable, weak or suppressed breathing. Usually on one side, and often displacement of organs.	At first merely signs of bronchitis, followed by consolidation, softening, or excavations in different parts. There is frequently nothing but scattered râles in very acute cases.
9. Course and termination.	Variable. No crisis. Tendency to death by apnœa or adynamia in the capillary variety.	Often a marked crisis—(1) From 5th to 8th day; (2) death; (3) gangrene of lung; (4) abscess of lung.	No crisis, and course often prolonged or followed by acute phthisis.	No crisis, and course very variable.	Generally very rapid course, and fatal termination.

PHTHISIS.

By phthisis we mean a morbid condition of the lung tissue due to the invasion of a specific bacillus; and characterised pathologically by formation of tubercles, which subsequently undergo retrograde changes, involving destruction of lung substance.

Ætiology.—It is still much debated whether phthisis is hereditary or not. It is certainly true that consumption does affect some families generation after generation, but still it is doubtful whether it is transmitted from parent to child. It is probable that whilst the disease is not strictly hereditary, a *predisposition* to it *is*. Or putting it plainer, whilst the offspring of consumptive parents are not *born with the disease*, they are born with a highly suitable soil for the development of such. The more common causes tabulated are—

Causes.—

1. Hereditary tendency.
2. Bad sanitary surroundings.
3. Deficient food.
4. Living on damp soils.
5. Undue exposure.
6. Certain occupations—stone-masons, knife-grinders, etc.
7. Excessive lactation.
8. Result of exhausting diseases, especially catarrhal pneumonia.

We have at page 55 considered the main facts relative to the bacillus, and the peculiar manner in which it exerts its pernicious influence. We have only to apply to the lung that which we have already seen occurs in tissues generally, to get a fair picture of a typical case of pulmonary consumption.

PATHOLOGY.	PHYSICAL SIGNS.	CLINICAL SYMPTOMS.
1. Damage to the lung tissue and invasion of the bacilli.	Weakened breathing; prolonged expiration, cog-wheel inspiration, etc., *plus* adventitious signs of the particular exciting cause.	Possibly no symptoms beyond a slight persistent cough, weakness, anorexia, etc.
2. Inflammatory changes around bacilli, resulting in the formation of grey tubercles.	*Consolidation.* Diminished movement. Slight flattening. Dull note. Vocal fremitus, and Vocal resonance increased. Bronchial breathing *(may be 'tubular' in quality).*	Increased weakness and cough; elevation of temperature, especially in the evening; diffuse pain in chest.
3. Inflammatory zone around the *tubercles.*		
4. Commencement of breaking down.	Consolidation, but attended *with moist sounds.*	Increase in the severity of above symptoms: there is now great emaciation, night sweats, oscillating temperature, hæmoptysis, distressing bouts of coughing, characteristic sputum, diarrhœa. Evidence of lardaceous disease in other organs, especially *liver.*
5. Formation of cavities.	Flattening is marked; movement much diminished; boxy note or cracked-pot sound; whispering pectriloquy; amphoric or cavernous breathing, *plus* adventitious sounds.	

Whilst the above table gives a short summary of the various aspects of this disease, it must in addition be remembered that cases widely differ. For instance, the disease may commence with very marked symptoms of bronchial catarrh; others again are so insidious in their onset that the greater part of a lung may be consolidated before any severe symptoms develop. And so with the *physical* signs, they vary enormously. We have, it is true, sketched the typical signs of *typical* damage to the elasticity of the lung, of consolidation, and of a cavity; but such typical signs are often absent or masked by the bronchial affection; indeed, the student, who has a clear idea how the various sounds are produced, will *learn more* by carefully examining six cases of phthisis than he will by digesting the most admirably written treatise on the subject. We shall now consider the various symptoms in detail.

Onset.—The disease usually commences at the apex of the lung by the formation of tubercles in the peri-bronchial tissue; but gradually deposits of tubercles form at lower levels, especially along the anterior margins. When the lower lobes are affected,

the seat of invasion is about that part of the lung corresponding to the vertebral border of the scapula, when the hand of that side is hooked over the opposite shoulder. Owing to the peculiar manner in which the disease begins and progresses, it is possible in *one* case to get the signs of invasion of tubercle at the *base*, consolidation in the *middle*, and excavations at *the apex*.

Course.—The disease may be rapid or extremely chronic. It may be hastened by severe complications, bad treatment, unusually suitable soil, or putrefaction of cavity contents. It may on the other hand be arrested or hindered by—

1. Formation of fibrous tissue encapsulating the tubercles.

2. Caseation and calcification of the tubercles or tubercular debris, thus causing cavities to dry up (natural cure). Remember, however, the disease having become chronic, may take on a rapid form through some inflammatory condition elsewhere.

(1) *Hæmoptysis.*—The blood is bright and frothy in the early stage, but later it may be dark from stagnation or venous congestion. During the excavation stage, small aneurismal swellings on unsupported blood-vessels *may burst and cause speedy death.* Often slight hæmoptysis is the first symptom of phthisis.

(2) *Cough* is a constant symptom; at first slight, then gradually becomes hacking, paroxysmal, and painful.

(3) *Expectoration* at first is muco-purulent, but later becomes more purulent and copious. It is non-ærated; often blood-stained, and shows the peculiar coin like arrangement when expectorated into water. Often little grey hard pellets are present, *with* abundance of elastic tissue and bacilli.

(4) *Pyrexia.*—At first the temperature is elevated only in the evening, but towards the end the temperature may oscillate continually between 100° and 104° F. Such a temperature is highly suspicious of sepsis due to absorption of broken down tissue. It is usually accompanied by the "hectic" flush, dilated pupils, and severe night sweats. When the *so called* night sweats

occur in the *earlier* stages, it is probably due to reflex vaso-motor disturbances *and not sepsis.*

(5) *Loss of Flesh.*—This is *nearly always most marked*, but extensive fatty degeneration or waxy degeneration, may somewhat mask the real wasting of muscle.

(6) *Diarrhœa.*—This is most often due to tubercular infiltration of the intestines, but may be excited by indigestible food, high pyrexia, or quinine, etc.

Complications.—

1. *Pleurisy* is most commonly present, but it is doubtful whether it should be looked up as a complication. Professor Gairdner in his excellent monograph says: "It is not to be regarded as a fatal complication, but as a healing power, in as much as it prevents perforation of the pleura, or even obliterates the cavity between the two layers, thus preventing empyema, pneumo-thorax, etc. *It is a curious and beautifully conservative arrangement* that in most cases the pleuritic adhesions are often *in advance* of the actual deposit of tubercle near the surface, and still more in advance of its softening."
2. Pyo-thorax.
3. Pneumo-thorax.
4. Spreading of the tubercular disease to the *larynx* meninges, peritoneum, etc.

Physical Signs.—We have already pointed out that the typical signs given in the table are often absent or masked by various conditions. It is often difficult to say whether a cavity exists or not, owing to—

1. Its small size.
2. It being deep down and more or less healthy lung intervening between it and the surface.
3. Thickened pleura.
4. Compensatory emphysema.
5. Breath sounds being drowned by extensive bronchitis.
6. Blocking of the tubes.

Again bronchial breathing is often heard in very thin healthy people; the "crack-pot sound" may be elicited in healthy children, and so on. We thus see there are many *pros* and *cons* to be considered before diagnosing a cavity, and unless the *general condition and history of the patient* are well considered, physical signs will often lead to an erroneous diagnosis. Post-mortem examinations prove this to be so.

Treatment will be considered under three heads—hygienic, dietetic, and medicinal.

Hygiene.—Patient must wear wool or flannel next to skin, take gentle out-door exercise, and sleep *alone* in an airy bed-room. He should be well rubbed after tepid baths, and absolute cleanliness observed as regards the teeth and mouth. The expectoration should be received into an antiseptic solution, and never allowed to dry on the carpet, etc. Whilst the patient should be kept warm, do not overload him with heavy clothes—do not, in fact, coddle him.

Dietetic.—We should aim at giving our patient *as much food as he can possibly assimilate.* Rum and milk is deservedly a favourite (though old-fashioned) remedy, given early in the morning. The patient's great dislike to fats of all kinds, hampers dietetic treatment a great deal. Koumiss and peptonised meats must always be remembered when the assimilative powers are weak.

Medicinal.—

1. *General.*—Tonics, such as hypophosphites, maltine and cod-liver oil are the principal remedies prescribed.

2. *Symptomatic.*—Any grave complication must be treated energetically.

(1) The cough.—As this is a persistent and constant feature of the disease, avoid rushing to the usual cough mixtures at once. A most common exciting cause of the nightly cough is the changing

from a warm room to a cold bedroom; or again, tickling of the fauces by the uvula. A very good cough mixture is—

℞ Tinct. Chloroformi et Morphinæ . . ℥ij.
Acid, Nitric Dil. . . . . ℥ij.
Glycerine ad . . . . . ℥iss.

Fiat mist. ℥i when the cough is very troublesome.

Inhalations "*moist or dry*" are of the utmost value.

(2) The *night sweats.* — Picrotoxin, quinine, atropine, and oxide of zinc are the favourite remedies.

(3) The *diarrhœa* —It is usually best controlled by mineral astringents, in combination with opium.

(4) Hæmoptysis.—Absolute rest and cold applications. Tincture ferri, hamamelis, gallic acid, erogotine, or *best of all a full dose of opium.*

Other complications must be treated on general principles.

Change of Air.—Under this heading we shall include—

1. Sea voyages.
2. Change of residence.

Sea voyages undoubtedly do good in many cases of early phthisis. Whether the benefit is due to a general bracing up of the nervous and muscular systems, or whether the sea air (containing as it does, iodine bromine, etc.), exerts a beneficial *local* influence is not yet determined, but probably both factors contribute to the cure. Before deciding to send a patient on a long voyage several points must be considered. Is the patient a good sailor? If *not*, is the severe sea sickness likely to produce hæmoptysis, or prevent proper feeding? Is the patient well off and likely to receive every comfort on board? Remember a woman cannot "rough things" as easily as a man; and further, many things which are not *in*convenient to a man, must be so to a woman. No patient should go without a relative or friend accompanying him. Lastly, sea voyages are contra-indicated in the later stages of phthisis.

Change of Air.—A phthisical patient requires either *dry cold* or warm air. In the earlier stage cold dry air is the best. New Mexico, Davos, and Canada, are the best examples of such residences. For warm climates we might select New Zealand, Madeira, Algiers, Torquay, Hastings, etc. Remember it is absolutely cruel quackery to send a patient far advanced in phthisis, away from home and friends, only too frequently to die amongst strangers.

Special Treatments.— Many special forms of treatment have been advocated—

1. By Koch's *Tuberculin*—

   A treatment which is the outcome of much patient research, and based upon very scientific grounds. Results are very conflicting, and this treatment at present is in evil repute. *Possibly its failure is the outcome of imperfect trial.*

2. Intra-laryngeal injections of guaicol, iodopyrin. Much success has been obtained by different physicians by this method.
3. Surgical Treatment.—Aspirating the cavities, followed by injections of antiseptics.

Amongst other treatments may be mentioned those which imitate natural cure by causing fibrosis or calcification. Alcohol in large doses. Rectal injections of carbonic acid gas. Other special treatments have been advised by many physicians residing at popular health resorts; many are either the result of fanaticism or something worse.

Laryngeal Phthisis will be considered under diseases of the larynx.

PNEUMO-THORAX.

Pneumo-thorax means the presence of air in the pleural cavity.

Causes.—

1. Traumatisms, such as punctured wounds, laceration from the end of a broken rib.

2. Causes from the lung side—

(1) Bursting of a pulmonary abscess into the pleura.
(2) Gangrene of the lung.
(3) Excessive intra-pulmonary pressure.
(4) Tubercules breaking down, causing undermining and perforation of the pleura when adhesions have not been set up.

Physical Signs.—

Inspection.—Bulging may be local or general on the affected side.

Percussion.—Usually yields a hyper-resonant note. If the valvular-like opening be patent, a typical crack-pot sound can be elicited.

Palpation.—Absence of vocal fremitus.

Auscultation.—Absence of breath sounds. On coughing the sounds produced are peculiarly amphoric and have a metallic ring. If the chest be struck with two coins whilst listening with the stethoscope the characteristic bell or anvil sound is heard.

HYDRO-PNEUMOTHORAX.

A condition where air is *present above* and fluid *below* in the pleural cavity.

Physical Signs.—*In the upper part*—those signs already described as diagnostic of pneumo-thorax, plus *hippocratic succussion* (the term applied to the splashing sound heard on shaking the patient.)

Below—the signs diagnostic of fluid already discussed under pleurisy.

Symptoms.—The symptoms of pneumo-thorax will depend largely upon the manner in which it was produced, and the quantity of air present. They may be summed up as follows—

1. Sudden pain at time of rupture with or without collapse.
2. Great dyspnœa.
3. Quick and small pulse.
4. Shallow and rapid breathing.

Treatment.—Subdue the pain by a hypodermic injection of morphine, hot poultices, diffusible stimulants, etc. Aspiration is of doubtful value.

HYDRO-THORAX.

Hydro-thorax—*i.e.*, dropsy of the pleuræ, is a term used to denote fluid in the pleural cavity, the result of a passive process, as seen in Bright's disease, etc.

The main points of difference between it and real pleuritic effusion are—

1. Not being preceded by acute symptoms of inflammation, therefore, absence of friction.
2. Usually bi-lateral.
3. The fluid gravitates with movements of the patient to a far greater extent (like ascites).
4. Aspiration is not likely to do more than *temporarily relieve pressure.*
5. Associated with signs of dropsy elsewhere.
6. Absence of pain, but dyspnœa is greater than in acute pleurisy.

ASTHMA.

Asthma is a disease characterised by sudden attacks of dyspnœa, which subside after a time, but tend to recur at intervals.

Ætiology.—Heredity is a most important factor. When it develops in children there is usually a history of post pharyngeal trouble, measles, whooping cough, or imperfect recovery after capillary bronchitis; gout, malaria, and subsidence of skin eruptions, are also held responsible for many cases. Probably, however, *most* cases have a neurotic origin. It is much more frequent in males than in females, possibly because of their greater liability to bronchial affections generally.

Pathology.—All authorities agree that the affection is due to diminished calibre of the smaller bronchioles, but the nature of such contraction is still disputed. The most probable theory is the first, viz. :—

1. *Reflex* spasm of the bronchial muscles, associated with hyperæmia and turgescence of the mucous membrane lining the smaller bronchioles, and the exudation of a characteristic mucus.

2. *Hyperæmic swelling of the mucous membrane*, like "nettle rash" (Sir A. Clark).

3. *Vasa-motor* paralysis.

Possibly all three theories are necessary to explain all cases.

Symptoms.—

1. *Premonitory Symptoms.*—There is usually some visceral disturbance as flatulence, etc.

2. *The Attack.*—It occurs most frequently during the night, the patient waking up with a feeling of great dyspnœa; he

feels as if there is not sufficient air in the room, and asks for the windows and doors to be opened. The characteristic attitude of asthma is assumed — *i.e.*, the patient grasps some support to fix the shoulder girdle, in order to bring his extraordinary muscles of respiration into play. Expiration is prolonged. Sibilant rhonchi cause a peculiar, noisy, pipe-like wheezing; but in spite of the extraordinary efforts little air enters the lung. A paroxysm of coughing and expectoration gives slight relief or even terminates the intense dyspnœa, and sleep may supervene, or a slight lull may be succeeded by another paroxysm.

The Sputum is usually expelled with the greatest difficulty, and is distinctly peculiar in its composition. The ball-like gelatinous masses can be unfolded, and is then found to represent casts of the small bronchioles.

Curschman describes the expectoration under the microscope as follows :—

At First.—The pellets show two forms of spiral threads. The one form entangles within its mesh work cells in various stages of fatty degeneration. The other form contains a central clear filament, surrounded by a network of other filaments.

Later.—The filaments disappear, and octahedral crystals of phosphates appear in the now muco-purulent expectoration. The course of the attack depends on the immediate cause, and the amount of bronchitis associated with it. As a rule, the asthmatic attacks tend to become less severe, but the bronchial affection more pronounced. Death seldom or never takes place from pure asthma.

Physical Signs.

During the Attack.—The thorax is expanded and fixed. Diaphragm moves but slightly, inspiration is short, and expiration prolonged. Auscultation : vesicular breathing is drowned by sibilant rhonchi, or later, with bubbling râles. Percussion reveals little, beyond slight hyper-resonance.

Treatment.—

During the Attack.—Remove any obvious cause of reflex irritation, such as an overloaded stomach. A brisk emetic often cuts an attack short; nitrites, especially nitrite of amyl or chloroform may be inhaled. Belladonna, stramonium, and lobelia in combination with ammonia are useful remedies; the fumes of burnt nitre paper, etc., are also recommended.

During Convalescence.—Change of air, careful diet, cod-liver oil, tonics, etc.

EMPHYSEMA.

By emphysema of the lungs is meant — (1) A condition where the air cells are over distended = vesicular emphysema; (2) A condition where the air has accumulated in the interstitial tissue = interstitial emphysema.

Emphysema is again divided into—

1. *Compensatory Emphysema.*—A condition where a portion of lung expands to take the place of a collapsed portion; seen in catarrhal pneumonia, pleuritic adhesions, in areas of old cicatrices, etc.

2. *Atrophic Emphysema*, or small lung emphysema, due to senile atrophy. The chest is of course small.

3. *Hypertrophic or ordinary Emphysema.* — A condition characterised by—

(1) Over distension of the vesicles.
(2) Atrophy of their walls.
(3) Obliteration of blood-vessels, and a consequent diminished "oxygenating" area.
(4) Changes in the shape of the chest.
(5) Changes in the heart (right).
(6) Changes generally, due to imperfect exchange of gases between the blood and the air.

Causes.—They include all those factors which keep up a more or less persistent high intra-alveolar tension.

1. Playing on wind instrument.

2. Certain occupations, such as glass blowing, colliers, etc.

3. Dr Jackson of Boston, lays great stress upon the hereditary character of emphysema.

Pathology.—The pleuræ are pale. The lungs pit readily on pressure, and have a peculiar soft downy feel. The dilated vesicles are well seen on the surfaces, and also projecting from *the free margins* of the lungs. The vesicles first become dilated, coalesce, atrophy, and the septa between the neighbouring cells get absorbed. The capillaries disappear. Though the changes are principally in the lung vesicles, there are always more or less marked changes in the bronchial tubes. Bronchiectasis is often a marked feature.

Symptoms.—They are too well known to require details; the important points to remember are — deficient æration means dyspnœa and cyanosis; the retention of waste products within the blood means impure blood and defective nutrition; the venous congestion is increased by the dilatation of the right heart, and thus a cycle of down grade changes is brought about, ending in, perhaps, dropsy, uræmia, or collapse of lung, etc.

Physical Signs.—Barrel shaped chest, prominent sternum, deep sternal fossa, prolonged expiration, and hyper-resonance are the more common features. A zone of dilated venules along the attachment of the diaphragm is spoken of by some writers. The epigastric pulsation and altered position of apex beat are important signs.

Treatment.—That of chronic bronchitis. Sudden and grave attacks of dyspnœa may be treated by inhalation of nitrite of amyl, venesection, etc. Sometimes a brisk emetic is the best treatment.

COLLAPSE OF THE LUNG.

Collapse of the lung may be of four types—

1. *Atelectasis.*—A condition found in weakly new-born children, when there is not sufficient inspiratory power to inflate the alveoli. The collapsed patches are of a slate colour and sink in water.

2. The form due to pressure from pleuritic effusion. The *whole* lung may collapse.

3. Collapse due to wounds of the chest wall, and perforation of the pleura. In this form the lung at first is congested, but finally becomes anæmic.

4. Ordinary or lobular collapse, as seen in broncho-pneumonia. Two theories are advanced to explain this condition—

(1) That a bronchus is plugged by a pellet of mucus which acts as a ball valve—*i.e.*, allowing air to pass *out*, but none to *enter*.

(2) That the plug does *not move at all*, but merely prevents access of *air* during inspiration. The alveolar air is exhausted by absorption, and none being taken in to replace it, the lobule collapses.

Symptoms. — Great dyspnœa, rapid pulse, cyanosis, all depending on the extent of the collapsed area. It occurs often in measles, whooping-cough, croup, and other conditions which give cause to catarrhal pneumonia.

Treatment.—Depends on the cause. Emetics and diffusible stimulants.

ŒDEMA OF THE LUNG.

Definition.—An accumulation of fluid in the interstices of the lungs, air vesicles, and bronchioles.

Causes.—Nearly always associated with general blood diseases, such as Bright's, the anæmic diseases, etc. It is common in the later stages of—

1. Valvular disease of the heart.
2. Malignant fevers.
3. Paralysis.
4. Long continued rest on the back.

} Hypostatic congestion.

5. *After the use of pilocarpine.*

Pathology.—The lung is heavy and bulky. On pressure a quantity of blood-stained serum exudes.

Physical Signs.—The breath sounds are deficient, and masked by fine râles. Contrary to what one might expect, the percussion note is more frequently resonant than dull. The explanation given is that a lung, when *partially collapsed* outside the body, gives forth a resonant sound, *due to diminished tension of the lung tissue.*

Symptoms. — Those of serious pulmonary embarrassment (already described under collapse of the lung), *plus* the abundant expectoration of a frothy serum.

CHRONIC OR INTERSTITIAL PNEUMONIA.

It is a most difficult task to explain what is exactly meant by chronic pneumonia. We have already seen how interstitial changes are present in all chronic affections of the lung. We have discussed such changes in Emphysema, Phthisis, Bronchiectasis, etc. Further, we have clearly demonstrated the fact, "that when the pulmonary tissue is damaged it forms a highly suitable soil for the development of tuberculosis." This statement makes it extremely difficult to say where chronic pneumonia ends and fibroid phthisis begins. I shall here describe as chronic pneumonia, that condition of the lung tissue characterised by

overgrowth of the fibrous elements, due to the action of slowly acting irritants, and attended clinically with symptoms of pulmonary embarrassment; later on, the symptoms are *dependant on grave structural change and breaking down of lung substance.*

Causes.—

1. Inhalation of dust particles (pneumonokoniosis). Examples, anthracosis (coal miner's lung); silicosis (mason's lung).

2. Irritants in the form of animal poisons.—Glanders, syphilis (white hepatization).

3. Excessive use of alcohol.

4. Result of acute pneumonia, pleurisy, etc.

Pathology.—The morbid anatomical changes depend on the cause, and so the reader is referred to a text book on pathology. We shall merely state here the conditions common to all.

1. Great overgrowth of interstitial connective tissue, encroaching upon and destroying the air vesicles.

2. Changes in the bronchial tubes; their muscular coats are replaced by fibrous tissue, which subsequently yield to intra pulmonary pressure, and form bronchiectic cavities (see bronchiectasis). Sometimes their lumen are occupied by caseous masses, and very frequently *obliterative* changes take place, and the lumen becomes totally obliterated.

3. *Obliterative changes in the pulmonary and bronchial blood-vessels, leading to severe systematic and local changes.*

Symptoms.—A careful digest of the outstanding features of the morbid structural changes ought to enable us to anticipate the more constant symptoms. The disease runs a very chronic course, and is usually unilateral. Cough may be the only symptom complained of for a long time. Later, the symptoms are those of bronchiectasis.

Physical Signs. — The affected side is retracted, much flattened, and the intercostal spaces more or less obliterated.

The percussion note will depend on the state of the bronchi, ranging from *absolute dulness to a boxy note;* indeed, in the later stages, the physical signs may be those of small cavities with extensive bronchitis.

Treatment.—Remove if possible the exciting cause, and then treat on general principles already laid down, in speaking of the various pulmonary affections.

TUMOURS OF THE LUNGS.

CANCER.

Primary cancer of the lung is very rare. The secondary variety occurs in the form of either *dense,* hard, irregular nodules, or soft (medullary) masses, scattered throughout the lung tissue. The original sources are mostly—

1. Cancer of the bronchial glands, which spreads inwards at the root of the lung, or

2. Cancer of the liver, which invades the diaphragm, and finally the pleura and lung.

Symptoms are insidious and varied. The amount of consolidation often causes pneumonic symptoms. The ulcerative process gives similar signs to phthisis; and hæmoptysis is generally present.

Diagnosis.—When there are symptoms of malignant mischief elsewhere, *marked pulmonary symptoms point to the invasion of the lung.* Pleuritic effusion may be so well simulated that puncture is sometimes required to clear up the diagnosis. The disease is usually fatal in about six months from the onset of symptoms.

Other tumours are too rare to need a detailed account in a work like this.

DISEASES OF THE LARYNX.

Inflammation of the larynx may be acute or chronic.

ACUTE LARYNGITIS.

Causes.—Contact with irritating vapours, drinking scalding water, impaction of foreign bodies, exposure to damp and cold air, extension of inflammation from other parts, and malignant blood conditions.

We have already referred to diphtheria as a cause, and we also discussed the differential diagnosis of croup and laryngeal diphtheria.

Pathology.—It is similar to that of bronchitis.

1. Hyperæmia, causing dryness.
2. Exudation of lymph = increased muco-purulent secretion.
3. Changes in the tissues beneath, etc., especially—
   (1) Œdema of the *submucous coat and the glottis.*
   (2) Changes in the nervo-muscular apparatus of the larynx.

Symptoms—Soreness and dryness accompanied by hoarseness, or complete loss of voice. Respiration may be stridulous and noisy. Rhonchi and râles are often abundant.

The loss of the voice is due to failure of the vocal cords to meet in the middle line, and to thyro-arytenoid paralysis.

When the glottis becomes œdematous the symptoms are most alarming. Intense dyspnœa, *dysphagia*, and stridor are the marked features. Death may occur in a few hours. Laryngoscopically: the epiglottis is seen to be much swollen and bright red in colour; the vocal cords may or may not be inflamed.

Treatment.—

Catarrhal Form.—Rest in bed, steam antiseptic inhalations of eucalyptol, and benzoin. A full dose of Dover's powder often cuts the attack short. Demulcent drinks and absolute rest to the voice.

Œdematous Variety.—Scarification of the glottis, sucking ice, æther spray, and tracheotomy.

It should particularly be remembered that very little irritation may provoke œdema of the glottis in gouty individuals.

HOW TO USE THE LARYNGOSCOPE.

1. *Position of Patient*: sitting, body and head erect, knees together, head slightly thrown back.
2. *Lamp*: in line with patient's ear, nine inches to the left of his head.
3. *Position of physician*: opposite patient with mirror properly adjusted to head and eye.
4. *Mouth*: wide open.
5. Reflect light upon fauces at correct focal distance of reflector.
6. Warm laryngeal mirror over lamp. Test it against cheek or hand.
7. Direct patient to protrude his tongue.
8. Hold it between thumb and index-finger, in napkin (thumb uppermost).
9. Hold laryngeal mirror like a pen.
10. Place its back gently against uvula.
11. Move your hand slightly towards patient's left, so as to keep it out of line of view.
12. Patient to drawn a deep breath, and say 'ah,' 'ur,' 'eh,' or 'ee.' Be always quiet and gentle; encourage the patient; let each examination be short, even if unsuccessful. Be careful not to hurt patient's tongue, or to burn his mouth, or to push either his uvula or the mirror against the back of the pharynx. Use cocaine to lessen sensitiveness of fauces (KEETLEY).

CHRONIC LARYNGITIS.

Chronic laryngitis may be the sequel of acute laryngitis, but it is much more commonly due to—

1. *Excessive use of the voice—i.e.*, "dysphonia clericorum," a condition characterised by an œdematous unhealthy condition of the mucous follicles, which present a raw or even ulcerated condition. Aphonia is very common after prolonged speaking.
2. Tumours, fibroid polypi, epitheliomata, etc.
3. Gout.
4. Various nervous affections—functional and organic.
5. Specific ulcerations—*i.e.*, syphilitic and tubercular.

PHTHISICAL ULCERATION.	TERTIARY ULCER.
1. Attacks first near artenoid cartilages.	1. Attacks epiglottis first.
2. Does not advance rapidly.	2. Progresses rapidly,
3. Great thickening.	3. Little thickening.
4. Granular appearance of posterior surface of epiglottis.	4. Inflamed appearance of the base.
5. Expectoration, frothy, thin, muco-purulent.	5. Expectoration thick, tenacious, yellowish.

Symptoms.—The severity of the symptoms depend on the cause. The more common are—

1. Constant hawking and desire to swallow.
2. Expectoration of muco-purulent phlegm.
3. Attacks of aphonia.
4. Spasmodic breathing (most often associated with irritation by tumours).

Treatment.—Depends on cause. Nervine tonics, such as strychnine and iron are nearly always indicated along with

plenty of fresh air; rest to the voice, and *douching the throat with cold water.* Locally, we may use astringents, such as tannic acid, nitrate of silver (10 grains to ℥j), chloride of ammonium vapour, and various antiseptic sprays.

If Syphilitic. — Pot. iodide internally, with the local application of bichromate of potash. Tracheotomy may be necessary.

If Tubercular.—Insufflations of morphia, scrape tumours with curette and apply lactic acid. Intra-laryngeal medication already described.

LARYNGISMUS STRIDULUS.

FALSE CROUP.

False croup or child crowing is a spasmodic disease of the larynx, occurring in infants and young children, consisting of a temporary closure of the rima-glottidis, causing great dyspnœa and other symptoms dependant on temporary suffocation.

Ætiology.—Predisposing causes are malnutrition brought about by imperfect feeding; syphilis, rickets, enlarged tonsils, and the tubercular diathesis. The exciting

Causes are reflex nervous disturbances, brought about by—

1. Intestinal irritation.
2. Worms.

} Sensory nerves.

3. Teething (*via* trifacial nerve).
4. Exposure to cold (vasa motor).
5. Overloading the stomach (pneumogastric).
6. Frights and "starts" (cortical).

It will thus be seen that the disease is essentially associated with a neurotic temperament, and disturbances of the various

nerve centres. There are three or four varieties of laryngismus. Goodhart describes three forms—

1. *Direct Spasm*, a crowing of a *convulsive nature* often rachitic.
2. *Infantile Spasm.*—crowing is due to a congenital valvular formation of the upper orifice of the larynx.
3. *Reflex Spasm.*

Symptoms.—Sometimes a slight cold *precedes the attack* (catarrhal form), but often the child is apparently well on going to bed, and wakes up suddenly at night with a brassy croupy cough and dyspnœa. The spasm is usually worse on first awaking. After a time the spasm passes off and the child, beyond having a slight croupy cough for a day or two, seems none the worse for the attack. Often a series of paroxysms, however, takes place, or recur at *night only.* The mother is usually much alarmed, and is very anxious for the arrival of the doctor.

Pathology.—It is to be regarded as a neurosis at present, but the palate and mucous membrane around the laryngeal orifice are often œdematous

Diagnosis from Croup, viz.—

1. Absence of false membrane.
2. Absence of marked local inflammation.
3. History.

Treatment.—When in doubt, treat as if it were the more severe disease.

1. Steam kettle and medicated vapour.
2. Emetic of ipecacuanha.
3. Clear out the bowels of any offending matter.
4. Bromides in the interval.

LARYNGEAL PARALYSIS.

During ordinary respiration, the glottis remains partially open, being widened with every inspiration. For the production of voice, the free borders of the vocal cords must be brought almost close to each other in the middle line, only a very narrow chink being between their parallel sides. At the same time the cords must be rendered tense. The narrowing of the chink is brought about by the adductors, viz., the lateral crico-arytænoid, assisted by the *posterior* arytænoid and *external* thyro-arytænoid muscles. The tightening is due to contraction of the crico-thyroid. The nerves involved are the superior laryngeal and recurrent laryngeal branches of the vagus. It will be easily understood that aphonia or loss of voice may be brought about by local muscular causes or central nervous lesions, and we have already referred to aphonia complicating bulbar paralysis, etc. Again the paralysis may be partial, or complete, of a functional nature, or due to organic lesions.

Dr Gower's gives the following table :—

LESION.	SYMPTOMS.	POSITION OF CORDS.
Total bilateral paralysis—	No voice; no cough; stridor only on deep inspiration.	Both cords slightly abducted and motionless.
Total unilateral paralysis—	Absence of stridor except on deep breathing; no cough; voice low and hoarse.	One cord motionless; the other moving freely, and *even beyond middle line in phonation.*
Total abductor paralysis—	The voice is little changed; cough normal; inspiration difficult and long, *with loud stridor.*	Both cords lie together, and not separated during inspiration.
Unilateral abductor paralysis—	Little affection of either *voice or cough.*	One cord not moving during inspiration.
Adductor paralysis—	No voice; perfect cough; no stridor or dyspnœa.	Cords are not brought together, but move during respiration.

Causes of Laryngeal Paralysis.

1. From the nerve side.
   (1) Central lesions.—Bulbar paralysis, disseminated paralysis, etc.
   (2) Peripheral.—Aortic aneurisms, mediastinal tumours, enlarged thyroid gland, diphtheritic paralysis, all through affecting the recurrent laryngeal nerve.

2. Local lesions.—*Ulceration*, due to syphilis, tubercle, or malignant laryngeal tumours affecting the cords.

3. Functional or hysterical paralysis (nearly always affect the *adductors.*)

To sum up. If there be an inability to cough or speak, suspect a serious paralysis. If the *voice is preserved* and the *cough lost*—means unilateral paralysis. If there is normal *cough*, but *no voice*—unimportant. Loud inspiratory stridor, means—double abductor paralysis.

Treatment.—Hysterical aphonia must be treated with galvanism. The other forms must be treated on general principles. For detailed treatment consult a good treatise or manual on the Throat.

DISEASES of the CIRCULATORY SYSTEM.

[The matter printed in small print is taken by permission from Dr Wyllie's original notes and diagrams.—Though printed in smaller type the reader must not think it less important on that account; on the contrary I think it a most essential part of the book.—A. Wheeler.]

PHYSICAL EXAMINATION.

PERCUSSION.

1. In health the area of *Superficial Cardiac Dulness* is of triangular shape; the apex or upper angle (truncated) reaches as high as the fourth left costal cartilage; the right border descends vertically along the middle of the sternum;

the left border passes obliquely downwards and to the left until it reaches the outer limit of the apex beat; the base cannot be percussed out owing to the proximity of the Liver, but corresponds to a line drawn from the outer and inferior limit of the apex beat inwards until it meets the perpendicular limit of cardiac dulness, about mid-sternum. The measurement of the basic line is important; normally it measures about three or four inches.

2. The area of *Deep Dulness*, obtained by heavy percussion, corresponds in shape to the area of superficial dulness, but is more extensive. It overlaps it about an inch on every side.

NOTE.—In Disease the areas of Superficial and Deep Dulness may be specially extended to the right or left, according as the right or left chambers of the heart are specially enlarged.

3. The percussion of the region of the *Aortic Arch* (above the level of the third costal cartilages) is especially important in cases of aneurism and of mediastinal tumour.

PALPATION.

1. Of the *Precordia*.

(*a*) Determine the *position* of the apex beat. The normal position is between the fifth and sixth ribs, about half an inch within the vertical line of the nipple. When the left ventricle is enlarged there is displacement of the apex beat downwards and to the left. When the right ventricle is enlarged there is *apparent* displacement of the apex beat to the right, and there is often pulsation in the epigastrium.

(*b*) Note the *limitation* or *diffusion* of the apex beat.

(*c*) Note the force and character of the beat: whether moderate and deliberate, as in health; or strong and sudden, as in nervous excitement; or strong and slow (heaving) as in hypertrophy; or weak or imperceptible, as in debility.

The range of variation is considerable even in health, owing to the shape of the chest, etc.

2. Of the *Aortic Region*. In cases of suspected aneurism note presence or absence of pulsation.

3. Of the *great vessels at the root of the neck*. Venous pulsation is scarcely palpable, though strikingly visible; arterial pulsation is as strikingly palpable as visible.

INSPECTION.

1. Of the *Precordia* and *Aortic Region*.

(*a*) *Form*. Is there bulging over the precordia or over the aortic region?

(*b*) *Movements*. (1) Movement of the apex beat: its situation, amount, and diffusion; (2) Pulsation in the Epigastrium; (3) Pulsation in the region of the Pulmonary Artery, common in anæmic debility; (4) Pulsation in the Aortic Region, often present in cases of aortic aneurism.

2. Of the *great vessels of the neck.* (*a*) Fulness of the great veins; (*b*) Pulsation in these veins; (*c*) Excessive pulsation in the arteries.

3. Of the *General Circulation*: as exhibited in the patient's complexion, the condition of his peripheral arteries and veins, the presence or absence of dropsy, etc.

4. Of the *Pupils*, in cases of Aneurism of the Aortic Arch.

EXAMINATION OF THE RADIAL PULSE.

1. Give the *Pulse Rate* per minute.

2. Give the *Rhythm:* regular or irregular; if intermittent, note the average proportion of the intermissions to the pulse-beats.

3. Note the size, force, and character of the *Blood-wave:* large, moderate, or small; deliberate or sudden; strong or weak; a double wave (dicrotism).

NOTE.—In *weak heart* the radial pulse may be almost or wholly imperceptible; or only a proportion of the heart's contractions may produce blood-waves sufficiently strong to be propagated perceptibly to the radial artery, and thus the radial pulse may appear to be much slower than the rate of the heart's contractions; or the weak pulse may be affected by the patient's respiration, its beat being weakened by Inspiration and strengthened by Expiration.

4. Between the beats of the pulse, test particularly the *Resistance* of the artery *to pressure.* Marked resistance may be due either to rigidity of the artery's coats or to high blood-pressure. Press the artery firmly against the bone, and examine the coats by rolling them beneath the finger.

5. Compare the two radial pulses, and note any *difference in strength*, or *want of synchronism*, in their beats.

6. In special cases take a sphygmographic tracing.

AUSCULTATION.

ENDOCARDIAL BRUITS.

1st Sound marks beginning of Systole. Systole continues through nearly whole of short pause.

2nd Sound marks beginning of Diastole. Diastole continues through nearly whole of long pause.

1. Aortic—



Systolic.
(Obstructive.)



Diastolic.
(Regurgitant.)

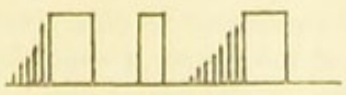


Systolic and Diastolic.
(Double.)

2. Mitral—



Systolic.
(Regurgitant.)



Presystolic.
(Obstructive.)



Presystolic and Systolic.
(Double.)

1. Aortic Bruits. As shown in the above diagram, there may be at the Aortic Orifice, a Systolic bruit, indicative of obstruction, or a Diastolic, indicative of regurgitation; and these bruits are frequently combined so as to constitute a Double Aortic bruit. A Systolic bruit is not always due to organic disease, being sometimes of Anæmic origin, as will be explained below, under "6. The Bruits of Debility." This Hæmic bruit is usually softer than a bruit of Organic origin. Aortic bruits, organic and hæmic, are produced at the Aortic Orifice, which is situated at the Sternal Articulation of the third left costal cartilage. They are thus Basic bruits.

2. Mitral Bruits. These are heard best at the apex of the heart. A Systolic bruit indicates Regurgitation. This may be due either to Organic disease of the Valve, or to dilatation of the Ventricle and its Auriculo-ventricular Orifice, the latter causing a "bruit of debility," owing to "Disparity of size" between the dilated orifice and its valve. The Obstructive bruits are always Organic, being due to stenosis of the Mitral valve. Two forces cause the blood to flow through the Mitral Orifice during the diastole of the Ventricle, viz., the suction of the Ventricle (a *vis a fronte*), and the propelling force of the Auricle (a *vis a tergo*). The suction is strongest near the beginning of the diastole, and the propelling force at the end of it, immediately before the Ventricular Systole. When there is obstructive disease, the bruit is developed at the time when the flow of blood through the contracted orifice is rapid enough to produce a bruit. Generally it is limited to the period of Auricular contraction, and is therefore Presystolic. Sometimes, when the Auricle is weak, its occurs only at the period of greatest Ventricular suction, and is therefore Diastolic. Occasionally, again, it may be both Diastolic and Presystolic. Obstructive bruits are rough and purring. They are often succeeded by the blowing bruit of Regurgitation, since the disease which produces obstruction often renders the valve incompetent at the same time. A common double Mitral bruit is thus a rough, obstructive Presystolic, running up to, and immediately succeeded by, a blowing, Systolic, regurgitant bruit. The relations of these bruits to each other are shown in the diagrams. In sound the rough Presystolic bruit might be represented by the letters *rrrp*, the terminal *p* representing the first sound of the heart, which, in such cases, is often loudly accentuated. A double Mitral bruit may be represented by the letters *rrrfff*.

3. Bruits at the Pulmonary Orifice correspond in time to Aortic Bruits. A *Systolic* bruit in this situation is pretty common. It may be "functional," and due to Hæmic causes, as Anæmia; or it may be due to organic disease that has produced Pulmonary stenosis—a form of congenital heart disease. A *Diastolic* bruit at this orifice is extremely rare.

4. Tricuspid Bruits correspond in time to Mitral. Practically the only Tricuspid bruit that is not extremely rare is a *Systolic Regurgitant*. It is a "bruit of disparity of size," and is due to enlargement of the orifice without corresponding enlargement of the cusps, a condition that is always present when there is much dilatation of the ventricular chamber. With this bruit venous pulsation in the neck is generally associated.

5. In cases of ANEURISM a *Systolic* bruit over the sac is pretty common. In very rare cases a *double* bruit (Systolic and Diastolic) is produced by the flow of blood into and then out of the sac. In many cases there is no bruit at all.

6. The BRUITS OF DEBILITY, so common in Anæmia, are both Vascular and Cardiac.

The *Vascular* bruits are (*a*) The *Arterial* bruit, systolic in time, heard over the great arteries of the neck. This is generally supposed to be common, but it is very often produced artificially by the pressure of the stethoscope. (*b*) The venous hum (the humming-top bruit, or "Bruit de Diable"), heard over the great veins of the neck, and sometimes over other large veins, such as the ophthalmic veins and the cerebral sinuses. This is very common and important.

The *Cardiac* bruits of Debility are variously classified and explained. A view largely supported is that they are *four* in number, one for each orifice of the ventricles: that two are therefore basic and are heard over the Aortic and Pulmonary regions respectively, being probably due to the onward rushing of the thin anæmic blood through the Aortic and Pulmonary orifices; and that the other two, Mitral and Tricuspid, are heard over the left and right apices, and are due to dilatation of the ventricular chambers, which has produced the "disparity of size" between the auriculo-ventricular orifices and their cusps, already alluded to. Both of the latter are thus regurgitant.

Of these *six* bruits (Vascular and Cardiac) *three* are common in Anæmia, namely, (1) the Bruit de Diable in the neck, (2) the Basic bruit in the Pulmonary region, (3) the Mitral bruit at the Apex; and that is the order of their development.

Observe that all the Cardiac bruits of debility are *Systolic* in time. Systolic bruits may thus be either of functional or of organic origin, while Presystolic and Diastolic bruits are always of organic origin.

7. EXOCARDIAL BRUITS. (*a*) *Pericardial friction*, due to Pericarditis, is generally a "to-and-fro" or double bruit (Systolic and Diastolic). It is most apt to be confounded with a double Aortic bruit, but its superficial rubbing and shuffling character generally renders the distinction easy. In most cases it appears first at the base of the heart, and spreads thence, if not arrested, over the whole organ. (*b*) A to-and-fro friction sound, of precisely the same character as the above, is sometimes produced by a *Pleurisy in the Precordial region*, the subjacent heart causing the inflamed surfaces of the pleura to rub against each other synchronously with its own movements.

PROPAGATION OF ENDOCARDIAL BRUITS.

1. AORTIC BRUITS are clearly heard about the third left costal cartilage at its junction with the sternum, that being the position of the Aortic valve. They are propagated to a distance by *three* agents, namely, (*a*) the *Heart* itself, which often carries them to the Apex; (*b*) the *Aorta* and its great branches, a spot of special importance in this respect being the junction of the second right costal cartilage with the sternum (here the Aorta makes it first

bend, and Aortic bruits are often heard even more distinctly than over the valve itself); and (*c*) the *Sternum*, which often conducts the sonorous vibrations of such bruits throughout its whole length. Obstructive Aortic bruits (Systolic) are carried best upwards, in the direction of the blood current, and are specially loud over the first bend of the aorta. Regurgitant Aortic bruits, produced by a descending current, are carried best downwards; and are very often heard better at the left edge of the sternum, close above its junction with the xiphisternum, than even over the Aortic valve.

2. Pulmonary Bruits, starting like the Aortic from opposite the third left costal cartilage at the Sternum, are carried obliquely upwards and to the left, in the second left costal interspace, for a distance of about two inches, the agent of propagation being the trunk of the Pulmonary artery.

3. Mitral Bruits are loudest at the left apex. The Regurgitant (Systolic) is propagated upwards and outwards towards the axilla and the angle of the scapula. The Obstructive (Presystolic and Diastolic) are not propagated in any special direction.

4. Tricuspid Bruits are heard best over the right ventricle, being audible over an area of some inches in diameter, whose centre is situated at the left edge of the sternum, close to its junction with the xiphisternum.

PERICARDITIS.

Inflammation of the pericardium may be primary, but is far more frequently secondary to some infective process.

Causes.—

1. Primary.—It is usually the result of direct injury, but sometimes it occurs idiopathically in children.

2. Secondary.—

(1) Rheumatic fever.
(2) Eruptive fevers, especially *scarlet* fever.
(3) Septicæmia.
(4) Tuberculosis.
(5) Gouty state of blood.
(6) Bright's disease.
(7) Extension of inflammation from neighbouring parts.

Pathology.—That of inflammation of a serous sac already described under pleurisy—*i.e.*,

1. Hyperæmia, with loss of lustre.

2. Exudation of lymph, which coagulates and gives a peculiar shaggy, or "bread and butter sandwich" appearance. The process might stop at this stage causing the dry or *plastic type of pericarditis.* Much oftener the process goes on to the next stage.

3. Effusion of fluid.

4. Absorption with more or less adhesions, which may or may not (as we shall see) subsequently hamper the heart permanently.

The fluid is similar to that described under pleurisy; it may, however, be purulent or hæmorrhagic when associated with tubercle or malignant disease.

Symptoms.—The symptoms are somewhat obscure, and may be masked by the previous existing disease. Taking a typical case as it occurs in the course of rheumatic fever, we usually get—

1. Precordial distress—sharp pain is rare, when present it is most marked at the lower end of sternum.

2. *Dyspnœa, and dusky appearance* of the face.

3. Rapid action of the heart with feeble pulse.

4. Symptoms due to pressure by the fluid on the neighbouring organs (trachea, and œsophagus, etc.)

5. Great restlessness.

It will be easily understood from the above table that we must rely more upon physical signs than subjective symptoms for diagnostic information.

Physical Signs.—

1. *Before Effusion of Fluid.*—On auscultation, is heard the *characteristic "to and fro" friction rub.* It usually begins at the base of the heart and then extends more or less over the whole surface. The friction may be felt by the hand. As the

effusion takes place the friction becomes less pronounced, but is *rarely entirely absent at the base* until complete resolution or organisation takes place.

2. *Effusion Stage.*—The physical signs are—

(1) *Marked increase of the cardiac dulness.*
(2) Displacement of the apex beat.
(3) Muffling of the heart sounds.
(4) Displacement of other organs (if effusion be great).

The shape of the dulness is characteristic. It is conical, *the apex of the cone being at the first rib, owing to the* close attachment of the pericardium to the great vessels. The apex beat is generally pushed *upwards* and to the left. The amount of bulging and displacement of organs will, of course, vary with the amount of fluid present. As resolution takes place the friction returns, and may be very coarse in character.

Diagnosis.—Often difficult in *fat* people suffering from acute rheumatism, and also, when great dilitation of the heart is present. Remember, however, the "*conical*" dulness of the pericardial effusion. The friction rub may be simulated by friction of the pectoral muscle. It is by no means uncommon to detect friction sounds over "rheumatic" muscles. The "pleuritic" rub can be readily distinguished by the difference in time. Do not forget that both pleurisy and pericarditis may however co-exist. See "exocardial bruits."

Prognosis.—Depends on cause, that of simple sero-fibrinous pericarditis is good, the fluid may absorb in from two to four days. Frequently in children though, permanent adhesions may cause some important alterations.

Signs due to permanent and extensive adhesions—

1. The heart remains hypertrophied.
2. A peculiar retraction in the "apex" region during *systole.*
3. Collapse of the cervical veins during diastole (Friedrich's sign).

Treatment.—

1. *Absolute rest in bed.*
2. A fly blister over the precordia.
3. An hypodermic injection of morphia if pain be severe.
4. Treat cause — if due to "rheumatic" poison, push salycilates (Brackenridge).
5. If heart failure threatens, give digitalis, *strychnine*, and diffusible stimulants. Paracentesis (?)

To promote absorption after the acute symptoms have subsided,—

1. Blister.
2. Purge with hydrogogue cathartics—*i.e.*, pulv. jalapæ co.
3. Administer iodides or chloride of ammonia.
4. When these fail, paracentesis should be performed. The puncture should be made one inch from the left sternal margin in the fourth or fifth interspace. If the fluid be purulent, incise and drain.

ENDOCARDITIS.

By endocarditis is meant inflammation of the lining membrane of the valves of the heart. It may be acute or chronic. The *acute* form is divided into the simple and malignant or ulcerative forms.

Ætiology.—Endocarditis is rarely a primary disease, but secondary to other affections, like pericarditis, which we have already discussed; and again we must emphasise the close relationship (before referred to) that exists between endocarditis, acute rheumatism, and chorea.

Malignant endocarditis may be primary, but is much more frequently the result of some septic or profound morbid change in the blood, such as diphtheria, scarlet fever, pneumonia, etc. The malignant form is not nearly as often associated with rheumatism and chorea as the simple endocarditis.

Pathology.—*Simple Form*—

1. Hyperæmia of the membrane of the valves.
2. Exudation of lymph and proliferation of cells.
3. Formation of small granulations.
4. Deposit of layers of fibrin from the blood, the whole process resulting in the formation of small vegetations. These vegetations are most marked at a slight distance from the free borders of the valves—*i.e.*, *those parts which come into opposition during closure.*

Malignant Form.—The initial changes are similar, but there are some important differences, inasmuch as ulcerations may *completely replace the vegetations.* When tabulated, the pathological differences are—

1. The vegetations when present are larger.
2. They have suppurating bases.
3. They contain colonies of micrococci.
4. When they become detached they form *septic* emboli, giving rise to metastatic abscesses.
5. The ulcerative process causes great destruction of the valves.
6. The subsequent or permanent changes in the valves are much more marked.
7. If the vegetation touches the endocardium as it flaps to and fro, the part touched is inflamed from contact.

As regards the side of the heart most affected, it seems that—

1. *Congenital* endocarditis attacks the right side of the heart.
2. The simple endocarditis attacks the left only.
3. The malignant attacks *both sides*, though the *left* is much more implicated than the right side.

The vegetations are upon that side of the valve opposed to the blood stream—viz., at the aortic valve the vegetations project into the ventricle, at the mitral valve into the auricle.

Symptoms.—

Simple Endocarditis.—The signs are extremely ill marked; possibly increased rapidity of pulse, dyspnœa, precordial distress, etc., may attract attention to the heart. On examination a *recently developed murmur* of a soft blowing or bellows-like character may be heard in the mitral or aortic areas.

It should be remembered, however, that in most fevers the heart is somewhat dilated, and a murmur, *not due to endocarditis*, may be present. We must, therefore, be cautious in coming to a too rapid conclusion that a suddenly developed murmur is indicative of endocarditis.

Malignant Form.—Two types are described.

1. *The Septic Type.*—Is characterised by the symptoms of septic infection—viz., rigors, sweats, oscillating temperature, emaciation and metastatic abscesses. Often this form occurs in patients afflicted with chronic valvular disease. The symptoms may continue for months.
2. *The Typhoid Type.* — Is characterised by irregular or intermittent temperature, looseness of bowels, petechial rashes, and a rapid assumption of the typhoid state. Great difficulty may be experienced in diagnosing this form from typhoid fever or meningitis.

It should not be forgotten that patients with chronic valvular disease may get frequent attacks of sub-acute endocarditis.

Treatment.—All forms of endocarditis require absolute rest. The primary disease should be treated, and special treatment directed to prevent heart failure. It is impossible to lay down any hard and fast treatment beyond repeating, *that the utmost vigilance should be kept up for signs of syncope or embolisms*, and such conditions treated on rational grounds. Much that has been said in the treatment of pericarditis may be said of endocarditis.

CHRONIC ENDOCARDITIS.

Chronic Endocarditis is usually the result of an acute endocarditis, but may be sub-acute in its onset, as a result of alcoholism, syphilis, gout, and Bright's disease. Amongst other factors is vascular strain, however brought about. One or more valves may be affected.

Pathology.—In those cases not directly attributable to acute endocarditis, the changes briefly are—

1. Formation of small nodular prominences, with thickening of the valve.
2. Formation of yellowish, opaque, fatty patches.
3. Great increase of fibrous tissue, which subsequently contracts, producing much deformity. The cusps become rigid, curled, and may cause great obstruction to the onward flow of blood, and at the same time fail to accurately close together when required.
4. Great narrowing of the valvular *orifice.*
5. Shortening of the chordæ tendineæ and papillary muscles.
6. Calcification of the fibrosed portion.

EFFECTS OF CHRONIC VALVULAR DISEASE.

We have seen that as a result of inflammatory affections very serious structural changes occur in the valves of the heart. We must, however, consider in detail the effects of such morbid changes. The points which must ever be borne in mind are that normally—

1. By means of the pumping force of the heart, the tissues generally are supplied with oxygenated blood necessary for their perfect nutrition, and to enable the various organs to carry out their functions.

2. The perfection with which organs carry out their functions depends upon the amount of oxygenated blood *passing through them in a given time.*

3. In order, therefore, for the heart to do that which is required of it, it must be perfect in its structure and properly nourished.

We can then have untoward and down-grade changes brought about by—

(1) Interfering with the heart's nutrition.

(2) Valvular defects.

(3) Changes in the innervation.

We shall study these changes in detail, but meanwhile let us bear in mind three other important points.—

1. The normal cardiac mechanism is adapted to meet a certain amount of sudden strain.

2. This reserve power is developed, and *actually increased under increased strain, provided the heart is adequately supplied with* blood, and the strain *gradually* applied.

3. Notwithstanding this grand reserve, a time comes when reserve force must fail, and symptoms of heart failure develop —in other words, hypertrophy keeps up the balance for a time; but ultimately dilatation becomes excessive, the downgrade process commences, to finally end in complete failure. Let us take for an example "aortic stenosis." The first effect will be an extra strain upon the valve and chamber behind. Under the extra strain the chamber dilates *slightly*, the walls hypertrophy *greatly*, and compensation is so thoroughly established that no bad symptom for a time may develop. Finally, however, nutrition of the cardiac muscle fails, dilatation becomes excessive, the valve behind (mitral) proves incompetent, and the aortic symptoms *first* developed *become masked by mitral symptoms.*

HYPERTROPHY OF THE HEART.

We have already seen that hypertrophy of the heart is a natural sequence of increased vascular strain, however brought about, *provided the heart muscle itself receives a sufficient blood supply to keep up its nutrition.*

Causes.—

1. Secondary to valvular lesions.
2. Adherent pericardium.
3. Secondary to diseases of the lungs.
4. As a result of increased peripheral arterial resistance—
   (1) Obstruction of arteries by pressure of morbid growths.
   (2) Atheromatous degenerations, as seen in Bright's disease, gout, etc.
5. Aneurism of the aorta, etc.
6. Over-exertion of a healthy heart, as seen in soldiers, hammer-men, professional runners, etc.
7. Long continued functional excitement.

It will be readily understood that hypertrophy is a compensatory change—*i.e.*, increased growth to meet increased work. But, whilst hypertrophy *per se* is a beneficial condition, it must also be regarded as a distinct weakness; for the patient has already called upon his normal reserve force; and a time comes when the nutrition becomes inadequate, dilatation replaces hypertrophy, and then compensation fails. The man with a hypertrophied heart may be compared to a country menaced and irritated by an enemy; and in order to prevent actual war the reserve forces are called out to supplement the standing army, which causes an additional drain upon the country's exchequer.

Signs of Hypertrophy—

1. Bulging of the precordia.
2. Alterations in the apex beat.
   (1) Its *visible* area is largely increased.
   (2) It is slow and heaving.
   (3) The apex beat may be *felt* in the sixth, seventh, or even eighth interspace *outside* the nipple.

The above, of course, are the signs of hypertrophy of the *left ventricle.*

Hypertrophy of the *right* ventricle is usually due to lung disease and mitral *obstruction.* When it is due to mitral regurgitation, dilatation usually accompanies the hypertrophy. For years the effects of mitral stenosis may be counterbalanced by perfect hypertrophy.

Symptoms of right ventricular hypertrophy.

1. Bulging of the lower part of the sternum.
2. Diffuse "apex" beat.
3. Great increase in the cardiac dulness towards the *right.*
4. Accentuation of the second sound in the pulmonary area.

DILATATION OF THE HEART.

Under "Failure of Compensation" we have already considered the main points in dilatation of the heart. It only remains for us now to consider the more minute changes that occur in this condition. Whilst undoubtedly the main factor in the production of dilatation is *obstruction*, either through valvular disease or increased peripheral resistance, still, we must remember that dilatation is hastened and even brought about by degenerative changes in the ventricular *walls*, such as—

1. Fatty degeneration.
2. Fibrosis.
3. Anæmia.

High temperature has already been referred to as a frequent cause of dilatation.

Anatomical Changes.—The heart is more globular in shape. The walls are much thinned, especially at the apex. The auricular-ventricular valves are usually markedly incompetent.

Physical Signs.—As described under hypertrophy, the precordial dulness is much increased, and the apex beat displaced, etc.; but in contra-distinction to hypertrophy the *impulse and heart sounds are very feeble* in pure dilatation.

Symptoms will be summed up under "Incompetence of Valves."

VALVULAR LESIONS.

We shall have to reiterate much that has already been said under endo-carditis. We have already seen that each valve may be affected in one, two, or three ways—viz.,—

1. They may be rendered obstructive—stenosis.
2. They may be incompetent—regurgitation.
3. Incompetent and obstructive.

These conditions are diagnosed by—

(1) The position of the murmurs accompanying such conditions.

(2) The general symptoms produced by such lesions.

Murmurs are propagated in the direction of the blood stream, and are best heard a little way from the valvular orifice where they are produced.

Always in describing a murmur, state—

1. Position.
2. Time.
3. Propagation.
4. What sound (if any) is modified thereby.
5. Accompaniments (if any).

Examples—

Aortic Stenosis.—A murmur heard in the aortic area, systolic in time; propagated up the sternum into the carotids, and usually *accompanied* by a loud first sound.

Aortic Regurgitation.—A murmur heard in the aortic area, *diastolic* in time; propagated *down* the sternum, and modifying or replacing the second sound.

Mitral Stenosis. — A murmur heard in the mitral area, *pre*-systolic in time; *runs up to the* first sound—not propagated, but often accompanied by a marked thrill.

Mitral Regurgitation.—A murmur heard in the mitral area, systolic in time; propagated to the axilla, *and modifying or replacing* the first sound.

Tricuspid Regurgitation.—A murmur heard over the fourth right costal cartilage; propagated to the left and towards the apex beat; systolic in time.

Other murmurs are too rare to require a description in such a book as this. One cannot help thinking two much importance is paid nowadays to the localisation of murmurs, and *too little* notice taken of the state of the heart nutrition. Post-diastolic and mid-diastolic murmurs are often diagnosed with lightning rapidity by the student, who, at the same time, fails to note whether compensation is established or beginning to fail. Perhaps this fault after all is due to faulty teaching. Re-duplication of sounds indicates a want of synchronism between both sides of the heart. Remember, the second sound is produced by the closure and stretching of the aortic and pulmonary valves; the first sound, by closure and stretching of the auriculo-ventricular valves, *plus* the *contraction of the ventricular muscle.* Lastly, do not forget that the loudness of a murmur is but a poor index of the gravity of the lesion. A careful consideration of the general state of the heart and *general symptoms* is of far greater importance than mere attention to any murmurs that exist. The diagnosis of cardiac lesions is sometimes extremely

easy, and at other times very difficult. Aortic disease may be masked by mitral disease; mitral disease, by extensive pulmonary disease, and so on. Remember that the tendency of all organic cardiac lesions is to *finally produce arterial emptiness and venous congestion.* The following table will show the more important differences.

AORTIC DISEASE.	MITRAL DISEASE.
Symptoms are mainly due to anæmia—viz.,—	Symptoms are mainly due to venous congestion—viz.,—
1. Pallor.	1. Cyanosis.
2. Throbbing of the carotids.	2. May get pulsation in the veins of the neck.
3. Attacks, of an "angina pectoris" type of pain.	3. Sudden attacks of severe dyspnœa — actual acute pain is rare.
4. Breathlessness on slight exertion.	4. Breathlessness, but often present without exertion.
5. Menses usually absent.	5. Often there is menorrhagia.
6. Nervous symptoms are prominent.	6. Pulmonary symptoms are most prominent—*i.e.*,— (1) Cough in the morning. (2) Chronic Bronchitis. (3) Hæmoptysis. (4) Dilated right side of heart, and later— (5) Symptoms due to tricuspid leakage.
7. The left ventricle is usually much hypertrophied.	7. The left *auricle* is much hypertrophied.

Tricuspid Disease.—The symptoms of failure of the right side of the heart may be summed up as—those symptoms which always arise from imperfect æration of blood, and venous congestion of the organs, generally. Those in connection with the congested organs are—

1. Lungs = dyspnœa, bronchitis, pulmonary œdema.
2. Liver (nutmeg) = dyspepsia, hæmatemesis, ascites.
3. Kidney = albuminuria, general dropsy, uræmia.

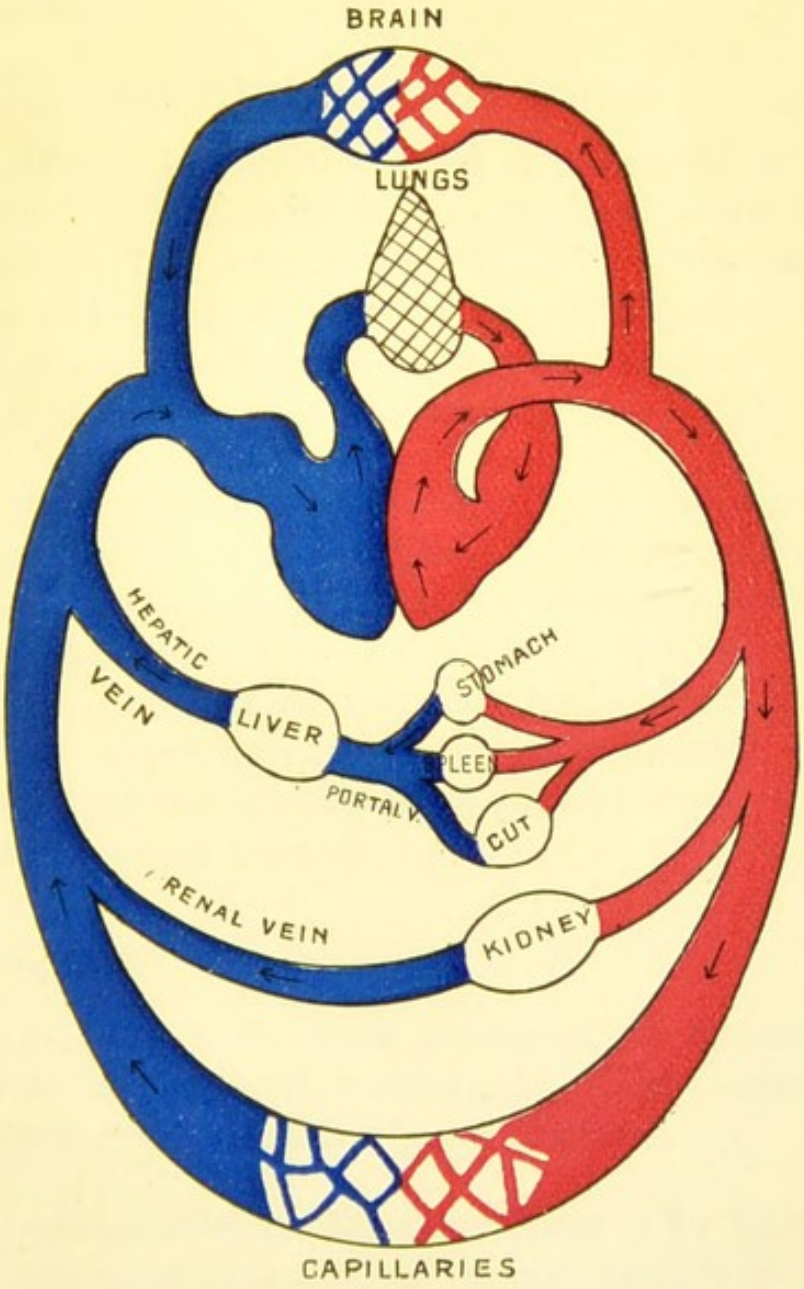


Diagram for studying the results of backward pressure. Note the areas in blue will become the seat of changes consequent on venous congestion.



The symptoms are, in fact, merely an exaggeration of those arising from mitral disease. The patient finally becomes water-logged. Locally there is epigastric pulsation. The jugular vein, if obstructed by the finger, *fills up from below during systole.*

THE PULSE IN CARDIAC LESIONS.

Aortic Stenosis.—Here the blood is obstructed in its flow into the aorta. The pulse is thus rendered somewhat slow in its ascent. The tension will depend on the amount of obstruction and degree of hypertrophy. The force with which the blood is sent through the narrow orifice often causes an aneurism of the first part of the aorta.

Aortic Regurgitation. — The ventricle in this condition is filled during diastole by *two* streams—one the regurgitant aortic stream, and the other in the usual way from the auricle. The chamber is thus filled more quickly, and the pulse made to beat faster. The sudden rise and the sudden fall of the pulse gives it a peculiar kicking or water-hammer character. The throbbing of the carotids often prevent sleep. *Capillary pulsation is often well marked.*

Prognosis—

Aortic Stenosis is the least dangerous, as hypertrophy is usually well marked and maintained. Often there are no symptoms suggestive of such a grave condition.

Aortic regurgitation on the other hand is by far the most dangerous lesion; sudden syncope through anæmia of the brain is often fatal. Angina pectoris is common in this lesion and may cause speedy death.

Mitral disease is characterised by frequent breakdowns, but patients often quickly recover their usual condition and may be long lived.

Tricuspid disease is always grave and it is impossible to give any general prognosis. Each case must be carefully considered on its own merits.

Treatment.—The beginner has my full sympathy in trying to understand the rational treatment of cardiac lesions. He is taught by some that "*rest*" is the best thing—by others, "*muscular exercise*" is the proper treatment,—again "*Digitalis*" is the *only* drug, or "*Strophanthus*" must be the sheet anchor; and lastly *use the whole lot* if he wishes to be successful. Possibly the last is correct; but at any rate, no *routine* treatment can possibly be prescribed. Remember, however, a few points and the indications for any particular treatment will soon be apparent.

1. Hypertrophy is nature's cure; and, in order

2. To get hypertrophy there must be a wholesome supply of blood to the heart tissue.

3. Remember the golden rule "avoid putting excessive strain on a diseased tissue."

4. "Don't" look upon the heart as an isolated structure, but as a *component and essential part* of a complex machine; and consequently sharing in the anabolic and catabolic changes of the body generally. In other words, if the system generally be lowered, then the heart must also suffer; but this does not prevent us from using those drugs which have a distinctly and particular action upon the heart. Our treatment must be then—

GENERAL AND MEDICINAL.

(1) *Diet.*—Avoid excesses, especially of nitrogenous food which tends to increase *peripheral resistance.*

NOTE—A full or distended stomach is a constant source of cardiac embarrassment.

(2) *Exercise.*—Provided the *assimilative powers are good, and plenty of food can be taken to provide* for the *increased*

expenditure of energy, then exercise must be good, as it imitates or helps the natural way of producing hypertrophy of the chambers behind the lesion; on the other hand

(3) *Absolute rest is the treatment* when the assimilative powers are weak, or compensation once established is failing.

(4) *Drugs* include (1) general tonics, and (2) those having a special action on the heart. Of the former, iron and arsenic with strychnine is always a valuable mixture.

Of the latter digitalis stands first. It acts beneficially by—

(*a*) *Decreasing* the rapid action of the heart, so increasing its natural rest, and diminishing its work in a given time.

(*b*) It aids the nutrition of the heart, which is supposed to be nourished during diastole. (?)

Its disadvantage is, that it increases peripheral resistance, and so produces greater strain. Digitalis can be given in *all* cases where the arteries are empty, and the pulse *quick and irregular*. Especial care, however, is required in aortic regurgitation for fear of prolonging diastole too long. "Don't" give digitalis, though there be cardiac disease, if compensation is well established; and secondly "don't" continue giving digitalis if the urine decreases in quantity after its administration.

Strychnine is always a powerful adjuvant to digitalis, and is possibly the best "cardiac tonic."

Strophanthus is held in high repute in Edinburgh, in cases where digitalis fails; it is supposed to have similar tonic effects to digitalis without the drawback of increasing peripheral resistance; opinions as to this are conflicting. Sudden breakdowns of compensation call for stimulants such as æther, ammonia, brandy mixture, *complete rest*, cupping over the lungs, etc.

Cardiac pain is best relieved by morphia and iodide of potassium. If there be arterial constriction, nitrite of amyl is

the best remedy. Do not forget the judicious use of an emetic in "mitral" disease when sudden dyspnœa occurs after a meal.

A Caution.—If your are called to see a patient who has been picked up insensible, and smells strongly of alcohol, do not hastily conclude he is drunk, but *make sure of the state of the heart.* Many patients finding themselves getting faint, take a dram of brandy, which, however, may fail to prevent syncope. Rough usage or a cold cell would probably kill a patient under such circumstances.

ANEURISM.

Before discussing the essential features of thoracic aneurism we must recall a few facts relative to *aneurisms in general.* An aneurism may be defined as "a localised and persistent dilatation of a blood vessel." Surgeons employ the term in a wider sense. Classified according to shape we have the following varieties—

1. Fusiform or spindle shaped, involving the *whole* circumference.
2. Sacculated — where one side of the vessel only is dilated.
3. Diffuse—where a large portion of the vessel is irregularly dilated.

Causes.—The two main factors are—

1. Damage to the vessels walls.
2. Increased vascular strain.

Consequently we shall have to consider predisposing, and exciting causes when dealing with any particular aneurism.

Pathology.—

The inner coat may be much thickened by atheromatous changes, but much more frequently it disappears altogether, a delicate layer of hyaline tissue taking its place.

The muscular coat.—The muscular fibres become stretched and dissociated, and ultimately disappear.

The outer coat becomes much fibrosed, and adherent to the surrounding tissues by an inflammatory process; thus the "sac" of an aneurism is most frequently composed of dense fibrous tissue.

Course.—An aneurism once started tends to increase, and, if not arrested, finally ruptures. But an aneurism may cure itself by becoming so large that the sac, by pressing on the artery, checks or even obliterates the blood supply. Again, when the orifice is small in a sacculated aneurism the circulation in the sac may be so impeded that thrombi form, ending in a large pale laminated clot, which may ultimately organise and transform the aneurism into a fibrous nodule. This occurs, however, only in the smaller aneurisms; but this natural cure gives the physician the cue as to what he should attempt.

Symptoms.—The cardinal symptoms of an aneurism are—(1) Tumour. (2) Pulsation. (3) Systolic murmurs. (4) Pain. (5) Other pressure effects.

The extent of these symptoms depend upon the site of the tumour and the nature of the adjacent structures. A word of caution is needed to guard the beginner against diagnosing *an aneurism of the aorta too quickly;* frequently there are no symptoms indicative of such a serious condition, and on the other hand, there are often grave and suggestive symptoms present, but *no* aneurism. We shall now consider the special points of

THORACIC AORTIC ANEURISM.

The aorta is the most frequent seat of aneurism in the body, and all varieties have been found here. There are

many reasons given why aneurisms should be so common in the arch of the aorta—

1. It is much curved.
2. The first part of the arch has very little support.
3. The blood stream ejected during systole of the heart, tends to bulge the aorta locally (especially in aortic stenosis).
4. This part is much more affected by the variation of the cardiac pressure than the distal arteries.
5. It gives off large branches in a very small area.
6. The vessels tend to dilate during inspiration.

Occurs most frequently amongst men who are either prematurely old through intemperance, syphilis, etc.; or in those engaged in occupations which tend to increase the normal aortic strain, such as hammermen, brewers, young soldiers, etc. Septic emboli lodging in the inner coat account for some cases. Traumatisms may also cause aortic aneurism.

Symptoms.—They depend on the portion of the arch affected, and size and shape of the aneurism.

Briefly they are—

1. *Symptoms in connection with the Circulation.*—Palpitation, angina pains, the arteries are filled less perfectly, and there may be a difference in the two radial pulses.

2. *Symptoms due to Pressure*—

(1) *Œsophagus.* — There is difficulty in swallowing, especially solids.

(2) *Respiratory System.*—Less air enters the lung pressed upon, and the breath sounds are consequently weak. There is much dyspnœa, or attacks of the so-called aneurismal asthma, a peculiar alteration of the voice and cough (leopard growl and gander cough). Hæmoptysis may occur later due to "weeping" of the aneurism.

(3) *Implication of Nerves.*—The symptoms will depend on the amount of pressure exerted on the nerves. Thus, if *slight*, we get symptoms due to *irritation*; if *severe*, symptoms due to *paralysis*. The nerves most likely to be involved are—

	IRRITATION.	PARALYSIS.
Left Recurrent Laryngeal	Alteration of voice; and stridor, due to spasm.	Aphonia.
Phrenic	Painful and persistent hiccough.	Intercostal breathing. Death.
Sympathetic	Dilatation of the pupils. Palor from constriction of the vessels on the one side. Rapid action of the heart.	Contraction of the pupil and flushing of that side of the face.
Vagus	Depressed action of the heart. Vomiting and nausea.	Irregular action of the heart. Pneumonia. Death.

Veins.—Œdema of superior extremities, one side of the head, etc.

Thoracic Duct—produces rapid emaciation, and fatty stools.

Bones—are eroded and absorbed; the process being accompanied usually with intense boring pain. When the spine is involved the pain is intense, due to irritation of the intercostal nerves and meninges—there may be much deformity, and even paraplegia through implication of the spinal marrow—a murmur may be heard over the spine.

Such then are the clinical symptoms; on

Physical Examination.

Inspection may reveal a swelling; or there may be a diffuse heaving impulse above the third costal cartilage, and displacement of the apex beat.

Palpation often detects the expansile character of the tumour, with its *systolic* thrill and diastolic shock.

Percussion.—Note is dull or flat and gives a feeling of resistance.

Auscultation may reveal, over the dull area, a ringing accentuated second sound and a systolic bruit.

Dr Osler calls attention to the absence of pulsation in the abdominal aorta or the femorals in cases of large thoracic aneurisms.

Surgeon-Major Porter lays stress upon the fact that if the chin be brought forward to relax the neck, and the cricoid jerked upwards, there is felt a peculiar tugging if the aneurism be attached to the bronchus or trachea. He says the same feeling is not produced by any other tumour.

The following table shows the structures most likely to be implicated according to the site of the aneurism :—

ASCENDING.	TRANSVERSE.	DESCENDING.
Right pleura.	Rarely external tumour.	Œsophagus.
Second and third interspaces.	Both Pluræ.	Thoracic Duct.
Sternum.	Sympathetic.	Erosion of spine.
Superior Vena Cava.	Trachea.	Often ruptures into pleuræ.
	Left Bronchus.	
Right recurrent Laryngeal Nerve.	Left recurrent Laryngeal Nerve.	
Left pulmonary artery.	Aneurismal Phthisis.	
Rupture into *pericardium.*	Ruptures outside the pericardium.	

Treatment.—

General.—Everything must be done to quiet the circulation, by the observance of rest and abstinence from all alcoholic drinks, etc. The diet should be nourishing, but limited, and *the quantity of liquid taken reduced to a minimum.*

Medicinal.—Anodynes and sedatives are called for, but iodide of potassium in large doses daily, seems the most efficacious of drugs. It is difficult to say how the iodide acts, it certainly does not lower the blood pressure to the same extent as many other drugs which are *less* efficacious in benefitting the aneurismal condition. Possibly the beneficial effects are due to the iodides causing absorption of inflammatory material, which would otherwise induce sclerosis.

Local treatment is highly unsatisfactory. All methods aim at starting coagulation. Briefly, they are—

1. Introducing needles, and scratching the walls of the sac to form a rough surface, and so bring about coagulation.
2. Introduction of fine steel wire or horse hair.
3. Electrolysis.

Considering the hopeless character of this affection, it is more than cruel where pain is severe to withhold morphia, as some anti-opium fanatics would have us do.

ABDOMINAL ANEURISM.

Site.—Most common near the cœliac axis. It may grow upwards and push the diaphragm before it, or forwards and project anteriorly, or backwards and erode the spine.

Diagnosis.—Palpation reveals a definite tumour with the characteristics of aneurism already described. The tumour may be better felt in the knee and elbow position.

Especial care must be taken in diagnosing this condition in pregnant or hysterical females; also in cases of tumours lying *beneath* the aorta.

Treatment.—As in thoracic aneurism. Compression under chloroform may be tried, though it is decidedly risky.

Prognosis.—Bad. Death may take place from—

1. Compression paraplegia.
2. Embolism of superior mesenteric artery.
3. Complete obliteration of the lumen by clots.
4. Rupture (oftenest into duodenum).

CONGENITAL ANEURISM.

A peculiar condition of the arteries, characterised by congenital deficiency of the muscular coat, thickening of the intima and a small cell infiltration of the outer coat, causing a nodular condition. The coronary arteries are most affected.

FUNCTIONAL DISORDERS OF THE HEART.

The functional disorders include—

1. Palpitation—*i.e.*, forcible and often irregular action of the heart, perceptible to the individual.
2. Arrhythmia, or intermittent condition of the cardiac beats.
3. Tachycardia or rapid heart.
4. Bradycardia or slow heart.

[For the details of these various conditions, the reader is referred to a standard work on physiology.]

Rapidity of the heart is usually associated with a neurotic temperament, menstrual epochs, excessive use of stimulants, mental excitement, use of certain drugs, such as belladonna, etc.

Slowness of the heart is most commonly associated with hunger, the puerperal state and melancholia, use of digitalis, excessive use of tobacco, poisoning *by bile, lead, etc.* Often, however, the same conditions will produce rapidity in one individual, and slowness in another.

ANGINA PECTORIS.

A condition characterised by a sudden severe pain in the chest, with a sense of impending death.

Ætiology.—It occurs most frequently amongst men above the middle age. Predisposing causes are "*all conditions which interfere with the nutrition of the walls of the heart:*" such as extensive fatty disease, valvular diseases, atheroma, however brought about, etc.; the exciting causes are:—

Sudden strain, an over distended stomach, powerful emotional disturbances, etc.

Symptoms.—The patient is suddenly seized with an acute and sudden pain across the chest. The pain is most marked at the lower end of the sternum and radiates down the *left* arm

most frequently, though the *right* arm is often implicated. The patient feels as if an iron band was fixed around the chest. The feeling of suffocation is intense, but the usual cyanosis of dyspnœa is absent, the face often being extremely pale. A sphygmographic tracing of the pulse taken during an attack, shows increased arterial tension. An attack may last from a few seconds to many minutes; may kill the first time, or recur at various intervals.

Pathology.—*Post-mortem* — the heart is distended with blood, and the walls and valves show extensive morbid changes. The coronary arteries are extensively diseased.

Causation of the Attack.—Many people have extensive heart disease without getting angina. Again, a patient may have only one attack, or have an interval of years between them, so, *per se*, heart disease is *not of itself sufficient to produce angina.* We must confess that at present the explanations of the condition given are not altogether satisfactory. The following statements possibly show the extent of our knowledge at present. Atheromatous coronary arteries cause—

1. More or less anæmia of the heart's substance.
2. Irritability of the intra-cardiac ganglia.
3. Diminished contractile power of the ventricles.

Let us suppose a heart with all these disadvantages is subjected to a severe strain. What will result?

1. Over distension of the ventricle.
2. A reflex message will be transmitted from the irritable ganglia to the medulla, resulting in sudden *general vasa motor constriction.* Therefore, the heart *already* embarrassed, has to pump against *increased* peripheral resistance, hence the feeling of suffocation, etc.

If this be true, then the use of nitrites is at once apparent, for, as they cause dilatation of the arterioles, the peripheral resistance *is at once diminished*, and the over distended ventricle allowed or enabled to empty itself.

Indeed, the only flaw in the foregoing explanation is, "That in some instances of angina no increase of arterial tension has been observed." This is but a poor argument though, for we can easily understand that the propelling force of the heart may be insufficient to cause any *appreciable tension*, notwithstanding the increase of peripheral resistance.

Treatment.—During the attack administer nitrites, preferably by inhalation; chloroform may be used as a substitute. During the interval, careful attention to the bowels, diet, and avoidance of severe mental or muscular exercise, with the administration of iodides, considerably lessens the chance of *another attack*. Lastly it must be remembered that many patients complain of something like angina, which, however, is not the true disease.

TRUE ANGINA.	PSEUDO ANGINA.
Most common past middle life.	At every age from six years.
Most common in men.	Most common in females.
Attacks—rarely nocturnal or periodical.	Often periodical and nocturnal.
Not associated with other symptoms.	Associated with *nervous* symptoms.
Agonising pain and sense of constriction.	Pain less severe—distension more than constriction.
Pain of short duration.	Pain lasts one or two hours.
Lesions—arterial sclerosis.	Neuralgic affection.
Prognosis grave; often fatal.	Never fatal.

(Huchard's table.)

EXOPHTHALMIC GOITRE.

Exophthalmic Goitre is a remarkable condition characterised by—

1. Rapid beating of the heart and palpitation.
2. Enlargement of the thyroid gland.
3. Exophthalmos or projection of the eyeballs.
4. Imperfectly co-ordinated movements of the upper eyelid.
5. Certain visceral disturbances.
6. Atrophic changes in the epidermal structures.

Pathology.—Quite unknown, and until we know more of the physiology of the sympathetic system, very little beyond speculation can result. *Post-mortem*—no constant or definite changes are found. Professor Grainger Stewart and Dr Gibson point out "no advantage can accrue from a discussion in favour of the disease being due to either—

1. A paralysis of the vagus.
2. Stimulation or paralysis of the sympathetic.
3. Changes in the medulla."

Any one who knows anything of physiology will endorse this honestly expressed opinion, and I cannot help expressing here, how much better it would be, if the heads of our profession would abstain from dogmatically expressing any definite opinion on a subject "*sub judice*"; and still more, how unjust it is for an examiner to ask a candidate for explanations of such difficult pathological problems.

Filehne says:—Injury to the upper part of both restiform bodies in *rabbits*, will produce all the characteristic symptoms. It may be so in rabbits, but I was once present at the post-mortem of a patient where extensive disease of the restiform bodies was found, and no such symptoms were evident during life. For examination purposes it should be remembered that exophthalmos may be produced by—

1. Stimulation of the sympathetic.
2. Over growth of the fat in the orbit.
3. Contraction of the muscular fibres of Müller in the lining membrane of the orbit.

Symptoms.—The patient is usually nervous, and complains of headache, vertigo, and great weakness. The prominence of both eyeballs gives a terrified aspect to the patient; the eyelids fail to meet and the upper eyelid is *not depressed when the patient looks down.* The temperature is periodically raised, and marked dyspepsia is often present. Diarrhœa is also a prominent symptom in many cases. Professor Grainger Stewart and Dr Gibson emphasise the marked changes in

the nails, which are brittle, corrugated, etc., and also other changes in the skin or its appendages, dependant on vasa-motor disturbance. The thyroid gland is enlarged as a whole, due to a general hyperplasia; the minute changes are by no means identical in all cases. The menses are usually absent.

Prognosis.—Bad. Excessive dilatation of the heart often brings about a fatal result after exertion. Hæmorrhages, diarrhœa, or other complications, usually terminate the disease in a few years after the marked symptoms appear.

Treatment.—Must of course be symptomatic. Iron is badly borne, and the amount of anæmia does not particularly suggest its use. Large doses of digitalis may be tried.

Belladonna. Thyroid. Sodii Phosph 10 grs t.i.d.

DISEASES OF THE LIVER AND KIDNEY.

Before attempting the study of diseases of the liver and kidney, the reader is earnestly advised to recall to mind, the chief physiological facts regarding their functions and *relations to each other*. It will be remembered that the kidney is to a large extent the hand servant of the liver; it excretes the waste material formed by the former, and thus enables the liver to perform its duties efficiently, and, at the same time keep the blood free from morbid products. But it must also be understood that an alteration in their perfect physiological relation may be disturbed by—

1. The liver putting excessive strain on the kidney; or,
2. The kidney, through disease, being unable to perform its normal functions, and thus clog the liver.

FUNCTIONS OF THE LIVER.

Will be considered briefly under four heads—

1. The metabolism of proteid material.
2. The metabolism of carbo-hydrates.
3. The manufacture of bile.
4. As a source of heat and energy to the body generally.

If we study these carefully, though briefly, it will be easy for even a beginner to understand those facts which are necessary, in order to get a grasp of "*how*" such conditions as gout, diabetes, Bright's disease, jaundice, and so on, are brought about. I must however warn the student, that though the following statements represent our present knowledge of such metabolic changes, they must be regarded as distinctly hypothetical, though sanctioned by the latest research.

1. PROTEID METABOLISM.

Proteids, after being subjected to the various digestive agents, saliva (mastication), gastric juice, pancreatic juice, and succus entericus, are finally absorbed into the portal circulation; carried to the liver, and there broken up or divided into (1) That which is to be used for building up the tissues. (2) That which is to be excreted. That which is excreted goes through many changes before it can be eliminated *via* the bile and urine. Graphically we might represent such changes as follows.

Proteids = substances of an albuminous nature, rich in nitrogen. *Under the action of*—

1. Saliva,—they are finely divided or masticated;
2. Under the action of gastric juice they are converted into—

(1) Albuminates (acid).
(2) Albumoses (many kinds). } = Acid chyme.
(3) Peptones.

3. Under the action of pancreatic juice and succus entericus they are converted into—

(1) Alkali albuminates.
(2) *Albumoses.*
(3) Peptones.
(4) Leucin, tyrosin—*to a slight extent.*

Skatol, indol, and phenol—*bodies which result from putrefaction* (of course under bacterial influence) are also formed, especially if absorption be delayed.

Proteids are thus (if fully digested) converted from non-diffusible into diffusible bodies termed "peptones," with the addition of some *putrefactive bodies* (which ordinarily should be excreted *via* the fæces). We thus see even at this stage how powerfully constipation can affect the system, by *increasing the formation* of these poisonous products, and by the same delay, increase the chance of their absorption, and thus break down corpuscles and cause profound anæmia. The further fate of peptones is wrapped up in much obscurity. Possibly the following table is correct as far as our present knowledge goes.

FATE OF PEPTONES.

Passing through the intestinal walls they are converted into albuminous bodies, and carried by the portal circulation to the liver.

In the liver they are broken up into that which is used for nourishment, and that which is to be excreted. That which is to be excreted undergoes the following changes :—

	Proteid Waste.		*Result of Retention.*
1st Group of Compounds.	Leucin. Tyrosin.		(See Acute Yellow Atrophy of the Liver.)
2. Intermediate Products.	Creatinine or Zanthin Group.	All having NH_3 for a base like the vegetable Alkaloids.	(See Uræmia.)
3. End Products.	Uric Acid..... Urea.		(See Gout. Chronic Bright's.)

In other words, within the liver, the waste products are converted into a very fine ash termed urea, which escapes into the blood, and is then excreted by the kidney. Nearly all the nitrogen is thus excreted from the body.

Apply this to disease.—*A failure on the part of the liver to do its work must result in imperfect oxidation of waste products.* The nature of the products so formed will depend on the extent or degree of oxidation. Thus a *slight* failure would mean excessive uric acid formation, and probably cause gout. A more serious failure would cause retention of the intermediate ammoniacal products, and cause *uræmia.* Whilst a still more serious handicapping of the liver would cause leucin and tyrosin to appear in the urine, as seen in acute yellow atrophy of the liver. We have seen that the perfection with which any organ performs its function depends on—

1. The quantity of oxygenated blood that passes through the organ in a given time.
2. The rapidity with which the *waste* products formed are excreted. (Every living act builds up and breaks down—*i.e.*, anabolic and katabolic changes.)

We can easily reason then, how much, vasa motor changes can affect an organ's nutrition, and consequently its work. Further, imperfect oxidation means the formation of a *coarser "ash" as it were*—*i.e.*, *uric acid* instead of *urea*, and as the kidney has in such cases to excrete highly irritating particles, "is it any wonder that it becomes fibrosed, and the red granular kidney form of chronic Bright's disease results?" Can we not easily understand that such irritating products floating in the blood must cause vasa motor peripheral constriction, and so heighten blood tension?—finally causing sclerotic changes in the vessels, and hypertrophy of the heart to overcome the obstruction? Further, will not such a state begin a cycle of *down grade* changes? Just a few more words in reference to the intermediate products or Zanthin group. We find there are many of these compounds, but all seem to have (like the vegetable alkaloids) NH_3 for a base. Now we know that different alkaloids have different poisonous and medicinal qualities, thus strychnine and morphia have

very different properties, and though both may cause death, they do so in totally different ways. Can we not grant the same thing in reference to the antecedents of urea? Surely *one* of these products may be more abundant than another! If so, we can easily understand why in uræmia some cases are attended with convulsions, whilst other cases are not. So much for proteid metabolism, as far as it is possible to reason at present. Do not forget, however, that the *down grade changes* just pictured, though described as beginning in the liver, we must allow that deficient excretion on the part of the kidney may be the *starting* point.

METABOLISM OF CARBOHYDRATES.

Under the action of—

Starch is converted into

1. Saliva.

Achroo-dextrine and Erythro-dextrine

2. Lactic Acid. (?) — Maltose.

3. Pancreatic Juice. — Dextrose.

4. Succus Entericus. — Lævulose—Dextrose.

Absorbed into portal circulation.

5. In the Liver. — Glycogen, and pass out when required into the Blood as Dextrose again: then in the

6. Tissues. (Muscles, etc.) — CO_2 \ Energy. H_2O / Heat. (Final Products.)

We see by the above table that carbohydrates, after passing through various changes, get to the liver as grape sugar. In the liver a portion is, however, stored up for a time, *not as sugar*, but as a *peculiar starch, termed glycogen.* This glycogen is again broken up by the liver cells into grape sugar or glucose; and, then by means of the blood

stream is conveyed to the various tissues, where it is finally oxidised to CO_2 and H_2O. Supposing the liver is too richly supplied with sugar, what might happen? It might store up a large amount as glycogen, but allow a large quantity to pass on unchanged. The blood would then be surcharged with sugar; the muscles say, "we do not want so much:" there is an increase of the supply over the demand—"a glut in the market" as it were, and the sugar thus rejected by liver and muscles is excreted by the kidneys, and *glycosuria* results. But supposing the liver did not allow the sugar to pass too quickly or unchanged, but the tissues through some wide spread neurosis or other condition, were *unable to use the normal amount of sugar:* then we should get glycosuria *plus wasting of muscles;* or in other words, *diabetes.* We thus see that glycosuria is a temporary excessive supply; diabetes on the other hand is a serious organic disease. But we must remember that a mere excess of sugar persistently floating in the blood must act as foreign matter and induce changes in the tissues by its irritating action; so a continued glycosuria may induce real diabetes. Other theories of diabetes will be discussed under that disease.

3. *Bile Formation.*—Bile consists principally of water, cholesterine, salts, bile pigments and mucin. It is to be regarded as—

(1) A secretion—concerned in the proper assimilation of fats, etc.

(2) An *excretion*—a means by which broken down blood pigments are excreted, with also a certain amount of proteid waste (glycin etc.)

FATE OF BLOOD PIGMENTS.

In the Liver. Hæmoglobin is converted into—
Hæmatoidin into—
Bile Pigments which are—

Excreted as— Stercobilin. (Fæces) | Urobilin. (Urine.)

The pigment of bile being derived from hæmaglobin, *per se,* we must have a larger amount of bile pigment in those diseases attended with the *destruction of red corpuscles.* Now bile pigment increases the thickness of bile, and Quincke has shown "When there is great destruction of red corpuscles in the liver, mucin is also increased, which in turn materially increases the viscosity of bile." Thus in pernicious anæmia the bile is rendered thicker by—

1. Increased pigment.
2. Increased mucin.

If we increase the thickness of a fluid running through a tube, we do two things—

1. Retard the flow.
2. Cause a greater tension of the tubal walls.

Now remembering all this takes place in the liver in all exhausting diseases, it is no wonder we get a sallow complexion, for the bile tension being higher than normal, there is every facility for transudation of bile into the blood. This is well seen in pernicious anæmia. *Probably all cases of jaundice are obstructive,* not indeed *actual stoppage* or suppression of bile, but through the increased tension within the bile ducts. Brunton has done much to throw light on this subject. Lastly, in summing up the functions of the liver, we must remember exercise and moderate eating and drinking must increase its functional activity; then the bile moves on more quickly, glycogen is properly stored up, and though more is formed than usual, it does not accumulate, as the muscles use it up more rapidly to furnish fuel for their increased work. Urates *decrease* and urea *increases.* The reverse must happen by sluggish habits and intemperance. *Local vasa dilatation* as a result of disease may also *increase for a time the functional activity,* but soon, venous congestion would occur under such circumstances, and torpidity succeed the activity.

JAUNDICE.

Jaundice is the name applied to a group of symptoms, arising from the circulation of bile in the blood. We have already pointed out that bile is to be looked upon as a secretion and also as an excretion. Its retention within the liver and its absorption into the blood must therefore give rise to symptoms due to—

1. *Its absence from the intestines.*—This interferes largely with the perfect assimilation of fat, and the imperfect absorption causes serious decomposition or putrefactive changes to take place in the intestines, skatol and indol are formed in excessive quantities, *complete* proteid digestion cannot go on, and thus we get absorption of albumoses and other poisonous compounds, which contribute largely to the serious blood changes to be next described.

2. *The circulation of bile within the blood* causes destruction of red corpuscles, and therefore anæmia; bile is also a neuro-muscular poison, paralysing both nerve centres and muscular fibres; consequently we get imperfect action of the heart, slow pulse, depression of the spirits, mental torpidity, and possibly profound coma. Two types of jaundice are described — viz., obstructive and non-obstructive.

In the former case no bile enters the intestine, and the seats of obstruction are probably in the large ducts. By non-obstructive jaundice is meant a condition of icterus in which a certain amount of bile escapes into the intestine. We have already shown how the increased viscosity of bile resulting from excessive breaking down of red corpuscles, might raise the bile pressure sufficiently to cause absorption of bile into the blood, and it is highly probable that all the so-called cases of non-obstructive jaundice are due to this condition, and a much better name would be Hæmatogenous Jaundice. It must be remembered however, that after *jaundice due to obstruction*

has existed for a time, the distended bile ducts may obliterate or obstruct the *vascular* supply to the hepatic lobules, and thus cause true suppression of bile formation. Obviously the symptoms of jaundice are not due to actual suppression, but to the causes which lead to such abolition of the biliary function. I append Dr Murchison's tabulated causes of jaundice.

A.—*Jaundice from Mechanical Obstruction of the Bile Duct.*

I. Obstruction by Foreign Bodies within the Duct.

1. Gall-stones and inspissated bile.
2. Hydatids and distomata.
3. Foreign bodies from the intestines.

II. Obstruction by Inflammatory Tumefaction of the Duodenum, or of the Lining Membrane of the Duct, with Exudation into its Interior.

III. Obstruction by Stricture or Obliteration of the duct.

1. Congenital deficiency of the duct.
2. Stricture from perihepatitis.
3. Closure of orifice of duct in consequence of an ulcer in the duodenum.
4. Stricture from cicatrisation of ulcers in the bile ducts.
5. Spasmodic stricture.

IV. Obstruction by Tumours closing the Orifice of the Duct, or growing in its Interior.

V. Obstruction by Pressure on the Duct from without, by—

1. Tumours projecting from the liver itself.
2. Enlarged glands in the fissure of the liver.
3. Tumour of the stomach.
4. Tumour of the pancreas.
5. Tumour of the kidney.
6. Postperitoneal or omental tumour.
7. An abdominal aneurism.
8. Accumulation of fæces in the bowels.
9. A pregnant uterus.
10. Ovarian and uterine tumours.

B.—*Jaundice independent of Mechanical Obstruction of the Bile Duct.*

I. Poisons in the Blood interfering with the Normal Metamorphosis of Bile.

1. The poisons of the various specific fevers.
   (*a*) Yellow fever. (*b*) Remittent and intermittent fevers. (*c*) Relapsing fever. (*d*) Typhus. (*e*) Enteric or pythogenic fever. (*f*) Scarlatina. (*g*) "Epidemic jaundice."
2. Animal Poisons.
   (*a*) Pyæmia. (*b*) Snake-poison.
3. Mineral Poisons.
   (*a*) Phosphorus. (*b*) Mercury. (*c*) Copper. (*d*) Antimony, etc.
4. Chloroform or ether.
5. Acute atrophy of the liver.

II. Impaired or Deranged Innervation interfering with the Normal Metamorphosis of Bile.

1. Severe mental emotions, fright, anxiety, etc.
2. Concussion of the brain.

III. Deficient Oxygenation of the Blood interfering with the Normal Metamorphosis of Bile.

IV. Excessive Secretion of Bile, more of which is absorbed than can undergo the Normal Metamorphosis.

1. Congestion of the liver.
   (*a*) Mechanical. (*b*) Active. (*c*) Passive.

V. Undue Absorption of Bile into the Blood from habitual or protracted Constipation.

It will be seen from the above table that Dr Murchison looked upon the liver as the sole organ in the production of bile. Physiologists, however, are not quite agreed on that point, but it would be out of place to discuss the various *pros* and *cons*, and unfortunately physiological facts are of a fleeting character.

Symptoms of Jaundice—

1. Icterus or tinting of the skin, conjunctivæ secretions, etc. The colour ranges considerably, from a lemon-yellow to a deep greenish-black (black jaundice).

2. Gastric disturbances, flatulence, nausea, and often complete anorexia.

3. Constipation often alternating with diarrhœa; the fæces are pale, intensely fœtid and pasty in character.

4. Slow pulse (has been observed in some cases to number only 20 per minute).

5. Extravasations of blood.

6. Cerebral symptoms. Marked depression of spirits, "patient sees things with a jaundiced eye," melancholia, and in the graver forms, an assumption of the typhoid state ending in death.

7. Itchiness of the skin.

The tests for bile acids and bile pigments will be found under "Examination of Urine."

Treatment.—As jaundice is merely symptomatic, the cause must be treated. In the milder forms a catarrh of the alimentary canal, or the presence of small biliary calculi will require attention. For detailed treatment see "Hepatic Diseases and Gall-Stones." Remember, however, the indications always are to—

1. Remove obstruction *if possible.*
2. *Promote functional activity by hepatic stimulants, after clearing old bile away with saline purges and calomel.*

It is nonsense to talk of the liver being *too* active, the liver cannot be too active. If there be an abnormal activity, *untoward* symptoms will not crop up until *incompetence* of the liver takes place.

ICTERUS NEONATORUM.

The form of jaundice which occurs amongst new born infants, may be of either a mild or a malignant type. The mild form appears on the second to the third day, and lasts from seven to fourteen days; beyond the pigmentation few symptoms are present.

Causes.—

1. The jaundice is probably due to the large destruction of red corpuscles, which takes place the first few days after birth (hæmatogenous jaundice).
2. Patency of the ductus venosus allowing the portal blood (which contains at this time bile pigment) to mix with the systemic circulation.
3. Diminished pressure in the portal circulation after severence of the placenta, the bile pressure may then exceed the blood pressure.

Malignant Form.—**Causes.**—

1. Congenital absence of the hepatic duct.
2. Congenital syphilitic hepatitis.
3. Phlebitis of the umbilical vein.

This form is almost invariably fatal.

ACUTE YELLOW ATROPHY OF THE LIVER.

(FATAL JAUNDICE).

A peculiar and rare disease probably due to septic organisms, and characterised by a rapid and often complete destruction of the hepatic cells throughout the gland.

Ætiology.—Women are more often attacked, and pregnancy is sometimes associated with the condition. Other causes put down

are abuse of mercury, fright, the poisons of malaria, and typhus. (Though there are many points of resemblance between acute atrophy, yellow fever, and pathological phosphorous poisoning, the conditions of each are not identical.)

Pathology.—The liver is much diminished in size, soft and flaccid. The capsule is wrinkled. On section, extensive fatty degeneration, destruction of hepatic cells, and empty bile ducts, are the marked features. Masses of golden pigment, red patches, and course granules are often present, *leucin and tyrosin can always be detected, and may crystallise spontaneously after death.*

Symptoms.—Usually there are precursory symptoms such as mental and bodily depression, constipation, and tenderness in the hepatic region. In the confirmed stage, intense jaundice sets in, with vomiting, etc., severe hæmorrhages, evacuations of pale fæces (or the fæces may be black from mixture of blood); pregnant women abort, and the typical typhoid state ushers in the end. Death usually takes place within seven days of the confirmed stage.

Diagnosis.—*The severity of the symptoms*, with the jaundiced aspect, diminished hepatic dulness, and *the presence of leucin and tyrosin* in the urine makes the diagnosis fairly easy.

We have already seen that leucin and tyrosin are probably the earliest antecedents of urea. Their appearance in the urine, and disappearance of urea in a condition where the liver is so largely affected, forms a strong link in the chain of evidence in favour of the liver being the chief seat of proteid metabolism, and urea the final product of such metabolism.

Treatment.—Probably useless. Large doses of quinine with the mineral acids, and diffusible stimulants may be tried.

ABSCESSES IN THE LIVER.

An abscess in the liver may be due to many causes, such as—

1. Traumatisms.

2. Suppuration of the bile ducts set up by gall-stones, parasites, etc.

3. Suppurating cysts, tubercular nodules, or syphilitic gummata.

But there are two forms which requires special mention—viz., the tropical or single abscess, and pyæmic or multiple abscesses.

TROPICAL ABSCESS.

Is probably due to the amœba coli flourishing in a suitable soil furnished by dysentery, excessive use of alcohol in hot climates, etc.

Pathology.—The abscess is usually single, but there may be two or more.

Situation.—Right lobe.

Walls.—Firm and thick, and have often three distinct layers—

1. *Inner.*—Gray in colour, and composed of necrosed tissue, amœbæ, and pus cells — no membrane internally.
2. *Middle.*—Brownish-red friable liver tissue.
3. *Outer.*—Hyperæmic liver tissue.

Contents.—Pus, often like anchovy sauce, and of a peculiar disgusting sourish odour. The abscess if left alone most often bursts *into the right lung.*

Symptoms.—The *earlier* symptoms are—

1. Pain, sense of weight and fulness, and tenderness in the right hypochondrium.
2. More or less jaundice.

Later.—We get rigors, elevation of temperature, "septic" in character; symptoms dependant on more or less peritonitis; the tongue becomes furred and dry, a sickly odour emanates from the breath; jaundice may become marked, and ascites develop. It must be remembered that the symptoms may be latent until those of rupture appear. *When it bursts into the lungs, the expectoration of anchovy paste-like sputum is characteristic.* If there be doubt about the diagnosis, puncture with a fine trocar.

Treatment.—Avoidance of alcohol; removal from tropical climate; treat on general principles. Rest, ipecac., warmth, and tonics. When pus has formed and can be fairly localised, aspirate.

PYÆMIC ABSCESSES.

Pyæmic abscesses of the liver are usually small and scattered throughout the liver substance.

Pathology.—The condition is essentially dependant on the presence of micrococci, and is most frequently a part of a general pyæmia. The micrococci may gain an entrance into the liver substance either—

1. *Through the hepatic artery* in cases of general pyæmia, especially after head injuries, (in which case the organisms must have first passed through the capillaries of the lung).
2. *Through the portal vein* in lesions of the portal area.
3. *Through the bile ducts* in ulceration from gall stones, etc.

In these cases the suppuration is in the course of the distribution of the portal vein (Dr F. Taylor).

The formation of pyæmic abscesses has already been described under "Pyæmia."

Symptoms.—Hectic fever, jaundice, with a tender, painful. enlarged liver, are the main symptoms. Often the symptoms are very obscure, and pain may be conspicuous by its absence, "Febrile jaundice" is a valuable diagnostic when present.

Treatment.—Almost hopeless. Can only hope to support patient until the septic processes have ceased.

CIRRHOSIS OF THE LIVER.

This is a condition characterised by a great increase of connective tissue at the expense of the proper glandular tissue.

Ætiology.—By far the most important factor in producing this disease is the excessive use of alcohol, especially spirits. Though essentially a disease of adult life, children who are addicted to intemperance frequently afford typical examples of hepatic cirrhosis. Other factors are supposed to be gout and syphilis, but as these two conditions are often associated with intemperance, possibly the alcohol, even in these conditions, is the most potent agent. Congenital syphilis may produce cirrhosis, but, I certainly have seen many hundreds of cases of congenital syphilis without serious hepatic troubles. The other factors enumerated as causes of cirrhosis are malaria, tuberculosis, and excessive use of hot condiments.

Morbid Anatomy.—During the hypertrophic stage, the liver is often enormous in size, extending even *below* the umbilicus; towards the latter end of the atrophic stage the liver may only

weigh two pounds, and be quite inaccessible to the touch. The more atrophied the liver, the more rough, nodular, or hobnailed is it likely to be. Conversely, the larger the liver the greater probability of it being smooth or but slightly roughened. There is a great tendency at present to regard all cases of cirrhosis as the result of a simple chronic inflammation, and therefore all have—(1) a stage of hypertrophy, and (2) a stage of atrophy. This seems to me to be by far the most probably theory, and we have no less an authority than Dr Frederick Taylor of Guy's Hospital, for this statement. If we grant this, then we can easily understand why few symptoms are present in the earlier stages, and why they become so pronounced (almost malignant) in character during the atrophic stage. Clinicians and pathologists, however, do not agree.

I append Dr Woodhead's table of the "biliary" and the ordinary cirrhosis :—

Common Cirrhosis.	Biliary Cirrhosis.
1. The bile ducts are not involved in the growth of connective tissue at first; consequently, jaundice is a late symptom.	1. The bile ducts are involved early, and jaundice is a severe symptom. Apparently there is a new formation of bile ducts.
2. The new growth involving the portal circulation is likely to cause ascites, hæmorrhoids, hæmatemeses, etc.	2. The portal veins are not involved, and symptoms dependant on portal obstruction are rare.
3. In the earlier stages the increased growth of young connective tissue in the portal spaces cause considerable increase in the size of the organ; but, in the later stages, this tissue contracts, and in contracting cause, a considerable decrease in the size of the liver.	3. The large amount of new tissue diffused throughout the organ, causes a great enlargement of the liver.
4. The liver rough and hobnailed on its surface; and the capsule much thickened.	4. Surface is smooth (like Morocco leather); the capsule is not thickened.

5. The masses of liver cells vary in size, some consisting of several lobules, whilst others are smaller than a lobule. Each of these masses form a *distinct area* having a rounded outline surrounded by a fibrous zone, and from the fibrous mass the mass of liver cells can be easily turned out.	5. The masses of liver cells consist of atrophied lobules, and the cut surface has a more or less uniform, and finely granulated appearance.
6. On microscopic examination it is seen that the process is going on *chiefly* at the periphery of the lobules, but, that groups of lobules are affected.	6. The single lobules above mentioned are surrounded by bands of fibrous tissue, which bands, however, are not confined to the periphery, but invade the substance of the lobules.

However the main thing to remember is *not* the varieties, but to look upon cirrhosis pathologically as a condition characterised by—

1. Increased growth of fibrous tissue in Glisson's capsule, and in the prolongations of it running into the liver substance.
2. Obstruction to the portal circulation.
3. Increased blood pressure in the hepatic arteries.
4. Obliteration of hepatic cells. } *later.*
5. Obstruction of biliary ducts. }

Symptoms.—At first there may be little disturbance beyond chronic gastric catarrh symptoms—viz., morning vomiting, anorexia, and acid eructations, but soon a severe hæmatemeses coupled with a distinct sense of weight in the hepatic region reveals the true nature of the disease, and on examination, the liver is found to be much enlarged. As time wears on the more characteristic features are—

1. Ascites.
2. Emaciation.
3. Jaundice (often very late).

And now on examination the liver is found atrophied.

The ascites as a rule is particularly well marked. The surface of the abdomen is covered by large distended veins, showing an attempt at collateral circulation to carry on the now obstructed portal circulation. This condition is highly important from a prognostical point of view. The more common anastamoses are between—

1. The gastric and œsophageal veins.
2. Middle hæmorrhoidal of the inferior mesenteric and inferior hæmorrhoidal of the internal iliac vein.
3. Coronary veins of stomach and phrenic veins.
4. Accessory portal vein of Sappey—*i.e.*, a vein running alongside the round ligament of the liver, communicating the portal vein with branches of the epigastric veins near the navel. This often produces a large bunch of varicose veins, the so-called caput medusæ.

It will be thus seen that the later symptoms are dependant on a grave interference with the hepatic metabolism plus general venous congestion. The toxæmia and exhaustion, consequent on serious obstruction, may bring about a fatal issue.

The clinical symptoms of the so called "biliary" cirrhosis already referred to are (as described by the French School)—

Acute intense jaundice, but *no* ascites.

Elevation of temperature.

Delirium and the rapid assumption of the *typhoid* state.

Death often occurs within ten days or three weeks.

Prognosis.—Bad as regards cure; but if the collateral circulation be well established, the atrophy not marked, and the patient made to live temperately, bad symptoms may not arise for a considerable period. There is very grave doubt as to the possibility of far advanced cirrhosis being cured.

Treatment.—It must be (1) general, and (2) symptomatic.

General.—Absolute temperance in eating and drinking must be observed with abstinence from hot condiments, etc.; the portal circulation should be depleted by the administration of a saturated solution of Epsom salts. Exercise, massage to the liver, and careful avoidance of chills, are the main points to remember. Ammon. chlor., arsenic, strychnine, etc., may be given.

Symptomatic. — *Ascites*, the fluid should be evacuated quickly by tapping, or by the slow method by means of Southey's tubes. Hydrogogue cathartics are sometimes of use. The hæmatemeses should not be treated too energetically as it often acts as a safety valve, *i.e.*, preventing internal hæmorrhage. If it be excessive, ice, recumbent position, ergot, acid sulph. dil., full dose of morphia, hydrastis, hazeline, etc., should be resorted to.

The Emaciation, calls for tonics, and the most nutritious diet compatible with the imperfect assimilative powers. Some authorities advise skim-milk diet.

WAXY LIVER.

The liver is often affected with lardaceous disease, and as elsewhere, the degeneration begins in the middle coats of the blood-vessels. The lobules are invaded from without inwards, the hepatic cells become destroyed, and the glandular structure converted into a firm, dense material. The liver is enormously enlarged, and smooth. On section, the substance is glistening, firm, and resembles yellow wax, the cut surfaces showing only faint traces of lobules. It must be remembered that waxy disease may co-exist with fatty liver, syphilitic disease, etc.

Causes.—Prolonged suppuration, as in strumous ulcerations, caries, and necrosis; as a result of malignant fevers, etc.

Symptoms.—Sense of weight in the hepatic region, anorexia and other dyspeptic symptoms; diarrhœa, the stools being pale and often fatty. Later, there may be jaundice, ascites, and albuminuria. *Pain is rare.* The disease is usually fatal.

Treatment.—Remove cause as far as possible. Iodide of potassium is often of great service, especially when the disease is due to syphilis. The general treatment is the same as that of tuberculosis.

FATTY LIVER.

By fatty liver is meant, a great increase in the quantity of fatty globules, naturally contained in the hepatic cells; so that on examination the cells are found loaded with oil globules, often obscuring the nuclei. The change takes place first at the periphery, but later the whole of the lobule is invaded. The liver is usually much enlarged with a smoothish surface.

Causes.—General obesity; emaciating diseases, such as cancer and phthisis; venous congestion may cause both nutmeg and fatty liver. Care should be taken not to confound fatty infiltration with fatty degeneration. In the latter condition the hepatic cells undergo a complete metamorphosis. Physiological chemistry leads us to anticipate fatty *infiltration*, in those cases where the oxidising power is insufficient to dispose of the ordinary *fat forming* elements of the daily food. Fatty degeneration is also due to deficient oxidation, but the tissues themselves form the fat, and experience has shown it is always associated with either mineral poisons or bacterial action. I do not think it can be proved to be ever due to imperfect oxidation simply.

Symptoms.—Often are very obscure, but the symptoms of chronic gastric catarrh, constipation alternating with diarrhœa, pasty complexion, together with the physical signs of an enlarged liver, *not* accompanied by either pain or jaundice, renders the diagnosis fairly easy.

Treatment.—Regulate the diet, avoid excess of proteids or carbohydrates particularly. Prescribe alkaline aperients, chloride of ammonia, calomel and podophyllin, massage and exercise.

CANCER OF THE LIVER.

Cancer of the liver may be primary, but is more often secondary to cancer elsewhere.

Ætiology.—Men are more frequently attacked than women: from forty to sixty years seems the most favourable period. The predisposing causes are similar to those of cancer elsewhere, viz.,—heredity, injuries, etc.

Pathology.—All types of cancer have been found in the liver, but the primary forms are nearly always epitheliomatous.

Primary Cancer.—Two distinct types are distinguished—

1. The massive liver, greyish white in colour, occupies a large portion of the liver and is abruptly defined from the liver tissue.
2. Nodular cancer, in which the nodules vary much in size, and are irregularly scattered *throughout* the organ.

Secondary Cancer.—The organ becomes *enormously* enlarged, and the nodules can easily be felt through the emaciated abdominal walls; indeed, they may be near enough to the surface to be visible (the so-called "Farre's tubercles"). On section we find—

1. Extensive areas of fibrosis.
2. Extensive areas of fatty degeneration.
3. Extravasations of blood.
4. Dense greyish hyaline masses.

Symptoms of Cancer of the Liver. — Daily increasing emaciation, *hepatic pain*, malignant cachexia, jaundice, *sometimes* ascites, attacks of local peritonitis, together with the characteristic physical signs of the rough, uneven, enlarged liver, are the principal features. It should, however, be remembered that cancer may be added to a cirrhosis of the liver. When the growth is of a melanotic sarcoma type, the pigmentation plus other symptoms, points to the true nature of the disease.

Diagnosis between cancer and cirrhosis is often difficult when there is a strong history of alcoholism. The following table may help—

	CIRRHOSIS.	CANCER.
Progress	1. Often slow.	1. Always rapid.
Liver	2. Enlarged at first, then smaller, and more nodular as atrophy becomes more marked.	2. Is large, and the nodular character developed from the first.
Pain	3. Not marked.	3. Well marked.
Ascites	4. Usually present.	4. Often absent.
Jaundice	5. Not till late.	5. Often a marked feature.

HEPATIC TUMOURS.

The more common tumours and growths, *otherwise than malignant*, are—

1. Hydatid tumours.
2. Cystic tumours.
3. Cavernous tumours (angioma).
4. Tuberculosis.

Hydatid Tumours occur more frequently in the liver than in any other organ (see "Tænia.")

Symptoms.—A hydatid tumour of the liver grows slowly, and usually in one particular direction. It is painless (until it

suppurates), and may cause no symptoms until it becomes extremely large or bursts.

Physical Examination.—Tumour is globular, tense, elastic, and with care fluctuation is detected. On palpation a peculiar vibratory thrill may be felt.

Prognosis.—The cyst may undergo spontaneous cure without rupture. Death of the parasite causes the cyst to contract. Rupture may take place into the lung or through the abdominal wall, into the intestines, or into the hepatic duct.

Treatment.—As soon as the cyst is discovered employ surgical means, such as—

1. Aspiration, and subsequent injection of iodine.
2. Electrolysis.
3. Careful incision, and free evacuation of cyst contents.

Cystic Tumours are due to inflammation of the hepatic ducts, and consist of a cheese-like substance showing under the microscope, granular debris, cholesterine crystals, oil globules, etc.

Cavernous Tumours, occasionally in children attain a great size, but are more common in aged people. They are found on the upper surface of the liver; on cutting into them they present a similar appearance to that of the corpora cavernosa of the penis, hence the name "erectile tumour."

Tuberculosis.—Typical tubercular deposits occur in the liver, secondarily to tubercle elsewhere, especially the abdominal viscera. Patient usually dies before the disease is far advanced.

As regards the treatment of these tumours, it must be entirely symptomatic, combined with careful hygienic measures in all cases.

DISEASES OF THE GALL BLADDER.

Inflammation of the gall bladder is most frequently due to extension of inflammation from neighbouring parts, but it may be caused by direct irritation of the cystic mucous membrane through the passage or impaction of gall-stones, parasites, etc.

Dropsy.—When the *cystic* duct is blocked, the mucous membrane of the gall bladder undergoes a peculiar degeneration, resulting in a secretion of watery fluid, which may distend the gall bladder enormously. *Often the tumour is so freely movable as to cause it to be mistaken for a movable kidney.*

Gall-Stones.—Biliary calculi are most common in females between the age of thirty and forty-five.

Composition—

Single gall-stones are almost entirely composed of cholesterine (a monatomic alcohol). The nucleus is usually a bit of dried mucus. The stone is semi-translucent, glistening, with a somewhat *granular surface*, and is very light.

Multiple gall-stones are much more common; a few or a hundred stones may be present. They differ from the single stone by—

1. Being stratified: the strata are of different shades of colour, some being very deeply coloured.
2. Presenting facets on their surface from pressure.

Effects *of Gall-Stones* are—

1. To cause more or less inflammation of the mucous membrane.
2. Impaction of the ducts, consequently giving rise to jaundice etc.

3. They may cause serious ulceration.

4. During their passage along the ducts they cause the intensely painful "biliary colic," and often

5. Subsequent appendicitis, obstruction of bowels, etc.

Symptoms of Biliary Colic.—

1. Excruciating pain in the hepatic region, and radiating all over the *thorax* to the shoulders, and with a sense of general thoracic constriction.

2. Vomiting, which often gives relief (probably by the contractions of the diaphragm so induced, aiding peristalsis).

3. *Subsequently* to the attack, jaundice is more or less pronounced.

4. A local rise of temperature (?).

It must be remembered that (as in renal calculus) the stone after obstructing for a time, may slip back again into the cystic duct or gall bladder, giving a sequence of events which may be several times repeated. Moreover, gall-stones may be the cause of hypertrophic cirrhosis, or the exciting cause of cancer. (Dr Saunby.)

Many observers have shown conclusively that the presence of gall-stones may give rise to a "bastard ague."

Treatment of *Biliary Colic.*—

1. Hypodermic injection of morphia.
2. Chloroform inhalations until morphia acts.
3. Hot fomentations over liver.
4. Nitrite of amyl.

Subsequently: purgatives, careful dieting, regular exercise, and avoidance of any constriction of the hepatic circulation by tight corsets, etc.

Persistent Biliary Obstruction. — Demands surgical interference, but care must be taken to diagnose the cause of

obstruction first. Harley advises a trial of massage and olive oil internally. I removed eighty gall-stones by this method in one case.

Diagnosis of Gall Bladder Affections.—A *distended* gall bladder forms a *smooth ovoidal* tumour below the ninth right costal cartilage. Take into consideration the history, jaundice, character of pain, etc.

DISEASES OF THE KIDNEY.

ALBUMINURIA.

By albuminuria is meant, "the presence of albumin in the urine." As albumin is one of the essential constituents of the blood, an escape of it in the urine must necessarily be looked upon with suspicion, if not with anxiety. Formerly, all cases of albuminuria were regarded as varieties of Bright's disease, but it has been conclusively shown, that albuminuria is often present in (as far as we can make out) healthy individuals. However, "*a persistent albuminuria, especially if the albumin tends to increase, must always be looked upon as a grave and significant condition.*" In other words, just as palpitation of the heart may exist without cardiac disease, it may on the other hand be a prominent symptom of a grave organic lesion; so with albuminuria, it may or may not be a serious omen.

Theories of albuminuria are at present speculative, possibly the best is number 1.—

1. That the cells lining the glomeruli exert a selective influence in excretion, inasmuch as they allow the water and certain soluble salts to pass, but prevent albumin from so doing.

2. That albumin *normally* escapes through the glomeruli into the tubules, but is rapidly taken up again by the cells

of the tubules, and *re-absorbed into the blood.* This is doubtful, but on the other hand, the cells of the convoluted tubules certainly have a decided *selective function*, as shown in the excretion of urea, etc.

The first theory would demand for the production of albuminuria) a diseased condition of, or at least some alteration in the epithelium of the glomeruli.

The second theory demands "increased blood pressure," in order to explain the escape. A great increase of the renal blood pressure injures the cells, and causes the urine to rush past the cells so quickly that re-absorption is prevented. Most probably both conditions play their parts, but neither one nor both are sufficient to explain all cases.

Experimentally, we may produce albuminuria by—

1. Pressure upon (not closure of) the renal veins; the pressure in the glomeruli is increased thereby.

2. Closure of the renal artery, and subsequent re-establishment of the circulation; this interferes with the nutrition of the renal cells.

3. Ligature of the aorta *below one* kidney, and extirpation of the other.

4. Ligature of the aorta above the renal arteries.

5. Compression of the trachea; this leads to asphyxia, and consequent rise of blood pressure. (HALLIBURTON.)

Clinically, we get albuminuria—

1. Due to—

Morbid conditions of the kidney, such as acute and chronic inflammation, waxy degeneration, renal calculi, tumours, etc.

Diseases of the urinary apparatus below the kidney.

Hæmic changes—anæmia, leucocythemia, etc.

Certain fevers, especially scarlet fever and diphtheria.

Pregnancy.

Certain poisons — arsenic, phosphorus, excessive use of morphia, etc.

Venous congestion, consequent on hepatic, pulmonary, or cardiac disease, etc.

2. After certain diets, especially in those who pass much oxalates in their urine (oxaluria).

3. In a *remittent or cyclic form*, occurs in apparently healthy people; albumin is in such cases only present in the urine at certain periods of the day.

4. In the so-called "paroxysmal" form, associated with a more or less jaundiced apppearance, and anæmia. Possibly is due to functional disturbance of the liver, whereby the breaking down of corpuscles is much increased.

5. *After severe exercise.*

Treatment of Albuminuria.—No condition requires more careful treatment. The first thing to do is to settle if possible *whether it is functional, or the result of structural disease of the kidney or urinary passages.* This can only be done by careful consideration, and *frequent* examinations of the urine. Do not forget that albumin is *temporarily* absent in many grave cases of renal disease. The following are the chief indications of treatment.—

1. Remove if possible any obvious cause.
2. Prescribe a non-nitrogenous diet as far as possible.
3. Avoidance of all substances (food or drugs) likely to increase unduly the blood pressure, or cause irritation of the renal tubules.
4. Give ergot, jaborandi, gallic acid, nitrite of amyl. Further treatment will be discussed under "Nephritis." For tests for albumin see examination of the urine.

BLOOD IN THE URINE.

Blood in the Urine is another very grave symptom. Two forms are described—

1. Hæmaturia, or bloody urine proper.

2. Hæmoglobinuria, a condition marked by the presence of blood-pigment in the urine, but *few or no* blood corpuscles. *The blood pigment is generally methæmoglobin* (chocolate colour).

Causes of Hæmaturia.—

Diseases of the kidneys and urinary apparatus below them.
Extensive bruises (absorption of blood pigment).
Scurvy (especially in children).
Malignant forms of fevers, especially the malarial type.
Traumatisms.
No discoverable cause (Gull's "epistaxis of the kidney").
Drugs which directly irritate the kidney, turpentine, cantharides, carbolic acid, etc.
Parasites, especially the bilharzia hæmatobia.

Diagnosis.—

1. *By the colour of the urine*, which ranges from a slight smoky tint to deep red or even porter colour.

2. *Microscope.*—Detection of blood discs (in fresh urines).

3. *Spectroscope.*—The characteristic bands of hæmoglobin (generally the reduced Hb).

4. Ozonic æther and guaicum gives a blue tint. (This is a very fallacious test.)

If the blood comes from above the ureters, it is usually freely mixed with the urine.

If from the ureters, blood may be clotted in the form of moulds.

If from the bladder, is either mixed, or appears after urine has been passed.

If from the urethra.—Before or during the *first* part of micturition.

There are no definite means of ascertaining the site of the hæmorrhage by mere examination of the urine alone.

Treatment.—Rest in the recumbent posture. Full doses of opium. Ergot.—Lead Acetate.—Hazeline.—Hydrastis Canad.

Parasitic Variety.—Injections into the bladder of iodide of potassium (5 grains to ℥i.) Santonine, or male fern, internally.

Causes of Hæmoglobinuria.—

1. *Toxic Form.*—Due to the action of certain poisons, such as *nitrites*, arsenuretted hydrogen, carbon monoxide, bile, etc.

2. *Paroxysmal Form.*—This condition is characterised by the occasional passage of bloody urine, the pigments *only* being present. The causes are not known, but there seems to be a great causal relation between this condition and disturbances of the vasa-motor system; so much so, that it has been termed "Raynaud's" disease of the kidneys. The most commonly associated conditions are—

(1) Vasa-motor disturbances.
(2) Increased hæmolytic action of the liver and spleen.
(3) Extensive superficial burns.

(I saw three such cases in a colliery practice).

In newly born infants hæmoglobinuria may accompany the usual destruction of red corpuscles.

Symptoms.—The attack may come on after the morning bath, mental exhaustion, or exposure to cold. There is usually pain in the lumbar region during the attack. Vomiting, anorexia and jaundice are often present.

Treatment.—Fresh air and tonics. During the attack keep the patient *warm* and give hot drinks.

URÆMIA.

Is the name given to the symptoms that arise from retention of urea (or what is much more probable the *antecedents of urea*). It is usually associated with disease of the kidneys and supression of, or diminished quantity of urine. The two theories before the profession at present are—

1. Inability of the kidney to eliminate the urea, which being retained in the blood, splits up into carbonate of ammonia *which is considered the toxic element.*

2. The liver being extensively handicapped by the deficient assistance of the kidneys, breaks down proteid waste imperfectly, and, consequently, the *intermediate products similar to alkaloids accumulate* and exert their poisonous action in different ways. Bouchard has shown conclusively that these poisons have markedly different qualities. This is probably the correct theory.

Against the first theory are four facts, viz.—

(1) Urea, if injected into the blood will not cause uræmic symptoms.

(2) Uræmic symptoms are not the same as those of poisoning by carbonate of ammonia.

(3) No two cases of uræmia are exactly alike.

(4) *Urea* which is present only in *minute* quantities in normal bile and fæces is much increased in these secretions during uræmia.

Symptoms of Uræmia are diverse and many. Taking a typical case we may expect—

Vomiting.

Headache of a dull character, especially in the occipital region.

Peculiar and characteristic dyspnœa, "Cheyne-Stokes' breathing" persisting for weeks without *coma.*

Muscular twitchings, deepening into

Convulsions.

Contraction of pupils (often uneven).

Suppression of urine.

Coma and death.

The odour of the breath is said to be ammoniacal, or sweetish like new mown hay; much oftener the breath is offensive from severe stomatitis.

Diagnosis.—The utmost *care* should be taken not to mistake the disease for alcoholism, apoplexy, opium poisoning, etc., but such diagnosis is often extremely difficult. Examination of the urine, of the eyes for albuminuric retinitis, of the pulse for evidence of arterio-sclerosis, are the chief points to be remembered. Lastly, remember that hæmorrhage into the brain during cirrhotic Bright's disease is common.

ACUTE BRIGHT'S DISEASE.

Acute inflammation of the kidneys is a disease characterised by febrile disturbances, grave changes in the urine, and dropsy.

Causes.—

1. Exposure to cold, especially when the body is overheated.

2. Acute specific fevers and septic states, especially scarlet fever and ulcerative endocarditis. (Albuminuria without the other symptoms of acute nephritis is common in diphtheria.)

3. Certain irritating drugs, *e.g.*—cantharides, copaiba, turpentine, etc.

4. Extensive burns involving the abdomen.

5. Parasites, etc.

Pathology.—Two types are usually distinguished,—*i.e.*, the ordinary or catarrhal form, and the so-called infective or glomerulo-nephritis.

1. *Catarrhal Nephritis.*—The kidneys are large, much injected, and the cortex is seen to be disproportionately enlarged. On section, the cut surfaces show up the Malpighian bodies as deep red points; here and there are patches of extravasated blood. The tubules show marked changes, especially the convoluted portions, the epithelium undergoes cloudy swelling, followed by proliferation and detachment of the cells, which form casts or masses of granular fatty debris, often to the extent of blocking up the tubules. It must not be forgotten, however, that though the tubules are the parts principally involved, the interstitial portion also takes part in the inflammatory process.

2. *Glomerulo-Nephritis.*—In this form the glomeruli principally suffer, and is usually the result of infective fevers. After a preliminary engorgement of the blood-vessels, extensive leucocyte emigration takes place, filling the glomerular capsule, the capillary vessels often burst, and the blood flows into the tubules. The epithelium of Bowman's capsules often proliferate to such an extent that the tufts become obliterated by the pressure, and in time the latter may be actually changed into fibrous nodules. Of course the tubules and interstitial tissue are also involved, though not to the same extent.

Symptoms are at first, chilliness, pain in the back, vomiting and pain over the brows; the temperature is raised (but may never be high), the characteristic œdema rapidly appears, at first in the conjunctivæ, eyelids, and cheeks, but later it becomes general. The pulse is usually quick and of *high tension* from the beginning. The urine is voided frequently, but scant in quantity, and indeed may be suppressed for a time. It is dark from the presence of blood, exhibits the well-known smoky hue, and generally gives a copious deposit of urates, blood discs, granular debris, fatty and hyaline casts, and *a large amount of albumin.* Though of such high specific gravity, the total amount of urea

passed daily is much decreased. As the disease advances, the anasarca becomes more marked; there may be dangerous dropsy of the serous sacs, œdema of the base of the lung or of the glottis. The bowels become constipated, the tongue dry, and there is great thirst. Hypertrophy of the heart is often marked. If improvement does not quickly take place, grave dangers arise from the accumulation of excrementitious material in the blood; anæmia becomes pronounced, and intense headache, convulsions, and coma often usher in a fatal uræmia. Most cases however, with care, recover; others pass into the subacute stage or "large white kidney."

The more important complications to remember are—

1. Excessive delatation of the heart in debilitated patients.
2. Dropsy of the lungs, pericardium, and glottis.
3. Albuminuric retinitis and optic neuritis.
4. Uræmia.

Treatment.—Absolute rest in bed; patient to be laid between blankets; hot poultices (not blisters), or dry cupping over the loins, a free saline purge, diluents in an effervescing form, and a *non-nitrogenous* diet, forms the routine treatment. Remember that urates are highly irritating to an inflamed kidney, *hence the necessity of diminishing the quantity of proteids taken*, and the indication for the use of these drugs which render the urates more soluble. Acetate of potash and nitrous æther with hyoscyamus, form a splendid mixture, as we have at once *a solvent of urates, a dilator of the peripheral arterioles, and a sedative to the urinary tract.* Complications must be treated as they appear.

Excessive Dropsy.—Hot air baths, pilocarpine, compound jalap powder, elaterium, Southey's tubes, tapping, etc.

Uræmia.—Same as for excessive dropsy *plus* chloroform during the convulsions, and wet cupping, or even venesection.

Severe Dyspnœa.—Nitrite of Amyl is of great service.

CHRONIC BRIGHT'S DISEASE.

"By Bright's disease we mean a non-suppurative inflammation of the kidneys. To understand the classifications that have been made, it is necessary to recognise the following structures in the kidney, which are liable to diseases, *more or less independent of one another.* They are—

1. The *Tubules* with their epithelium, forming the parenchyma of the kidney.

2. The *Interstitial tissue*, very small in quantity in the healthy organ, but liable to considerable increase by inflammatory processes.

3. The *Blood-vessels* and the glomeruli; consisting of the vascular tufts, the capsule, and the epithelial cells covering the former and lining the latter." (Dr Taylor's "Practice of Medicine.")

Obviously in a nephritis the inflammatory changes may be most marked in the *tubules*, or in the *interstitial tissue*, or in the *blood-vessels;* but it must be equally clear that neither *one* of these special tissues can be affected to any great extent *without implicating the other components of the kidney structure.* In other words, though the various types of Bright's disease are based upon the variety of tissue principally involved, still, it must be distinctly understood that *the kidney structure as a whole shares in the degenerative changes.* This applies with particular emphasis in acute cases.

The best known forms of chronic Bright's disease are—

1. The chronic tubal nephritis (large white kidney).
2. Chronic interstitial nephritis (small red or gouty kidney).
3. Lardaceous or waxy disease of the kidney (pale waxy kidney).

Beginners in medicine frequently complain that they get hopelessly muddled on trying to distinguish or to remember these varieties; we must therefore, before describing them in detail, consider some *general* facts in reference to renal disease. Now suppose we get defective renal secretion, what must happen?

1. Retention of waste and poisonous products in the blood.
2. Damage to the vascular walls by those poisonous products.
3. Anæmia; and consequently all the troubles produced through imperfect nutrition of *all the tissues and organs in the body.*

We must expect and therefore describe changes under six headings—

1. *Changes in the kidney structure*—

   Interstitial tissue.
   Tubules and their epithelium.
   Blood-vessels.

2. *Changes in the urine*—as regards quantity and quality—*i.e.*, the presence of pathological constituents—

   Albumin.
   Blood.
   Casts, which may be granular, fatty, or hyaline.

3. *Changes in the circulatory system*—

   The heart is often enormously hypertrophied and dilated.

   The arteries are sclerosed and thickened.

   The pulse is characteristic—it is peculiarly hard, and of high tension, the dichrotic wave being almost obliterated.

[The cause of the hypertrophy, and the nature of the arterosclerosis are still unsettled. As the high tension occurs quite early in even *acute* Bright's disease, increased peripheral tension

set up by irritation of the vascular walls is the probable cause, and this would account for subsequent hypertrophy of the heart. Cohnheim considered that the fact of more force being required to drive the blood through a *diseased* kidney, would explain the cardiac hypertrophy. The question, however, is still *sub judice.*]

4. *Ocular changes* consisting of—

Œdema of the retina, causing opacity and swelling.
White glistening patches due to fatty degeneration of Müller's fibres, etc.
Flame-shaped extravasations of blood.
More or less atrophy of the optic disc.

5. *Dropsy* first appears as œdema of the conjunctivæ or lower eyelids: later, general anasarca, and dropsy of the serous cavities—*i.e.*, ascites, hydro-thorax, or immense dropsy of the scrotum, may all occur.

The dropsy is at first most apparent in the morning. Many factors contribute to the causation of renal dropsy—

(1) The anæmic and hydræmic state of the blood.
(2) Degeneration of the vascular walls.
(3) Deficient secretion of urine.
(4) Imperfect cardiac action and pulmonary engorgement (late).

6. *Tendency to Uræmia.*—Uræmia has already been discussed fully.

There is no doubt that Professor Grainger Stewart struck the keynote to the proper understanding of the various clinical phenomena, when he pointed out "that chronic inflammation of the kidney differed in no way from that of other organs; that, given a slowly progressing inflammatory action, we should expect at first a stage of enlargement, and finally a state of atrophy."

Chronic inflammation may or *may not* be preceded by an acute attack; and lastly, when the condition begins as an *acute* nephritis, before it can become chronic, *there must be an intermediate or subacute stage.*

I would strongly urge the junior student to digest the foregoing remarks, and especially remember the relations between the liver and kidneys. If he does this, there can be no difficulty in understanding a most important section of practical medicine.

The "large white kidney" is more frequently a sequel of an acute attack; the "red gouty," on the other hand, is more often due to overwork, eliminating coarse products, or poisons. I would advise the reader to get up thoroughly the symptoms of a typical *acute* nephritis—and then a typical case of *chronic* interstitial nephritis. He will easily understand then that the "subacute" form will at first approximate to the acute form (scanty and albuminous urine), *and later*, to the chronic form (urine more copious and less albuminous).

The onset of chronic Bright's disease when not preceded by acute nephritis, is most insidious. Often there is nothing to attract the patient's attention to such a serious condition, beyond langour, a desire to micturate more frequently, diffuse headache, and slight stiffness of the eyes in the morning. When the disease is well marked, and failure of cardiac hypertrophy begins, the symptoms become very pronounced, such as marked pallor, breathlessness, attacks of uræmic asthma, dimness of vision, and dropsy of serous cavities. It is very essential that the condition should be early diagnosed, and careful examination of the pulse, retina, and urine, ought to go far to make an accurate as well as an early diagnosis. A diagnostic table, and the foregoing remarks, should enable the beginner to have a clear idea of the elementary facts of Bright's disease. A more exhaustive account cannot be given here, but after the final examination is passed, no practitioner should lose time before perusing one of the many valuable monographs on the subject.

Treatment of chronic Bright's disease must be considered under two heads.—

1. General treatment.
2. Symptomatic treatment.

General Treatment.—

Our indications under the first heading are to put the patient under the most favourable hygienic conditions. Exercise in the open air, and woollen or flannel underclothing are imperative. Diet is no less important; therefore food which contains the *most* nutritive properties with the *least* nitrogenous compounds must be prescribed. No rigid diet can be laid down for all cases, each must be treated on its own merits, but in all cases, beer, heavy wines, and excessive consumption of sugar should be condemned.

Medicinal Treatment.—Keep the bowels open by the use of saline purges. Improve the vascular tone by the administration of strychnine and iron.

Though it is generally held that mercurials are badly borne in renal affections, there can be no doubt as to the efficacy of small doses of pil. hydrarg., or calomel, given at night-time, and followed by a tumblerful of Friedrichshall water in the morning. Hepatic depletion by brisk cathartics is often more efficacious than any other treatment in allaying urgent symptoms.

Symptomatic Treatment.—

Headache may be relieved by a mixture containing pot. iodid., digitalis, and caffeine. When the tension is very high, a good purge and a few whiffs of nitrite of amyl often act like a charm.

Dropsy.—When excessive, may demand removal of the fluid by Southey's tubes. A saturated solution of mag. sulph., administered in the morning often removes enormous quantities of water.

Albuminuria.—If the escape of albumin be excessive, ergot or gallic acid may be tried.

	CHRONIC TUBAL NEPHRITIS, OR LARGE WHITE KIDNEY.	CHRONIC INTERSTITIAL, OR GOUTY CIRRHOTIC OR RED GRANULAR KIDNEY.	AMYLOID OR WAXY KIDNEY (OR LARDACEOUS DISEASE).
Causes	Secondary to acute; exposure; irritating drugs; pregnancy, etc.	As a sequence of large white kidney; most often chronic from beginning: gout; lead; alcoholism; syphilis (?), etc.	Secondary to suppurative disease or syphilis.
Age	Any age if following acute; if not, middle age.	Any age — usually after 40 years of age.	Any age.
Kidney	At first enlarged, smooth; capsule thin, but easily separated. Section shows tissues to be whitish-yellow, fatty, and granular. Slight increase of the interstitial tissue; tubules are most affected. Later, approximates to red granular condition.	Is red small and granular; *capsule adherent*; cortex much reduced. Tubules atrophied — *some cystic*; great increase of fibrous tissue; arteries are sclerosed, varicose, and some obliterated. Epithelium much atrophied.	Usually large, *cortex increased*, capsule strips readily, glomeruli stand out as glistening points; blood-vessels show waxy disease, epithelium and base membrane also involved.
Urine	*Early Stages*— Scant, high coloured. Albumin plentiful. Blood often present. Tube casts abundant. Fatty, Granular. Hyaline. *Urea* much diminished.	Gradually becomes copious. Low sp. gr.—patient has to rise frequently in the night. All diminished—*albumin and blood most frequently absent.* Tube casts are more hyaline and less fatty when present. Urea is not diminished, considering the quantity of urine, until the later stages.	A *copious*, highly albuminous urine is characteristic. Casts vary immensely, often very few. Waxy blood and pus casts may be present in the later stages.
Dropsy	Early; well marked in face which is puffy and pasty. Anasarca is often extreme.	Very slight until cardiac failure occurs. Ascites from the hepatic condition may occur. Serous cavities often affected.	Rare, except perhaps œdema of ankles.
Blood and Retinal Changes	Fairly well marked.	Highly marked; "cow's heart;" aneurismal swelling of cerebral vessels which may rupture. Pulse is extremely hard.	Arteries more softened, waxy changes well marked.
Course and Duration.	Depends on cause. May end quickly through implication of serous cavities; becoming water-logged; or uræmia; may, however, pass into the red granular kidney.	Very protracted, but patient is markedly dyspeptic, and attacks of uræmic asthma are frequent. Œdema of glottis; and apoplexy or uræmia terminates the disease.	Depends on source of suppuration. Death most frequently due to primary cause. Exhaustion or critical diarrhœas.

PYELITIS.

By pyelitis is meant an inflammation limited to the pelvis of the kidney. If the kidney substance be also inflamed then it is termed pyelonephritis; and if the inflammation terminates in pus formation, the resulting collection of pus is called a "pyonephrosis." Now, it is most improbable, that the pelvis of the kidney ever becomes inflamed without involving the kidney substance; still more improbable, that such a condition could be diagnosed during life, so that clincally at all events, by pyelitis is meant a *suppurative* inflammation of the pelvis of the kidney, such a condition being usually associated with a certain amount of nephritis and pyonephrosis.

Causes.—Anything which causes severe irritation of the lining of the pelvis of the kidney, such as—

1. Calculi, blood-clots, parasites, tumours.

2. Morbid states of the blood, such as malignant fevers, diabetes, Bright's disease, etc.

3. Certain drugs—cantharides, etc.

4. Extension of inflammation from below (surgical kidney).

5. Pressure upon the ureter, by causing the urine to be dammed back, and this urine subsequently undergoing putrefaction may cause intense pyelitis.

6. Injuries to the spinal cord.

Symptoms.—Depend upon the cause, the stage, and the complications. A simple pyelitis gives rise to but few definite symptoms, a localised pain in the lumbar region may be the chief or only complaint on the part of the patient, or perhaps he seeks advice on account of passing too little urine, which, on examination, may be found to contain epithelia, blood, etc. When suppuration commences the symptoms are much more definite: the urine now contains blood, pus cells, and albumin,

but may be still faintly acid in reaction. As the kidney *substance* becomes involved, the symptoms of more or less acute nephritis appear. If the suppurative process becomes marked, there are usually rigors, sweating, and fluctuating temperature, and the urine becomes ammoniacal, viscid, and "ropy," from the excessive amount of mucus and pus.

Sometimes the debris chokes up the ureter and the urine which escapes by the free ureter will be clear for a short time, only to become purulent again when the plug moves away. If the pus be pent up, it then causes a cystic swelling of the renal pelvis, that is "pyo-nephrosis. It will of course be easily understood, that if such a condition occurred in both kidneys, death would speedily result from uræmia.

The physical signs of pyonephrosis are—

A tumour which (when large) causes distinct bulging in front and behind. Such a tumour

Can be separated from the liver and spleen;
Is always crossed by the colon;
Yields a dull note on percussion; and
Tenderness on pressure.

Prognosis.—Is of course always grave, but recent advances in surgery have rendered the outlook much brighter. If left alone the condition is frequently fatal, but sometimes even after pyo-nephrosis has occured, the pus dries up, the walls of the cyst become coated with patchy layers of phosphatic deposits, and the cavity in time contracts. In other cases the abscess bursts externally into the perinephritic tissue, or into the kidney substances, or through the peritoneum. Death may be brought about by extensive lardaceous disease.

Treatment.—Our treatment should aim to—

1. Remove the cause if possible.
2. Relieve the inflamed kidney by rest, cupping, and the administration of sedative salines, such as pot. citras. with hyoscyamus, and the free drinking of warm liquids.
3. Treat complications as they arise.

The removal of a calculus, the emptying of a pyonephrotic cyst or hydronephrosis demand surgical treatment, for details of which consult a surgical work.

DIABETES INSIPIDUS.

Is a rare condition in which an excessive quantity of pale limpid urine is secreted, free from sugar, albumin, and other abnormal constituents; and accompanied by insatiable thirst.

Causes.—Unknown. Bernard produced polyuria by puncturing the fourth ventricle higher than the centre for producing saccharine urine; and it has been suggested that diabetes insipidus is due to some disturbance of this centre, but it is far more probable that the condition is due to a local vasomotor paralysis of the renal blood-vessels. It has sometimes been produced by severe mental shock. The temporary polyuria after a hysterical fit is well known.

Symptoms.—Polyuria, the watery constituents alone being increased; as much as 15 to 20 pints of this pale urine may be voided in the twenty-four hours. Dyspepsia, great thirst, mental irritability, and muscular weakness are also frequently prominent symptoms. Though the disease is rarely fatal, the persistent thirst and frequent micturition prevents sound sleep, and undermines the general health.

Treatment.—Valerianates, iron, strychnine, gallic acid, galvanism, and iodide of potassium.

Codeine & other derivatives of Hyoscyamus.

Diagnosis.—Care must be taken not to mistake this condition for the polyuria of Bright's disease, etc. Note the absence of casts, and the low sp. gr.

EXAMINATION OF THE URINE.

The examination of the urine in disease is of the utmost importance. Where quantitative analysis is required a specimen *of the urine passed in the twenty-four hours should be used,* as obviously the urine is richer in certain constituents at certain parts of the day. Observe—

1. *Quantity.*—Normal, 45 to 52 ounces (1300 cc. to 1500 cc.).

2. *Specific Gravity.*—Normal, 1015 to 1025. If above 1025, test for sugar. If necessary, estimate the total amount of solids from the sp. gr. and quantity.

3. *Odour.*—The normal odour is peculiar and characteristic It becomes ammoniacal and putrid when the urine decomposes. It smells like honey when sugar is present, and like sweet violets after the administration of turpentine.

4. *Reaction* is normally acid, except after a meal containing much vegetable food. Decomposed urine is alkaline.

5. *Colour.*—Normal, pale-straw to dark-amber.

   (1) If red, or reddish-brown, or smoky, suspect presence of blood, and search for blood corpuscles; test with guaiacum and ozonic ether, and if necessary, examine with the spectroscope.

   (2) If greenish or yellowish-brown, suspect presence of bile pigment; observe colour of froth; test for bile pigment and bile acids.

   (3) If pale, quantity large and sp. gr. over 1025, test for sugar.

6. *Deposit.*—Allow the urine to settle in a cylindrical or conical glass and a cloud of mucus invariably forms. It is always light and moves easily with the fluid.

A deposit of urates is the next most common. It is usually of a brick-dust colour, and moves easily when the vessel is inclined.

A deposit of earthy phosphates has usually a white or dirty-white appearance and is somewhat heavy.

If the deposit be slimy, it probably contains much mucin or pus.

If the urine is milky on passing, it is usually due to deposit of the earthy phosphates. It is common after a full meal, especially when vegetables have been freely partaken of; the urine in such a case is usually neutral or alkaline. Do not mistake this deposit for pus, nor confound *earthy* phosphates with triple phosphates.

	URATES.	EARTHY PHOSPHATES.
Colour	Usually brick red.	White or milky.
Heat	Usually clears up.	Unaffected.
Alkalies	Dissolve.	Do not dissolve.
Acids	Insoluble.	Soluble.

Albumin.—The tests for albumins depend on their coagulability by heat and precipitation by nitric acid, picric acid, etc. The most delicate test is—

1. *Heller's Test.*—Put a little nitric acid in a test tube, pour the albuminous urine cautiously upon it, and an opalescent ring of coagulated albumin appears above the acid. If the albumin is abundant it appears quickly; if there is only a trace of albumin, its appearance may be delayed for two or three minutes.

But nitric acid in the cold also precipitates *albumose;* this is distinguished from albumin by the precipitate dissolving when heated and returning when cooled.

In using Heller's test, remember that the addition of nitric acid to undiluted urine may cause a precipitate of *uric acid;* this, however, is always very scanty, and the microscope shows crystals. Further, if the urine be very concentrated the addition

of nitric acid may cause a precipitate of nitrate of urea, but its appearance is very different from that of the flocculent precipitate of coagulated albumin. Nitrate of urea is not thrown down when the urine is diluted before adding the acid.

2. *The quantitative test* may be roughly performed by Esbach's Albuminimeter—but since picric acid is the reagent used, it should be remembered that "*all the proteids*" in the urine, other than albumin, are thereby precipitated.

Method.—*Esbach's Albuminimeter* is a graduated tube for roughly estimating the percentage of albumin in urine. It cannot be successfully used if the percentage is large. It succeeds best when the urine is diluted until its sp. gr. is not above 1010. If necessary therefore, an equal volume or two volumes of water may require to be added to the urine, and this must be taken into account in making the calculation.

1. Fill tube with urine up to mark *U*.
2. Add picric and citric acid solution to mark *R*.
3. Mix thoroughly and set aside for twenty-four hours.
4. Read height of coagulum on scale. The numbers on the scale indicate grammes of albumin per litre of urine. Result only approximative, because the bulk of the coagulum depends much upon its density.

The most accurate method of determining the exact quantity of albumin is Brandberg's modification of Sir W. Robert's method—the process is a tedious one and requires much practice to be accurate. (For details of this test see von Jaksch's Handbook.)

Globulins in the urine are precipitated by a saturated solution of magnesium sulphate.

Peptones are tested by the Buiret test—*i.e.*, giving a pink or violet reaction with KOH and a *trace* of sulphate of copper. Peptones are not coagulated by heat.

SUGAR OR DEXTROSE IN THE URINE.

The various tests used are those of Moore, Fehling, Trommer, the phenyl-hydrazin test, the fermentation test, and by means of the polarising saccharometer.

Fehling's solution is an alkaline solution of potassio-tartrate of copper, and is made of such a standard strength, that 1 cc. *of recently* prepared Fehling's solution is reduced by 5 milligrammes of grape sugar. It may be used to demonstrate the presence of a reducing sugar, or for the quantitative estimation, but in the latter case it is better to use the Pavy-Fehling solution, which contains ammonia in addition to the other salts. The ammonia prevents the precipitation of cuprous-oxide, and thus the complete reduction is indicated at once by the disappearance of the blue colour of the re-agent.

Method.—Having removed albumin (if present) from a sample of the urine passed in twenty-four hours.—

1. Dilute the urine to 1 in 20 (5 cc. urine—95 cc. water).

2. Place the urine in a burette.

3. Measure ·50 cc. Pavy-Fehling solution, and place in porcelain basin.

4. Heat to boiling, and while boiling drop in urine until blue colour just disappears.

5. Calculate the total amount of sugar in the urine passed in twenty-four hours.

For details of the other tests consult a work on Examination of the Urine.

It must not be forgotten that traces of sugar may exist in healthy urine.

Acetone.—A very dilute alkaline solution of sodium nitro-prusside is added to the urine. If acetone be present a red

colour is developed which changes to yellow; on boiling after the addition of an acid, the yellow colour gives way to a violet hue. Acetone does not strike a Bordeaux red with ferric chloride, thus differing from æthyl-diacetic acid. (See Diabetes.)

Bile in the Urine.—

Bile pigments give from a deep yellow to almost a porter-like hue to the urine; it froths easily, *the froth being tinged with the pigment.*

Method.—Add impure nitric acid (nitric-nitrous) cautiously to the urine in a test tube, at the junction of urine and acid a display of colours occurs, due to oxidation of the pigments.

Bile Acids.—Cannot be detected in bilious urine without evaporating, or unless the amount of bile is excessive.

Method.—Add a solution of cane sugar to the urine in a test tube, shake let a drop of H_2SO_4 trickle down he tube, at the junction of the acid and froth a cherry rose-red colour will develop.

Blood in the Urine.—(See page 253.)

Urea.—The quantitative test is of the utmost importance; for by comparing the amount of nitrogenous material taken in, with the amount of urea passed, we are enabled to gauge the state of metabolism in the body. Urea is increased during active metabolism as in fevers, etc., and is of course decreased when the metabolic functions are in abeyance. It must not be forgotten, however, that the formation of urea may be for a time excessive, and yet not appear in the urine through inability of the kidneys to excrete it.

Volumetric Estimation of Urea by the Hypobromite process depends on the fact that the solution of sodium hypobromite decomposes urea into CO_2, N, and H_2O. The CO_2 combines

with the free soda, and nitrogen therefore alone passes into the burette. One gramme of urea by this method yields 371 cc. of nitrogen, therefore 37·1 nitrogen equals 1 decigramme of urea. If we measure therefore, the amount of N given off from a known quantity of urine, we can calculate the total amount of urea passed daily.

Apparatus.—Burette inverted in tall jar of water. Burette connected by elastic tube with a small glass flask in which urine is to be decomposed. A jar with water to cool urine and gas during decomposition. A glass tube to contain a measured quantity of urine.

Process—

1. Place about 15 cc. hypobromite solution in flask.

2. Place 5 cc. mixed urine of twenty-four hours in small tube, and introduce it into flask without tilting.

3. Elevate burette in jar till water inside it is at the 50 cc. line. Cork the flask, and read position of water in burette again.

4. Tilt flask slowly and mix the urine with the hypobromite. Jar should be left to cool for ten minutes.

5. Elevate burette till water inside is on the same level with that outside, and read off gas, then calculate amount of urea present in 5 cc. urine, and then the amount in the total urine in twenty-four hours.

For perfect accuracy, the volume of the gas should be corrected for temperature and barometric pressure.

It would be useless to calculate the amount of urea passed per day if we did not know how much *should* be passed. All things being equal—*i.e.*, an average diet, average labour, and average health—the excretion should at least reach three grains for every pound weight. In other words, a man weighing

150 lbs. should excrete at the lowest 450 grains of urea; he might with advantage excrete up to 500 grains, but it should not fall less than 450 grains per diem.

Uric Acid appears in the urine to the extent of 7 to 12 grains in combination with soda, etc., as urates. It is much increased in fevers, deficient action of the liver, gout, etc. In order to test for its presence we first displace it from its base by adding an acid, HCL or acetic acid, and letting the urine stand awhile. When uric acid is deposited it forms a cayenne pepper or red sand-like deposit.

Tests for Uric Acid.—1. *Garrod's Thread test.* Concentrate fluid. Place 5 cc. in a watch glass. Add ten drops glacial acetic acid. Place thread in fluid, and leave in cool place for twenty-four hours to allow crystals to form.

2. *Murexide test.* Place five drops urine in porcelain capsule. Add one drop nitric acid. Evaporate *gently* nearly to dryness. Add small drop of ammonia = purple colour, due to murexide or purpurate of ammonia.

3. *The Volumetric test* is estimated by Haycraft's method. Uric acid is made to combine with silver, forming a gelatinous precipitate; this is separated by filtration and made into a solution with nitric acid. The amount of silver is then tested by a colour test and the amount of uric acid calculated by the amount of silver found.

Chlorides.—The amount of *chlorine* excreted daily amounts to about 100 grains, chiefly in combination with soda.

Test.—Silver nitrate preceded by acidulation with nitric acid.

The chlorides are markedly diminished in all cases of croupous or fibrinous inflammations; this is particularly the case in croupous pneumonia.

Quantitative Estimation.—

Silver Method.—The solutions required are—

1. Solution of *silver nitrate*, containing 29·075 grms. of the fused salt in 1000 cc. of distilled water; of this solution 1 cc. = 0·01 grms. of sodium chloride.
2. Saturated solution of *potassium chromate* (neutral).

Method.—Take 10 cc. of the urine, and dilute with 100 cc. of distilled water. Add to it a few drops of solution (2). To this mixture in a beaker allow the standard solution (1) to drop in from a burette. A precipitate of silver chloride will occur as long as any chloride is uncombined. When the whole of the chloride is satisfied, a reddish or pink (since there is much white precipitate present) precipitate of silver chromate appears. This indicates the time to stop the addition of the silver nitrate, and the amount of the solution which has been used is read off. This will indicate the amount of silver nitrate necessary to convert all the chlorine present in 10 cc. into silver chloride. It is known that 1 cc. of the solution = 0·005837 grm. of salt, and from this the total amount of chloride present can be estimated. A correction should be made by subtracting 1 cc. of the silver solution used, as the white contains certain other substances more easily precipitated than the chromate.—("Power and Harris' Handbook.")

Phosphates.—The test for phosphates generally, have already been considered. The test for orthophosphoric acid is precipitation by uranium acetate, the precipitate being insoluble in acetic acid.

Pus.—

1. Examine under microscope for pus cells.
2. KOH added to the urine causes it to become stringy.

For the various casts and calculi found in the urine, see a work on examination of the urine. (Beale's plates are excellent.)

UNCLASSED SPECIAL DISEASES.

DIABETES MELLITUS.

A disease characterised by an increased discharge of pale urine of high specific gravity, containing a quantity of sugar and attended with great debility and progressive emaciation.

Ætiology.—Many cases are distinctly hereditary. Men are more often affected than women. Jews especially seem prone to this affection. It is rarely seen amongst children. The principal exciting causes are—

1. Tumours (especially gummata) affecting the fourth ventricle.
2. Mental disturbances, such as worry, grief, fright, etc.
3. The gouty state.

Lately much attention has been drawn to the fact that obese persons frequently suffer from diabetes; but it is quite unsettled to what extent the "fatty habit" may play the part of an ætiological factor.

Morbid Anatomy.—

1. *The Nervous System* shows no constant changes. In many cases, tumours in the medulla, and sclerosis of various sympathetic fibres have been found.

2. *The Pancreas.*—Is nearly always atrophied, fibrosed, and on section shows extensive areas of fatty degeneration, and fat necrosis.

3. *The Liver.*—Is often cirrhosed and peculiarly pigmented, the changes however are by no means constant.

4. *The Kidneys* are usually cirrhosed, though not extensively so; hyaline degeneration of the tubules is usually most marked.

5. *The Blood* shows four remarkable changes.—

(1) *The presence of polynuclear leucocytes containing much glycogen.*

(2) *An excessive amount of sugar in the plasma.*

(3) *The presence of much fat; which may form a creamy layer on the clotted blood.*

(4) *A reduction in the alkalinity of the blood.*

The Urine.—Both quantity and specific gravity are increased, the former to the extent of ten or even twenty pints per diem—the latter from 1025 to 1045. It is brilliant in colour, and notwithstanding the presence of ammonia, is acid in reaction. The quantity of sugar ranges from *two to forty grains per ounce*, or twenty ounces and upwards daily. Uric acid is not increased, but both *urea* and *phosphates* are. Amongst other constituents which must be remembered are β-oxybutyric acid, and æthyl-diacetic acid. The relations of these products to diabetes are still undetermined. *Outside the body*, glucose can, by taking up oxygen, be transformed into β-oxybutyric acid, with an intermediate stage of aldehyde. *A very tempting theory is to assume that such a change takes place in diabetes.*

Æthyl-diacetic acid is also important, for it has often been confused with and mistaken for acetone. This is of the utmost importance, for "Acetonuria" has been held responsible for causing the well known coma of diabetes. It has however been conclusively shown by von Jaksch and others, that *acetone* cannot be the *cause* of the coma; yet even von Jaksch admits that when the appearance of the acetone reaction appears in the urine, it usually heralds the onset of the coma and death. Possibly the explanation is that æthyl-diacetic acid undergoes the following changes in the alkaline plasma. It first combines with the soda in the blood; then splits up into acetone, alcohol, and the bi-carbonate of soda—$C_6H_9NaO_3 + 2H_2O = C_3H_6O + C_2H_6O + NaHCO_3$. This change would account for the

diminished alkalinity of the blood, and *possibly* the alcohol so formed may account for the coma. But the coma is usually held to be due to fat embolism, or the circulation of fatty acids, or to the same causes as uræmic coma.

Pathology.—When we come to discuss the real causation of diabetes, we are beset with great difficulties; in fact we are not even out of the realm of speculation, in spite of much hard honest labour expended on the subject; nor, do I see how we are to get nearer the truth until it is decided first by physiologists, how sugar *is formed* in the body, *where it is formed*, and *what its functions* or uses are. It would certainly be out of place here to add more speculation or guess-work on the subject. We briefly sketched the fate of carbohydrates in the opening chapters on Liver and Kidney; and we then discussed what would happen from excessive intake of carbohydrates on the one hand, and inability on the other hand, of the tissues to utilise the normal amount, and we then suggested possible explanations of the occurrence of glycosuria and diabetes. But it must be remembered that sugar may be formed by destruction of proteids. Seegen goes so far as to assume that the diabetic sugar is entirely derived from this source; he concludes that in diabetes the liver takes on an *increased* destructive power as regards proteids, and the sugar so formed, is excreted by the kidney. He, however, does not explain how or why such a peculiar and perverted state of hepatic function is brought about.

The **Pancreatic Theory** is also a tempting one, for we know—

1. That normally the pancreas is much concerned in splitting up proteids, fats, and sugar.

2. Excision of the pancreas in animals, is followed by diabetes.

3. The presence of an excessive amount of fat and sugar in the blood points not to excessive consumption of such, but

to the conclusion that they have not been sufficiently acted upon by a certain "something" which must be placed amongst either the pancreatic or hepatic functions.

Seegen's views are the outcome of much hard work and skilful reasoning, yet it is difficult to imagine a *too* active liver linked on to excess of fat in the blood. Is it not possible, however, that through a perverted state of the pancreatic functions, the food-stuffs are given up in such an incompletely digested state, that the liver is unable to perform its full metabolic functions, resulting in the formation of albumoses and fatty acids, and hence a toxic condition of the blood? The products so formed would account for many of the manifold symptoms of diabetes. It is impossible to conjecture what poisonous compounds these albumoses may not form, acting as they do like vegetable alkaloids, and ever ready to form new combinations. The relation of fatty acids to acetonuria, and also to the synthetical building up of fats cannot be over-estimated. The pathology of diabetes, then, is quite speculative, and no good can arise from a dogmatic statement that the chief blame lies with either the pancreas, liver, or nervous system; such statements only tend to keep up the ignorance and mystery surrounding this important disease.

Symptoms.—There can be no doubt that *two* forms of diabetes exist—viz., the acute and the chronic forms. In the acute form, the symptoms assume a grave type very rapidly, and post-mortem in such cases, the pancreas has been found to be extensively diseased. In the chronic form, on the other hand, the symptoms are often obscure, and beyond the peculiar characters of the urine and attacks of dyspepsia, the patient may for a long time maintain a fair standard of health.

Taking a typical case of diabetes, the characteristic symptoms are, the passage of large quantities of pale urine, great thirst, voracious appetite, progressive muscular weakness, a dry parchment-like sallow skin, emaciation, and the development of some

of the complications tabulated below. In the later stages the pulse becomes very feeble, severe attacks of diabetic dyspnœa, or "air hunger" add to the general misery, the breath gives out a peculiar sweetish apple-like odour, and only too frequently coma ends the scene suddenly. Often, however, the patient is carried off by a critical diarrhœa, pneumonia, etc.

In chronic diabetes some of the more common complications require special mention. They are—

1. *Cutaneous Lesions.*—Boils, carbuncle, eczema, especially of the vulva in women, and accompanied by intolerable itching.

2. *Visual Changes.*—Retinitis, soft cataract, and more or less optic neuritis. These are most common complications.

3. *Atrophic Changes.*—Gangrene, especially perforating ulcer of the foot, brittleness of the nails, etc.

4. *Renal Changes.*—Albuminuria; with more or less cirrhosis of the kidney and symptoms arising from such a condition.

In addition to the above common complications, there are often grave mental changes. A peculiar form of high stepping ataxia has been described by some authors. It should not be forgotten that in diabetes diarrhœa may be easily provoked, and I have seen two deaths from this cause, following the administration of simple purges. The dyspnœa is distinctly peculiar, for it is *rarely accompanied by cyanosis* and has received the name of "air hunger."

Diagnosis.—The urine properly tested at frequent intervals forms the best means of diagnosis, see "Examination of Urine." Remember the wasting of muscle may be concealed by obesity.

Prognosis.—In confirmed cases the outlook is bad. In the more chronic varieties the disease may be arrested for a considerable time. Some patients have had diabetes for twenty years.

Treatment.—Diabetic patients often eat enormously, and it must be at once apparent how difficult it is to balance the economy under such conditions, for if we limit the *carbohydrates* we must increase the *proteids*, and thus *throw an enormous amount of work upon the already handicapped liver and kidneys.* If physiology be correct, to give an excessive amount of proteids is certain to prove in time disastrous to the liver, and hence such practice is to try to cure diabetes by doubtful means. *But all authorities agree that the use of carbohydrates must be restricted to a minimum.* Fatty diet has been suggested as a compromise; but there is already an excessive amount of fat in the blood in most cases, and further it must be remembered that the tissues have the power of building up fat from the fatty acids which must be formed during excessive decomposition of proteids; still some eminent authorities advise a more or less fatty diet. Sir William Robert's dietary is appended as a guide.

ALLOW.

Butcher's meat, poultry, game and fish. Cheese, eggs, butter, fat and oil. Broths, soups, and jellies made without meal or sugar. Cabbage, endive, spinach, broccoli, lettuce, spring-onions, water-cress, celery. Dry sherry, claret, brandy and whisky. Tea, coffee (without sugar), soda-water, bitartrate of potash water.

FORBID.

All saccharine and farinaceous food, *bread*, potatoes, rice, tapioca, sago, arrow-root, maccaroni; turnips, carrots, parsnips, beans and peas.

Liver contains much sugar-forming substances, therefore, oysters, cockles, and mussels, which contain enormous livers, are forbidden.

All sweet fruits. All sweet wines.

It will be seen from the above table that there is a pretty extensive range or choice of food, but unfortunately the two great drawbacks are the non-allowance of bread and potatoes.

The substitutes for bread are either nauseous or too expensive for general use. The principal substitutes are almond biscuits, gluten bread, charred bread, soya bread, etc.

Donkin's treatment, by an exclusive diet of skimmed milk, does not usually act well, except in obese people.

MEDICINAL TREATMENT.

Codeine or morphine seem to be the most beneficial drugs. Antipyrin is credited with the power of reducing the amount of sugar, if given in doses of ten grains thrice daily. Saccharine and glycerine are no good as curative agents, but they may be used for sweetening purposes. Arsenic with strychnine are useful tonics. Massage, electricity, cod-liver oil, etc., are all useful in various cases. The great thirst may be relieved by the administration of large doses of citrate of potassium freely diluted. The complications must be treated on those principles laid down under their respective headings.

MYXŒDEMA.

A disease associated with atrophy of the thyroid gland, a peculiar condition of the skin, over-growth of the subcutaneous tissue, and serious mental conditions.

Ætiology.— Most common in women between thirty and fifty-five years of age; hereditary factors have not yet been proved; the poorer classes are said to furnish the larger number of cases.

Pathology.—That the disease is dependant on atrophic changes in the thyroid gland seems now certain, reasoning from the results of the removal of the gland on the one hand, and from the beneficial effects following the internal administration of the healthy thyroid.

Professor Horsley distinguishes three stages after removal of the thyroid gland in *monkeys*.

STAGES.	DURATION.	SYMPTOMS.	REMARKS.
1. *Neurotic*	1 to 3 weeks.	Tremors, rigidity, dyspnœa.	*Young* monkeys often die in this stage.
2. *Mucinoid*	3 to 7 weeks.	Commencing hebitude, and mucinoid degeneration of the connective tissue.	*Older* monkeys die at this stage if not treated.
3. *Atrophic*	5 to 8 weeks.	Complete imbecility, atrophy of all the tissues.	Monkeys survive if kept in warm air bath.

The untoward symptoms were associated with enormous fall of blood pressure and temperature.

It will therefore be seen that the presence of the thyroid is necessary to avert myxœdema. Possibly its normal functions are *to keep the blood pure and free from some pernicious products*, the nature of which has not yet been discovered, but which if retained causes myxœdema.

The morbid anatomical changes are most marked in the **subcutaneous tissues**—

1. Nuclear proliferation or formation of connective tissue all around the hair follicles and sweat glands.
2. Increased deposit of subcutaneous fat.
3. Presence of mucin (not always present.)
4. Formation of elastic œdematous swellings above the clavicles.

The **Thyroid Gland** is atrophied, often indurated, and shows scattered groups of cells, the remains of the normal vesicles.

Interstitial nephritis has been observed in some cases, but this is probably an accidental coincidence.

Symptoms.—Patient presents a heavy stolid countenance, with injected or diffuse red cheeks. The hair is scant, coarse, and brittle. She speaks in a heavy, slow, and thick manner (leathery voice). Exhibits great intolerance of cold—skin is

enormously thickened, *dry*, and often scaly, but does not pit on pressure. The hands are said to be spade-like. Teeth become carious. The gait is peculiarly clumsy. Bowels are constipated. Later, the mental condition becomes very grave; hallucinations, convulsions, and coma may occur. It must not be forgotten that often towards the end albuminuria may occur, and the skin may pit on pressure.

Treatment.—Guard patient against cold—give nourishing food. Pilocarpine and arsenic are useful sometimes.

Specific Treatment.—Injections of—

1. Emulsionised thyroid gland,
2. Extract of thyroid gland hypodermically.

Lately, it has been shown that eating the cooked thyroid is equally efficacious.

SPORADIC CRETINISM.

Is to be regarded as the infantile form of myxœdema, and, like that disease, is due either to the congenital absence of the thyroid body, or an absence of its functions. Formerly it was assumed to be a form of goitre, and endemic in certain districts; but an enlarged thyroid has rarely been associated with cretinism, and the cases cited have not been fully investigated, as the thyroids were not examined post-mortem.

Symptoms and Signs.—The condition as regards the subcutaneous œdema, supra-clavicular swellings, and mental defects are much the same as in myxœdema, only more marked. No disease presents more strikingly characteristic features. The child is dwarfed or stunted in growth; the face is very ugly, moon-shaped, and the cheeks hang in pendulous folds; tongue is too large for the mouth; and the voice is peculiarly harsh or squeaky, like Punch in the show. The hair is abundant but coarse, except over the swellings between the scapulæ, which are covered by a soft down-like hair. The swellings are due

to fatty deposits. The belly is very prominent and pendulous; umbilical and inguinal hernia are very common. The gait is clumsy, and of a waddling type. The sexual organs are rarely developed, though in female children the menses may appear once or twice; but sexual desire is never excited, even in those cases that live to adult age. Cretins are usually incapable of being taught reading or writing, and their vocabulary is always limited. They are often peevish and cross, but the bulk are placcid and affectionate. Death most often occurs during childhood, though a few cases have reached adult life, without however advancing at all in intelligence.

Treatment.—At present hopeless. A trial may be made of the same treatment as in myxœdema. Granting even the possibility of arresting the disease, it seems doubtful if the brain would under such circumstances greatly improve and develop, unless the treatment be commenced early.

Ætiology.—Nothing definite is known. For examination purposes it may as well be remembered that the following theories have been advanced as ætiological factors:—

1. That the ancestors of cretinic children have suffered from goitre, and thereby transmitted a weakness in the development of the thyroid body in successive generations.

2. That such children were conceived during intoxication on the part of one or both parents. If this had any claim for serious attention, we should expect sporadic cretinism to be very common, considering the large families many drunken parents rear. It must be admitted, however, that drunken parents are often sterile, showing some change in the condition necessary for procreation.

3. Traumatisms during fœtal life. This is too vague for serious thought.

Lastly, it would be better not to bias or retard future research by assuming *any* ætiological factor at present.

ADDISON'S DISEASE.

Is a peculiar constitutional affection, first described in 1855, by Dr Addison of Guy's Hospital, as a "disease of the supra-renal capsules, attended with a bronze discolouration of the skin, and incurable progressive anæmia. His description, however, requires modification; as *anæmia* is by no means always a prominent feature. Dr Byrom Bramwell sums up the condition in his beautiful atlas (page 51), as a condition marked by—

1. *Asthenia*, feebleness of the action of the heart, and the symptoms and signs which result therefrom.

2. *Nausea*, vomiting, and other symptoms which result from gastro-intestinal irritation.

3. *Pain* in the neighbourhood of the supra-renal bodies.

4. Pigmentation of the skin and mucous membranes.

5. Anæmia.—Often absent until the later stages.

6. Symptoms due to derangements of the cerebro-spinal nerve apparatus, such as headache, delirium, convulsions, etc.

7. The *absence* of—

   (1) Elevation of temperature.

   (2) Marked emaciation.

   (3) Signs or symptoms of local organic disease (other than the supra-renal capsules) to account for the asthenia and other symptoms enumerated.

Pathology.—At present is obscure. All observers agree that it is essentially a disease of the adrenal bodies, plus implication of the abdominal sympathetic system. Some lay most stress upon the adrenal disease; others upon the involvement of the sympathetic system. Bramwell, after a most exhaustive

analysis of this disease, considers it "*to be, in the majority of cases, a tubercular degeneration of the capsules primarily, and that the alterations in the nervous structures are secondary.*"

So far so good, but when we attempt to explain the symptoms, we are at once floored by the fact that we know little about the functions of the abdominal sympathetic, and still less about the adrenal bodies.

It is held, judging from the enormous vascular supply of the adrenal bodies, that they are blood glands, and that their function is to prevent the retention within the system of certain poisonous products, which, if retained, produce the peculiar symptoms of Addison's disease; and if we bear in mind the case of thyroid myxœdema, this sounds feasible.

If it be due to the involvement of the sympathetic, the symptoms must be the outcome of atrophic changes, and the consequent disturbed and imperfect metabolism through imperfect innervation.

Anatomical Changes are—

1. Atrophy of one or both glands; presenting
2. The various stages of tubercular degeneration—
    (1) Interstitial growth.
    (2) Fatty degenerated patches.
    (3) Caseous masses.

The semilunar ganglia are degenerated and much pigmented, and they may through cicatricial contraction become entangled as it were in the diseased tissue of the adrenals. The nerve fibres show extensive sclerotic changes.

Dr Alezais and Arnaud assert that "Addison's disease will not result if the *pericapsular ganglia* be not affected, though the other portions of the adrenal bodies are at the same time extensively diseased." We have already summed up the main symptoms. It is peculiar that even when the exhaustion and anæmia are profound, that the temperature tends to keep down; this is in marked contrast to pernicious anæmia.

The pigmentation is most marked where pigment is normally found—*i.e.*, areolæ, scrotum, axilla, etc. The mucous membranes are often deeply pigmented, presenting blackish patches. The marked exhaustion is altogether out of proportion to the general condition, and it must not be forgotten that the slightest untoward circumstance, such as a simple purge or chill, may cause death. Provided, however, the patient be treated as a hothouse plant, the disease may run a protracted course. In some cases, after very slight symptoms (and a still slighter amount of bronzing) have been noticed for a few months, the patient, without any obvious cause, sinks into a semi-comatose state which rapidly proves fatal.

Diagnosis.—It must be remembered that bronzing may occur in pregnancy, through exposure to the sun, through dirt and vermin, in diabetes, etc.

Treatment can be summed up as follows—Treat the patient as a rare and valuable exotic plant. It has been suggested to introduce the extract, etc., of healthy adrenal bodies, after the manner of the thyroid in myxœdema. Arsenic, phosphorus, iron, strychnine, etc., have their advocates.

RHEUMATIC FEVER.

An acute febrile disease characterised by poly-arthritis, a tendency to hyper-pyrexia, a special tendency to involve the pericardium and endocardium, and marked anæmia.

Ætiology.—The larger number of cases can be traced to exposure to cold, inclement weather, etc. It is most common during spring, in humid climates, and affects *young* adults most frequently. Males are more often attacked than females.

Pathology.—The true cause or causes of rheumatic fever are still unknown; and never has there been a clearer case of

arrested research through the dogmatic statement "that acute rheumatism is due to the presence of lactic acid." Lactic acid has never been found in excessive quantity in the blood, or in the effusion of the inflamed joints in this affection; moreover, the only evidence yet advanced in favour of such a theory, is that certain physicians have produced acute arthritis by injecting lactic acid. All I can say is that we can produce a similar arthritis by injecting many other substances.

The Germ Theory assumes that the arthritis is due to a specific but as yet undiscovered microbe; and certainly reasoning from analogy of many other acute affections, this seems a feasible theory.

The Nervous Theory assumes that the nerve centres are primarily affected by cold and exposure; this interferes with metabolism; the products of imperfect metabolism accumulate and irritate certain tissues, which are also rendered more vulnerable by the diminished trophic influence, through the depression of the said nerve centres.

The nature of the materies morbi is therefore still undecided and offers a grand field for original research. One thing, however, is certain, that the poison causes a very rapid destruction of red corpuscles.

Morbid Anatomy.—The synovial membrane undergoes the changes common to inflammation of serous membranes—viz., hyperæmia, exudation of lymph, and effusion of fluid. The fluid is usually turbid, but seldom becomes purulent. The ligamentous structures are swollen, and often the cartilages are eroded by central necrosis. The blood, though it contains an excess of fibrin, clots more slowly than normal; this, however, occurs in many other conditions.

The fluid of the secondary affections such as pericarditis, pleurisy, and the vegetations of the endocarditis contain pus organisms.

Symptoms.—After a feeling of more or less soreness, and general stiffness, the pain becomes localised in one or more joints: usually the knee, wrists, or ankles.

The arthritis extends very rapidly to any of the medium-sized joints. At first the affected joints are red, hot, swollen, and intensely painful; later, the redness becomes less marked, and the joint may assume a dead white appearance. Frequently the inflammation rapidly subsides in one joint only to appear in another.

The temperature may be very high, even to 107°; indeed, hyper-pyrexia is common in this fever. In the bulk of cases, however, it remains between 101° and 103°.

Marked sweating of a peculiar sourish smell is a constant feature, and the various hair follicles, and other cutaneous glands may become inflamed and painful thereby.

As the disease advances *anæmia becomes very pronounced.* This anæmia may be increased by the salycilates, but is mainly due to the rheumatic poison.

The tongue is usually very large, flat, and covered with such an extremely thick fur that it has been named the "blanket" tongue.

The urine is scanty, highly coloured, and often loaded with urates.

The more common complications are—

1. Pericarditis.
2. Endocarditis.
3. Pleurisy.
4. Hyper-pyrexia.
5. Certain skin eruptions, such as sudamina, milaria, and "pelioses" or small red petechial spots around the ankles.
6. Pharyngitis and tonsillitis.
7. Meningitis.

Frequently subcutaneous fibrous nodules develop over bony ridges. Though rarely fatal, and though the severe symptoms usually subside in about fourteen days, no disease is more often attended with relapses and grave results. Chorea, valvular diseases of the heart, etc., constantly follow in its wake.

Treatment as regards the general management. See there is absolute rest, perfect protection of the limbs by wrapping them up in cotton-wool, and support the bed-clothes by cradles. The diet should consist principally of milk and soda water; beef-tea and chicken may be allowed later on.

As regards treatment, **undoubtedly the salycilates rank first now-a-days.** There can be no two opinions as to their efficacy in easing pain, but from the experience of fourteen well-marked and well-watched cases, I share the belief of that school which attaches more value to the old alkaline treatment—*i.e.*, letting the patient drink *ad libitum* of citrate or bicarbonate of potash, with an occasional dose of quinine, and the administration of pulv. ipecac. co. to ease the pain. Dr. Brackenridge asserts that salycilates if *pushed*, even when heart failure threatens, is the only treatment for rheumatic fever. A solution of the salycilates locally as a lotion or fomentation is of the utmost value. Pot. iodid. may be combined with salycilates in obstinate cases. Treat complications on rational principles already laid down.

GOUT.

Is a disorder possibly nutritional in its character, associated with an excessive amount of uric acid in the blood, and deposits of biurate of sodium in the joints. Clinically, it is manifested by periodical attacks of acute arthritis, certain visceral disturbances, and later, deformity of the joints attacked.

Ætiology.—1. Heredity is certainly a most important factor, and is curiously developed in the grand-children.

2. Alcoholism, especially indulgence in sweet wines, and heavy malt liquors. Strange to say, Italians who drink large quantities of light wines, and Scotsmen who certainly freely indulge in good whisky, are singularly free from gout.

3. Gluttony. This is as important a factor as excessive drinking.

4. Certain poisons; such as lead, etc., which tend to decrease the metabolic activity of the body.

5. Insufficient food (the poor man's gout). This condition however, is associated with defective hygiene, and only too frequently, with excessive consumption of poor or raw spirits, etc.

Pathology.—In no disease can we point to a more constant feature than excess of uric acid in cases of gout. This salt is found in excess in the gouty blood in the gouty joints, and in the exuded serum of the gouty arthritis; therefore there cannot be the least possible doubt that the disease is due either to the accumulation of uric acid in excess, or, *to those conditions which give rise to the excessive formation of uric acid.* Now, if we were to inject uric acid into the blood of a fairly healthy individual we would not produce gout; and, moreover, von Jaksch and others show that uric acid may be always produced by deficient oxidation, and that excess of uric acid is common in many diseases, such as leucocythæmia, pernicious anæmia, etc.; yet gouty arthritis is by no means a common complication in these diseases, therefore there must be a "something more" than mere uric acid in excess to cause such a condition as gout.

Sir Dyce Duckworth assumes, "There is a basic arthritic stock—a diathetic habit of which gout and rheumatism are two distinct branches. The gouty diathesis is expressed in (1) a neurosis of the nerve centres, which may be inherited or acquired; and (2) a peculiar incapacity for normal elaboration within the whole body, not merely in the liver, or in one or two organs of food, whereby uric acid is formed at times, or is

incapable of being duly transformed into more soluble and less noxious products." (Sir Dyce Duckworth's Handbook on Gout.) According to such an eminent authority, gout means something more than uric acid in excess.

Sir William Roberts has shown that *normal* uric acid is a quadurate which requires an alkaline medium in order to be kept in a perfectly soluble condition. In other words, the quadurate is deposited in a *weak* alkaline medium as a biurate compound, a salt which is found in the joints affected by gout. I would suggest the following statement as representing our present knowledge of the pathology of gout:—Mere excessive formation of uric acid within the body is either the outcome of excessive nitrogenous (or proteid) consumption, or of deficient oxidation on the part of the tissues (liver, etc.) This condition constitutes the "lithæmia" of the older writers, and is one highly predisposing to gout. Now, if we assume that through certain changes, nervous or otherwise, the blood, or certain tissues, become less alkaline than is sufficient to hold the quadurate in solution, a precipitation of a more insoluble salt, viz., the biurate, must occur; and, if such a precipitation takes place in the joints, no wonder that acute arthritis is excited thereby. Excess of the quadurate may be physiological and compatible for a time with health; *deposit of the biurate, on the other hand, is pathological, and results in gout.* This seems rational when we remember that the circulation in the smaller joints is very sluggish, and consequently such parts are prone to low inflammatory processes, resulting in a local diminished alkalinity of the blood, thus favouring the local precipitation of the circulating quadurate. The consequent arthritis and febrile disturbance set up further explains why gouty patients feel so much better after an attack of gout, for the increase of metabolism of the febrile state, and the alkaline treatment, all tend to burn up as it were, or promote the excretion of the excessive uric acid in the blood. The restricted diet also gives the liver a holiday, and enables it to perform its functions with renewed vigour.

Further, our theory harmonises with the suggestion that gout is more or less of a neurotic origin, for we pre-suppose *some* devitalisation of the tissues and joints, and joints are notoriously influenced by atrophic nervous changes, as witness Charcot's joint, etc.

Let us remember one thing more, that once a joint is affected with gout, if the deposit is not all cleared away, like a calculus in the bladder, it simply forms a nidus for the formation of a large concretion.

Morbid Anatomy.—The metatarso-phalangeal joint of the great toe is most often affected. If we examine the joint at various stages, we find the following conditions—

A deposit of fine crystalline needles in the *superficial* parts of the cartilages; next, the synovial membranes, cartilages, and ligaments become covered with a chalk-like deposit of biurate of soda. Underneath the deposit, the tissues are in a more or less necrosed condition. The synovial fluid contains crystals of uric acid.

Later, collections of biurate of soda *plus* calcium phosphate form the so-called chalk stones or *tophi* of chronic gout. These deposits may occur elsewhere than in the joints, such as the lobes of the ear, tendinous aponeurosis of muscles, sclerotic of the eye, etc. Garrod believes these concretions are merely calcification of gouty inflammatory products, and no doubt he is right.

Symptoms of Acute Gout.—Usually there are some premonitory symptoms, such as giddiness, mental depression, flatulence, irritability of temper, scanty and high-coloured urine, etc. The attack most commonly commences in the early hours of the morning, with severe pain in the big toe. The pain increases to acute agony in some cases, rendering sleep or rest out of the question. The patient becomes very feverish, or he may feel chilly, and shiver violently without any rise of temperature. The joint at first is bright red, and exquisitely

tender; later, it is more swollen, and of a livid or dull dusky red, with distended venules standing out. The swelling extends some distance from the joints. The skin desquamates in thick flakes when the attack is over. An attack lasts from five to twelve days, but the severe pain is not constant, or rather there are lulls, with exacerbations of severe pain at intervals of the day, especially at night-time. The urine at first is scanty and high-coloured, *but the uric acid is diminished in amount;* later, the excretion of *uric acid is very much increased.* During the attack the patient usually is most irritable, the tongue is furred, the breath may be offensive, he has no appetite, and the bowels are constipated. Subsequent attacks may affect the same joint first implicated, or a number of joints become involved. If the attacks are fairly frequent, they cause the so-called chronic gout; that is, a condition characterised by a characteristic deformity of the joints, important changes in various organs, especially the red granular cirrhotic, or "gouty kidney."

No disease is quoted more frequently than chronic gout as an important ætiological factor in the causation of other diseases. Such a statement, however, is largely the outcome of habit and fashion. Many eminent men employ the terms, "gouty habit," "specific taint," in a ridiculously loose fashion. It is sometimes actually refreshing to hear a nervous disease described as *not* dependant on one of these causes.

Chronic Gout.—Must not be confounded with rheumatoid arthritis, though the sufferer may be crippled in much the same manner.

The small joints of the toes and fingers are most often affected. They are often distended by "tophi," which at first lie under the skin, but may ulcerate through.

The kidney undergoes a marked cirrhotic change (see Chronic Bright's Disease). In such cases, the pyramidal area of the kidney often show yellowish-white streaks of urate crystals.

Other complications than those of "gouty kidney," are—

1. Œdema of the glottis, which is most easily excited, and is singularly fatal in the gouty state.

2. "Gouty" urethritis, which may resemble a gleet.

3. Renal calculi.

4. Extensive sclerosis of the arteries, and the dangers arising therefrom.

5. Retention of urine.

6. Cirrhosis of the liver, with its train of symptoms.

Treatment.—During the acute stage—

1. Absolute rest, elevation of the limb, and hot fomentations to the joints affected.

2. Diet to consist of milk and potash water, or at least *non-nitrogenous* food.

3. Medicinal treatment.—Clear out the bowels with five grains of calomel, followed by a full dose of Carlsbad salts. Give a mixture containing colchicum, iodide of potassium, bicarbonate of potassium, and hyoscyamus. Iodide of potassium, with laudanum and salycilate of soda added to the hot fomentations, speedily relieves the painful joint.

When the acute attack subsides, massage must be employed, for it is absurd to think "medicines" will remove the biurate deposit from the joints.

Treatment of Chronic Gout.—Careful regulation of diet, avoidance of alcoholic excesses, and proper exercise, are the principal points to remember. Arsenic and strychnine as medicines, lithia water as an habitual beverage, frequent Turkish baths (when they agree), and massage to the affected limbs, go far to complete a cure.

RICKETS.

Rickets is a general disorder, which attacks infants and young children who are subjected to improper feeding, want of sunlight, and other unhealthy hygienic conditions.

Clinically, it is manifested by wasting, stunted growth, characteristic physiognomy and deformities.

Ætiology.—Occurs mostly among the poor of overcrowded cities. It is occasionally seen among the offspring of the wealthy in whom there is a taint of syphilis or tuberculosis. There cannot be any doubt that many cases of congenital syphilis subsequently develop rickets. The *absence of lime salts* in the food must be looked upon as an important ætiological factor, if such absence is prolonged. Often, however, calcium salts are abundantly supplied in very marked cases of rickets.

Pathology.—Again we have to do with a disease, the causation of which is shrouded in mystery. We must first briefly consider the more constant morbid anatomical characters.

1. Change in bones—
   (1) Their specific gravity falls, the water and organic matter are increased, whilst *the earthy matter is decreased.*
   (2) The amount of fat is increased.
   (3) They occasionally do not yield normal gelatine.
   (4) The epiphyseal ends show under the microscope—increased vascularity, irregular calcification, small islets of uncalcified matrix, and large spaces due to absorption by osteoclasts; at the junction of the epiphysis with the shaft, the bluish cartilagenous zone is thickened, and very irregular; the bone formed is more vascular and spongy than ordinary bone.

2. The blood changes are a deficiency of red corpuscles, and of hæmoglobin in the corpuscles present; associated with the *presence* of much granular detritus (Goodhart).

3. Changes in the lymphoid tissues.—The spleen, and lymphatic glands nearly always show extensive fibrous changes.

As rickets is often associated with a too excessive administration of starches, it has been assumed that lactic acid is thereby formed in abundance, and that this excess of acid is the real cause of rickets, by causing solution of the calcium salts. This has not been proved, and, according to Gamgee, cannot be the cause. But I must add, that whilst Gamgee has held this theory up to ridicule, his chemical criticism is by no means faultless.

From a careful consideration of the morbid anatomy, we must assume at present that rickets is due to either—

1. Non-absorption into the blood of lime salts, through some morbid condition of the alimentary canal; or—
2. An inability on the part of the bone-forming tissues to appropriate the lime which is conveyed to them.

The first theory may be usefully contrasted with the explanation of chlorosis—*i.e.*, disordered condition of the alimentary canal, interfering with the assimilation of iron.

Symptoms.—The symptoms of a typical case of rickets are very characteristic.

General configuration.—The head is elongated from back to front, the forehead is square and overhanging, the fontanelles are slow in closing, and the veins in the skin are distended. The flat bones are usually much thickened, and indeed, are frequently "bossed" by irregular masses of calcified material. It is important, however, to remember that very often the occipital bone, and certain parts of the parietal bones are *thinned* out. Though the skull as a whole is enlarged, the

puny face makes the enlargement more noticeable by comparison; measurements show the enlargement to be more apparent than real. The brain development may be seriously handicapped by this peculiar deformity, inasmuch as it allows the hind part to develop, but not the intellectual portion—*i.e.*, the frontal lobes. Hydrocephalus is a somewhat common complication.

The ribs often show characteristic "beading" at the junction of the costal cartilages. The thorax tends to become cottage-loaf in shape.

The lower limbs are bowed, or sickle-shaped, and show the well marked epiphyseal enlargements, especially at the lower end of the *tibia.*

The upper limbs also show the most marked changes at the lower ends of the ulna and radius, but the humerus, scapula, etc., are also affected.

The pelvis is often much deformed, and may add serious complications to parturition.

The spine is frequently distorted.

It will be seen from the above, that the deformities depend partly on excessive deposits of calcareous material, and to a certain extent to the yielding of the softened bone to mechanical causes; hence the necessity of rickety subjects avoiding any undue or avoidable strain, such as too early walking, sitting in cramped positions, carrying weights, etc.

The abdomen is usually prominent from distension by gas, and the presence of the enlarged liver and spleen.

The other clinical symptoms are—general and marked tenderness of the body, throwing off the bed clothes at night, profuse sweating, and constipation alternating with diarrhœa. The stools are usually pasty, or green, like boiled spinach, and are intensely fœtid at times. These symptoms, taken with the general emaciation and deformities already

described, make a diagnosis very easy. Of the complications there are no end, but we must specially mention, convulsions, tetany, strabismus, and laryngismus stridulus. Green-stick fractures are not uncommon.

Treatment.—No cases deserve and require more thoughtful and careful treatment. If the mother be unhealthy, a wet nurse, or the "sterilised milk" plan of artificial feeding must be prescribed. Plenty of fresh air, sunny rooms, and gentle massage, must be the routine treatment in all cases. The diet for older children must be varied, and an excess of carbohydrates avoided. Carefully planned gymnastic exercises may help to prevent deformities, or cure those already formed. Of drugs, phosphorus, lacto-phosphate of iron, arsenic, iodide of iron, cod-liver oil, etc., all have their advocates. But careful hygiene is of far more value than medicinal treatment. Dr Palm, of Wigton, points out "that sunlight in the dwelling not only reveals unsuspected dirt, but is nature's disinfectant as well as a stimulant and tonic." There can be no possible doubt as to the all important influence of sunlight to consumers of large quantities of carbohydrates.

CHRONIC RHEUMATISM.

Chronic rheumatism is sometimes a sequel of rheumatic fever, but is generally a separate constitutional affection.

Ætiology.—Is most common amongst the middle-aged poor. Senility seems to be a predisposing cause, both amongst poor and rich.

Pathology.—Is at present ill-understood; the fibrous textures around joints, enveloping nerves, or the aponeurotic sheaths of the muscles, periosteum of bones and tendons all suffer more or less. Though these structures become

inflamed, very little alteration results—*i.e.*, the joints are rarely deformed, though the ligaments and synovial membranes may become much thickened, and the movements of the joint limited.

Symptoms.—Pain and stiffness of the parts involved are the main features. The pain is usually worse on movement, but it should be noted that continued exercise mitigates the pain: for example, a man goes to bed with muscular rheumatism, as long as he keeps quiet the pain is not severe, but the slightest movement causes acute suffering. In the morning, on attempting to rise the pain may be very severe, but after working for a time the pain becomes tolerable though it does not disappear.

Varieties—

1. *Lumbago.*—The aponeurosis of the erector spinal and latissimus dorsi are most frequently involved. The pain is often intense, and may affect the locomotion markedly. Often the onset of the pain is traced to some physical exertion, such as getting up into the saddle, or lifting heavy weights. The latter point must be borne in mind in colliery practices, for the colliers frequently demand pay on account of injury—*i.e.*, "strained back."

2. *Pleurodynia.*—The sheaths of the pectoral muscles, intercostals, or serratus magnus, are most commonly affected. The respiratory movements of the affected side are much embarrassed. This affection may be mistaken for pleurisy, as the movements of the affected muscles often cause a distinct fremitus. The absence of other physical signs should prevent such an error.

3. *Muscular Torticollis, or Stiff Neck.*—Here the cervical muscles, especially the sterno-mastoid, are affected. This condition must not be confounded with spasmodic torticollis. Many other varieties are described.

Treatment.—Keep the bowels well open by saline aperients. Turkish baths and massage, combined with the administration of iodides, form the routine treatment. The A. B. C. liniment is most useful for local application. If the condition does not speedily improve, a visit to Buxton or Bath, etc., should be insisted upon, where the patient may undergo a thorough system of massage, and course of saline waters.

RHEUMATOID ARTHRITIS.

Rheumatoid arthritis is a chronic inflammatory affection of the joints, resembling gout and chronic rheumatism in many of its characters, but differing essentially from both. Although a chronic disease, the patient may have acute or subacute attacks superadded.

Ætiology.—Most common amongst females, between the age of twenty and thirty. The disease may, however, attack any age. Heredity, phthisical history, ovaritis, grief, worry, etc., are looked upon as important ætiological factors.

Pathology.—At present speculative. Probably it is due to neuro-trophic changes; and reasoning from the number of joints attacked, the symmetrical distribution of the trophic changes which lead to such grave alterations in the skin, nails, etc., this view seems to be the correct one.

The morbid changes found in the joints affected, are identical with those of Charcot's joint disease, so frequently a complication in cases of locomotor ataxia. According to Adams, the disease begins in the cartilages and synovial membranes—as splitting of the cartilage and liberation of the cells; next, absorption of the cartilage and approximation of the two articular surfaces of the joint, which by rubbing together, become very dense, hard, and highly polished; the surfaces at the same time, through pressure, become broadened out—*i.e.*, lateral

expansion. Underneath the new and dense ivory-like bone, rarefaction and atrophy occur, leading to much shortening and deformity.

Meanwhile the synovial membranes become inflamed and thickened; often portions become detached, and form loose bodies in the joint. The ligaments become much thickened, and often contracted; sometimes they calcify and cause more or less ankylosis. At the margins of the joints where the pressure is less, ossification goes on, resulting in the formation of irregular bony outgrowths, termed osteophytes. The ankylosis of rheumatoid arthritis is never complete, and there is no tendency to suppuration.

Symptoms—

The Ordinary Chronic Form.—The joints are involved symmetrically. At first the joints may be swollen, red, and tender, but the more constant features are stiffness and gradual deformity, without any marked signs of inflammation. In confirmed cases the joint changes, and the accompanying muscular atrophy cause the deformities to assume a very characteristic appearance. The lower ends of the ulna and radius project at the wrist, the metacarpo-phalangeal are flexed, the first phalangeal joints are over-extended, the second are flexed, and the fingers deviate to the ulnar side. The joints give forth a creaking sound when moved.

When the disease is confined to the hands, the osteophytes receives the name of "Hebeden's Nodosities." After the hands and wrist, the knees and ankles are most commonly attacked, but cases have been known where all the articulations of the body have become implicated. The muscular atrophy, though no doubt due largely to atrophic influences, is largely contributed to by non-use.

Besides Heberden's nodosities, a mono-articular form of rheumatoid arthritis has been described. This type differs from the poly-articular form, *by affecting the spine or hip-joint, in elderly men.*

Diagnosis.—From Charcot's joint, by the history and absence of ataxic symptoms. From gout, by the absence of tophi, no excess of uric acid, etc.

Treatment.—No treatment can cure this disease, but it is often arrested by careful attention to the general health. Arsenic and iodide of potassium with strychnine are often of great service. Massage, however, is probably the best treatment that has yet been adopted.

GONORRHŒAL SYNOVITIS.

Gonorrhœal synovitis is a more or less acute arthritis, following in the wake of gonorrhœa, and probably due to the introduction of some specific virus. It must not, however, be concluded that it is the gonorrhœal poison which sets up the condition, for it has followed balanitis and ordinary profuse leucorrhœa. Probably the gonorrhœal poison creates a suitable nidus for another specific organism to flourish. Some authorities still hold that it is ordinary acute rheumatism added to a gonorrhœa.

Symptoms.—They are much the same as regards the joint condition described under Rheumatic Fever, but the redness may be more intense. The fever is not usually high. The peculiarities of the sub-acute cases are—iritis, extreme pain in the plantar fascia, the occurrence of conjunctivitis, and the tendency to involve the sterno-clavicular and temporo-maxillary joints—*i.e.*, those joints *not* usually involved in ordinary acute rheumatism. The disease is extremely likely to recur, and may render life miserable.

Treatment.—The alkaline treatment may be pursued for a short time, but quinine and liquor ferri are the best drugs in this affection. If the joints remain obstinately swollen, nothing is more efficacious than the hypodermic injection of two or three minims of carbolic acid into them. The actual cautery may be of use in chronic cases; galvanism, etc.

DISEASES OF THE BLOOD.

Healthy blood means perfect health, therefore in all diseases the blood must be altered in its quality; so it is difficult to explain what is really meant by the above heading; but, under this title we mean those diseases which show the most marked changes in the blood, either in quantity or quality.

ANÆMIA.

Anæmia really means want of blood, but, we employ the term in a much wider sense, and we shall under anæmia, include—

1. Oligæmia—deficiency of blood.
2. Hydræmia—thin or watery blood.
3. Oligocythæmia—a diminution of the number of corpuscles.
4. Poikilocythæmia—irregularity in the shape of the corpuscles.

Special Anæmias.—

(1) Simple.
(2) Chlorosis.
(3) Pernicious Anæmia.

It will save much recapitulation and help to avoid much haziness regarding these affections, if we start with a clear idea what anæmia really means, as regards its effects upon the body generally.

Pathology. — We must expect imperfect oxygenation, deficient nutrition, and, therefore—impaired function, fatty degeneration, atrophy of certain tissues, and, softening of the blood-vessels; a vicious cycle of retrograde changes thus begins, each new failure on the part of an organ increasing the original

condition. The nerve centres are only too frequently hyper-excited at first, as shown by powerful emotional manifestations; or diminished sensibility, as seen in the dilated pupils, which shows the depressed condition of the retina. Œdema in unsupported tissues is most common.

SIMPLE ANÆMIA.

Causes.—

Deficiency of food, either through actual want, or inability to take food—as in cancer of the œsophagus, etc.

Excessive discharges—i.e., pyæmic abscesses, diarrhœa, etc.

Hæmorrhages.—The blood in these cases, if the bleeding is profuse, becomes rapidly diluted with lymph.

Certain occupations—lead workers, colliers, etc.

In these cases, removal of the cause, and placing the patient under favourable hygienic conditions, bring about a cure.

CHLOROSIS.

The usual definition in text-books is,—a peculiar anæmia attended with a greenish, transparent, wax-like condition of the skin, common in females at the age of puberty.

Ætiology.—From a careful observation of the cases deserving the name "green sickness," seen in the out-patient department of Edinburgh Royal Infirmary, I am convinced that the condition is more common at a slightly *older* age than puberty, *i.e.*, between eighteen and twenty-one years.

Symptoms.—Languor, palpitation on exertion, headache, noises in the ears, and dyspeptic symptoms, as in simple anæmia. The more characteristic symptoms in addition are

plumpness, systolic murmurs at the base of the heart, "*bruit de diable*" at the lower end of the jugular vein, constipation, irregular, profuse, or scanty menstruation, and the waxy appearance of the skin already referred to.

Pathology.—The red corpuscles are diminished in number, and of various shapes; some swollen, others crenated, but all *show a remarkable diminution of hæmoglobin*, which must be considered the chief peculiarity of chlorosis. There cannot be the least possible doubt that chlorosis is due to defective blood formation—something wrong in the building up, *not an excessive breaking down.* The theories advanced to explain such a condition are numerous, and the following may be borne in mind for examination purposes—

1. Due to constipation. (Sir Andrew Clark.)

2. That the period of puberty causes an increased demand on the blood-forming glands, which are unable to meet the call.

3. The demands of puberty reveals a congenital narrowness of the aorta, which prevents a proper supply of blood to the various tissues.

4. *That the organic iron-compounds of the food are broken up by excessive decomposition in the alimentary canal, and the iron thus liberated, is rendered incapable of being absorbed into the blood.* (Bunge.)

5. The development in the female, of a peculiar gum-like substance intended for the nourishment of the future embryo, (*i.e.*, in preparation of the demands of pregnancy), which acts injuriously on the hæmoglobin molecule. (Landwehr).

It is painfully interesting to note that neither of the above explain the disease, for constipated subjects are not always chlorotic, chlorosis is not confined entirely to women, and chlorosis may be present with a sound aorta. At present we may assume that chlorosis is due to many factors,—the chief being the *adolescent period*, which causes an increased demand on the blood-forming glands, and develops latent weaknesses;

further, such period conduces towards an unstable state of the nervous system, manifested by dyspepsia, constipation, etc.

As to the *immediate* cause, it is highly probable that Bunge's theory is correct.

Treatment.—Is highly satisfactory. Fresh air, exercise, nitrogenous food, purgatives, and iron will cure the worst cases.

An excellent combination is iron, aloes, and carbonate of potash. The iron acts in two ways—

1. It combines with many of the products of decomposition in the duodenum, and thereby forms insoluble and inert salts, which are excreted, thus preventing the pernicious gases, etc., entering the portal system.

2. No doubt a small amount of the iron itself *is absorbed and assimilated.*

Carbohydrates as a rule are harmful. Chlorotic girls should be released from all studies, and exciting literature should be kept from them.

PERNICIOUS ANÆMIA.

This is a progressive and profound anæmia, developing without any evident cause, and ending most frequently in death.

Ætiology.—Most frequent in the Swiss Cantons. Affects males more than females. Is rare under 25 years of age. Many cases have started during pregnancy.

Pathology.—*The Blood* shows a remarkable diminution of red corpuscles; they may sink to less than half a million per cubic millimeter. The amount of hæmoglobin in each corpuscle, so far from being diminished is often relatively increased. The corpuscles exhibit a number of various shapes, some are tailed, others crenated; often the hæmoglobin can be

seen protruding prior to its escape from the stroma, some discs are extremely minute, and may be nucleated. A large number of yellowish bodies termed microcytes are usually found. *The blood when shed coagulates with difficulty.*

The Liver shows marked changes—

1. Is exceedingly rich in iron.
2. There is an excess of pigment within the liver cells.
3. There is fatty degeneration in the central third of each lobule.

Neither *the marrow* nor *the spleen* show any constant or definite changes, beyond those that result from deficient oxygenation.

There can be no doubt that pernicious anæmia is due to *excessive destruction of the cellular elements of the blood.* Dr Hunter, of Cambridge, after a most exhaustive and critical analysis of this disease, believes that pernicious anæmia is due to the circulation of poisonous cadaveric alkaloids within the *portal* circulation, which hasten the disintegration of the red corpuscles. Possibly the disease is due to a *special* organism rather than cadaveric products.

Prognosis.—Highly unfavourable, most cases die in from six to fifteen months.

Symptoms.—Are those of profound anæmia already described, but in addition we have in pernicious anæmia—

1. Fever.—The pyrexia is usually paroxysmal.
2. Indican in the urine.
3. Hæmoglobinuria.
4. Retinal hæmorrhages.
5. Primrose colour of the complexion.

Though of course the muscular wasting is considerable, the subcutaneous fat is often increased; hence the wasting is not apparent, and the patient may present a fairly well nourished appearance as regards the general contour of the body.

Treatment.—Is far from satisfactory. Arsenic, transfusion of blood, β-napthol, etc., have in certain cases proved successful, but the bulk of the cases are quite unamenable to any treatment yet discovered.

There cannot be the slightest possible doubt that an aseptic state of the intestines as far as possible is highly desirable, and salol and β-napthol are certainly our best drugs for obtaining such a state.

Iron seemingly is of no use.

LEUCOCYTHÆMIA.

Leucocythæmia is an affection characterised by persistent increase of white corpuscles, hæmorrhages, and changes in the blood, marrow, spleen, lymphatic glands, etc.

Ætiology.—The disease may develop at any age, but by far the bulk of cases occur in the middle aged. Men are more often attacked than women. The more important ætiological factors that have been observed are—

Heredity,
Syphilis,
Malaria,
Traumatisms, and
Pregnancy.

Pathology.—*The blood* is markedly altered, and shows—

1. The white corpuscles to be enormously increased, so that the proportion of white to red corpuscles may be as high as 1 to 6, or even 1 to 3, instead of 1 to 350 as in normal blood. The white corpuscles are of different shapes and sizes, and hence an attempt has been made to distinguish various types of the disease:—*i.e.*, The small *non*-nucleated cells are

held to be derived from the marrow, the small *nucleated* cells from the lymphatic glands, large coarsely-granular corpuscles of various shapes from the spleen. Thus the preponderance of any particular shape points to the structure mostly involved.

2. An excess of fat, so that a drop of the leukæmic blood dropped on to white paper leaves a greasy stain.

3. The presence of elongated, octahedral, colourless, phosphatic crystals (similar to those found in asthma).

4. The abundance of hypo-zanthin and other antecedents of urea.

5. A substance which is soluble in hot water, and which like gelatine sets into a jelly when its solution is cooled.

The Spleen.—It is usually firm, much enlarged and may be bound by adhesions to the abdominal wall, the diaphragm, or the stomach. The capsule is much thickened. On section, the organ presents a pinkish appearance, appears fatty, and feels greasy to the touch. The pulp is dark, and the Malpighian tufts stand out as pale spots. The sinuses are often distended with leucocytes. Hæmorrhagic infarctions, old and recent, are very common. Later, the changes are more fibrous in character, and the splenic pulp may be much atrophied from the pressure.

The Lymphatic Glands.—Nearly all the lymphatic glands are enlarged and softened; later, they may assume the same characters as observed in Hodgkin's disease.

The Marrow.—The marrow may be dark brown in colour, or present a peculiar yellowish-red appearance. Large numbers of nucleated red corpuscles and non-nucleated leucocytes are found.

The other tissues and organs, such as the liver, heart, etc., show those grave changes consequent upon deficient oxygenation, *plus* extravasations of blood.

Symptoms.—The onset is most insidious, and the patient usually applies for medical advice on account of breathlessness, dyspeptic symptoms, palpitation, and other symptoms of anæmia. Hæmorrhages of various kinds may be the first symptoms. When the disease has advanced, the condition of the patient is most characteristic. The abdomen is prominent; the countenance of a deadly white, or with a slightly sallow tint; the sclerotic of a pearly lustre; pupils dilated, mucous membranes blanched. The pulse is soft and compressible, contrasting well with the high tension pulse of chronic Bright's disease. Hæmorrhages are common under the skin in mucous membranes, and into the retina. The retinal changes are often most marked, the vessels being tortuous and often distended with the white corpuscles. Towards the end, grave waxy changes set in and intermittent diarrhœa, *attacks of fever* and œdema, or general dropsy ushers in a fatal issue. It must not be forgotten that the urine usually is of high specific gravity, containing excess of uric acid and urea. The pallor, usually so well marked, is sometimes wanting. The special points to note are the condition of the blood, marked exhaustion, pyrexia, and the tendency to dropsy associated with highly coloured urine.

Diagnosis of the enlarged Spleen.—It enlarges in the axis of the tenth rib, and, therefore, tends to go forward to the right iliac fossa. If much enlarged, it is found to present as a firm, hard tumour, having a distinct notch in its anterior border; it may reach from the flank to or below the umbilicus, and weigh from six to eighteen pounds. Note in contrast to floating kidney—the colon never lies in front of an enlarged spleen.

Treatment.—Everything must be done to protect the patient from cold, and the general hygiene must be as perfect as possible. Massage and galvanism may be applied with advantage to the spleen. The medicinal treatment is as described under the treatment of the Pernicious Anæmia.

Bone marrow + Spleen

HODGKIN'S DISEASE.

It is an affection characterised by progressive enlargement of the lymphatic glands, destruction of red corpuscles, and secondary lymphoid growths in the various organs.

Ætiology.—It may attack any age, but is by far more common in children and young adults. No special ætiological factor is known. Possibly syphilis, tubercle, and traumatisms are important factors, in as much as they weaken the glandular system.

Pathology.—The exact cause of Hodgkin's disease is quite unknown, though there can be no doubt that it is due to some chemical irritant formed either in the blood, or introduced from without, but which, when in the blood, causes an overgrowth of lymphoid tissue.

The affected glands are usually much enlarged, single, painless, and non-adherent to the skin. Though they may feel to the touch hard and solid, they are far more frequently somewhat soft and elastic. *Their two great characteristics are—they neither tend to suppurate or caseate.* Of course, like all other growths, they may under certain circumstances suppurate and mass together; but, as Dr Bramwell emphasizes, "such results are to be regarded as accidental." The disease may cause large deposits of lymphoid tissue in the subcutaneous tissue and marrow of long bones.

Histologically.—

The Glands.—The chief change is a general hyperplasia of the whole gland, accompanied by the formation of a large number of small cells resembling leucocytes. The amount of new fibrous tissue formed governs the degree of hardness: if the lymphoid cells are abundant, the gland is soft; but when the fibrous tissue element is in excess the glands are hard.

The Spleen.—Is always enlarged in Hodgkin's disease, but not nearly to the same extent as in leucocythæmia. In consistence it is firm, and purple patches from venous congestion often appear on the surface. On section can be seen golden-yellowish masses, consisting of lymph corpuscles and pigment enclosed in a fibrous reticulum. The Malpighian bodies appear as translucent yellowish suet-like masses. The general fibrous stroma is much increased like in the lymphatic glands.

Symptoms.—Briefly are—

1. Enlargement of the lymphatic glands.
2. Anæmia—Often marked decrease of red corpuscles, and slight increase of the white.
3. Symptoms pointing to the malignant nature of the disease—*i.e.* marked emaciation, cachexia, secondary deposits of lymphoid tissue.
4. Attacks of pyrexia.
5. Symptoms due to pressure from the lymphoid growth.

The glandular enlargement most frequently begins in the anterior chain of glands at the posterior border of the sterno-mastoid, then the axillary, inguinal, and finally all the glands of the body may show extensive changes. Often both sides of the neck become involved, and the growths meeting anteriorly in the middle line may compress the trachea to a dangerous extent. Osler points out that when the abdominal glands are affected the sympathetic system is often profoundly disturbed and pigmentation of the skin may occur. The enlarged mediastinal growth may cause severe dyspnœa, inequality of the pupil, pleurisy, etc.

The periodical elevations of temperature may be so marked as to simulate ague. It will be at once seen, that almost any symptom may crop up in Hodgkin's disease, and no wonder if we remember the extensive nature of the disease, and the important structures that may be involved thereby. Lastly note, that in some cases the red corpuscles are not diminished until marked cachexia has set in.

Treatment.—It has been suggested that when the disease has affected only one set of glands, that the diseased portion should be excised: such procedure is certainly as rational as operative measures in other malignant conditions. We may hope for benefit from subcutaneous injections of arsenic, or thyroid extract, combined with massage and careful hygiene. The disease is, however, only too frequently fatal in from two to four years.

DISEASES OF THE STOMACH.

ULCER OF THE STOMACH.

By this lesion is meant a form of ulcer, which is peculiar to the stomach, the first part of the duodenum, and (according to some authorities) the lower end of the œsophagus. Though usually single, there may be more than one ulcer present.

Site.—Solitary ulcers are nearly always situated on the posterior wall, near to, or involving the lesser curvature. Sometimes they are observed near the pylorus, but rarely do they attack the greater curvature.

Characters.—In size the ulcer varies much, but is usually about an inch or slightly less in diameter, and of a circular or oval shape. Its appearance suggests that it might have been produced by punching out a piece of mucous membrane. The edges are *not* undermined, and there is an absence of vascularity in the margins and base. The floor is formed by either the submucosa, by the muscular coat, or the serous coat; the latter is not unfrequently thickened, and adherent to the neighbouring organs. These adhesions are of the utmost importance, and go far to explain why perforation is

not more common in cases of gastric ulcer. As the ulcer deepens, its floor or base becomes narrower, so that the walls come to have a terraced appearance, and an oblique direction. If perforation occurs, the aperture in the serous coat is of small size, but presents the same clean cut punched-out appearance, that distinguishes the margins. In the healing of the ulcer, if the mucosa be alone involved, the granulation tissue develops from the edges and the floor, and the newly formed tissue gradually contracts and unites the margins, leaving a smooth scar.

In larger ulcers which have become deep, and involved the muscular coat, the cicatricial contraction may cause serious changes, the most important of which is, narrowing of the pyloric orifice, and dilatation of the stomach (Dr Osler). A common result of the ulcerative process is perforation of the gastric walls, and consequent fatal peritonitis. On the other hand, it is not uncommon for adhesions to form between the walls of the stomach and neighbouring organs, and the ulcer after perforating the gastric walls, burrows into the pancreas, spleen, or liver. Large vessels may be eaten into in a similar manner, and bring about a fatal issue from hæmorrhage. Other complications through the ulcerating process are—

1. Perforation into the pleura.
2. Gastro-duodenal fistula.
3. Perforation into the lesser peritoneum, constituting the so-called subphrenic-pyopneumothorax.

Pathology.—Nearly all observers agree that the ulcer is due to two immediate factors—

1. A devitalisation of certain areas of the gastric mucous membrane.
2. Erosion of these areas through either the digestive action of the gastric juice, or the mechanical irritation of indigestible food.

Probably in all cases many factors are present, and I think the best way of summing up is as follows: deficient or indifferent quality of blood supply to the walls of the stomach tend to cause—

1. Alterations in its secretions.
2. Local thrombosis of the nutrient arteries.
3. Deficient movement or peristalsis of the viscus.

Thus we may get hyperacidity of the gastric juice; certain areas of the stomach are rendered very vulnerable through the local thrombosis, and lastly the defective peristalsis favours the concentration of the irritating gastric contents at certain points. Consequently there can be no difficulty in understanding how ulceration of the mucous membrane comes about in the thrombosed areas. If we grant this, then we must allow that such a downgrade process must be powerfully assisted by irritating food, and anything which tends to diminish the alkalinity of the blood, for it must be remembered that physiologists hold that the alkalinity of the blood normally plays a part in preventing the stomach from digesting itself.

Bucquoy emphasizes the following symptoms as diagnostic of the duodenal ulcer—

1. Sudden intestinal hæmorrhage in an apparently healthy person, which tends to recur.
2. Severe pain in the right hypochondrium, two or three hours after a meal.
3. Local tenderness.
4. Severe gastralgia.

Treatment.—What are our indications in such a case? Obviously (1) Improve the general hygienic surroundings; (2) Give the inflamed stomach rest; (3) Promote the healing of the ulcer; (4) Improve the blood condition; (5) After the acute symptoms are over, improve the local supply of blood by gentle massage to the stomach.

Diet.—A wine-glassful of milk, with a little lime water added, every two hours, must be the only food allowed by mouth. It may, however, be supplemented in some cases by nutrient enemata of pancreatised beef extract.

Medicinal.—The favourite remedies are bismuth (powder) with morphia, and the administration of Carlsbad salts. The former act mechanically by coating the ulcer, the latter, by depleting the portal circulation, removing the constipation, and adding to the alkalinity of the blood.

Ætiology.—Gastric ulcer is most commonly met with in young women, especially those who lead a sedentary life, who live in areas shut out from the sunlight, etc. Tight-fitting corsets are undoubtedly important factors. In men—shoemakers are prone to this affection, due probably to pressure of the "last" on the stomach, thereby impeding or hampering the gastric circulation. Extensive burns may be followed by gastric ulcer, especially of the duodenal type.

Symptoms.—No disease or condition may have on the one hand more characteristic symptoms, and on the other hand more ill-defined ones than gastric ulcer. Taking a typical case, we may expect—

1. Pain and tenderness over the epigastric region. The pain is severe and shoots through to the back (xiphi-vertebral), is rendered worse by eating, or by firm pressure.

2. Vomiting; the food usually returns quickly, and often gives much temporary relief.

3. Hæmatemesis may be very copious, but is not frequent.

These symptoms in combination with the history, anæmia, and the absence of a tumour, point strongly to the presence of an ulcer. I must, however, warn beginners not to always expect such typical signs. Often, indeed, the symptoms are

very slight, and a copious or even fatal hæmorrhage may be the first indication of such a condition.

Note.—When the healing process has fairly advanced, the patient may have bland broths, raw meat sandwiches, whipped cream and eggs, etc. Pepsin is often of great use. The great drawback to successful treatment is the lack of co-operation on the part of the patient as regards diet.

CANCER OF THE STOMACH.

Cancer of the stomach is by no means a rare condition, and most varieties have been found at one time or other; the principal primary forms however are—

1. Malignant adenoma at the pyloric and the cardiac end.
2. Schirrus cancer; this form constitutes the bulk of the pyloric growths.
3. Encephaloid.
4. Colloid.

3 and 4 are in all probability degenerations of the other two. Epithelioma at the cardiac orifice is probably an œsophageal cancer which has extended.

Ætiology.—The majority of cases occur between the ages of forty and sixty years. Rich and poor, male and female, seem to furnish an equal number of cases.

Pathology.—As the pylorus is the most common site, and hard schirrus the most common variety of cancer in that region, we shall discuss its growth briefly.

The cancer usually begins as an overgrowth of the epithelial cells lining the gastric glands; the new growths infiltrate the

submucous tissue, proliferate rapidly, and finally all coats are invaded resulting in the formation of hard nodular masses. The pyloric opening becomes much narrowed, and the resulting stenosis gives rise to the marked dilatation of the stomach, so characteristic of pyloric cancer. The subsequent pathological course of the cancer does not differ from that of cancer elsewhere. It may remain more or less stationary for a time, then undergo one of the various degenerations that morbid growths are liable to—*i.e.*, colloid degeneration, ulceration, etc. If we get a clear idea of these changes, remember the anatomical site, and the physiological importance of the neighbouring viscera, there cannot be any doubt as to what symptom may or should crop up. The ulcerative process may invade important blood-vessels, cause hæmatemesis, or form adhesions with other organs, and seriously handicap their functions by actual invasion, and lastly aggravate or hasten the malignant cachexia.

Symptoms.—They are for a time most insidious, but sooner or later the persistent vomiting, hæmatemesis, constant pain, emaciation, and cachexia, with the presence of a local tumour, declares the true condition. The symptoms are considered in detail in the diagnostic table appended.

Just a few words about hydrochloric acid being absent in this condition. I have examined the vomited matter of many cases of gastric cancer, and have found hydrochloric acid in some cases, and none in others. As HCl may be absent in other conditions than gastric cancer, I cannot help thinking that its absence as a diagnostic agent in cancer had better be dismissed. Have we a reliable test for hydrochloric acid? Moreover, as the absence of HCl is not noted until the disease *is advanced*, one fails to see its great importance.

Prognosis.—The disease is usually fatal in from six months to two years, but severe complications may bring about a fatal issue very speedily.

Physical Examination.—A tumour may be discovered three or four months after the onset of symptoms. It is found anywhere within an area formed by joining the ensiform cartilage with the umbilicus, and the umbilicus with the ninth costal cartilage. The tumour is painful on handling, may receive an impulse from the abdominal aorta, and though at first is somewhat mobile, it afterwards becomes fixed.

Physical examination also reveals the dilated condition of the stomach. It should however be remembered, when the body of the stomach is involved, that the stomach is atrophied and hour-glass shaped instead of being dilated.

Diagnosis.—See Table appended.

Treatment.—Must be mainly palliative. Digestible food and hypodermic injections of morphia help to make the patient's life tolerable. Surgical proceedures seem to offer some chance of prolonging life. Dr Mayo Robson says:—when the disease involves the pylorus there are three courses open—

1. Complete removal by pylorectomy with suture of the ends of the stomach and duodenum.

2. Pylorectomy with closure of the open ends of the duodenum and stomach by invagination and suture, the communication between the stomach and bowel being that effected by gastro-enterostomy.

3. In extensive disease, or where the patient is unable or unfit to bear the major operation, gastro-enterostomy may be performed, the stomach and jejunum being fixed in apposition by means of Senn's plates.

[TABLE

CANCER.	ULCER OF STOMACH.
1. Is rare under forty years of age.	1. Usually in young adults especially women.
2. Epigastric pain is more or less continuous, and not much relieved by vomiting.	2. The pain is rendered worse by food, and vomiting gives much relief. The pain is also more localised and xiphi-vertebral in character.
3. Vomiting is not frequent, but is copious; and in the vomited matter may be found—Sarcinæ ventriculi, torula, etc. Cancerous fragments (rare). HCl is absent (?)	3. Vomiting is frequent, and the vomited matter contains HCl.
4. Hæmorrhage is seldom copious, but may be frequent; most common in the later stages.	4. Hæmorrhage is not so frequent, but is copious at times. May be the first symptom.
5. Loss of flesh; the development of cachexia is rapid.	5. When the gastric symptoms are severe and prolonged, a cachectic appearance may develop, but never to the same extent as cancer.
6. Epigastric tumour is usually easily detected.	6. No epigastric tumour, though there may be some thickening of the walls.

EXAMINATION OF THE STOMACH.

In order to have a clear idea of the various disorders this viscus is subjected to, it will be necessary to recall the main facts relative to the digestive processes that go on in the stomach. We know that starchy foods become converted into maltose, with an intermediate stage of dextrines; proteids into peptones, with an intermediate stage of albumoses; and that fats are also acted upon to a slight extent, inasmuch as the proteid envelopes of the fat cells are dissolved. We can easily understand then, that such digestive processes can be retarded by—

1. Putting into the stomach a larger amount of food than it is capable of digesting.

2. Deficiency either in quantity or quality of gastric secretion.
3. Deficient movements of the stomach, through muscular atony.

It is highly necessary then, to know the exact state of secretion, and the amount of motor or peristaltic power.

During the process of digestion, the stomach contents at various intervals give three distinct reactions—

1. Immediately after food is taken it is faintly alkaline from the saliva swallowed.
2. The next half-hour it reaches the neutral stage.
3. After this time it gradually becomes acid from the presence of HCl.

The first acid to appear as a rule is lactic acid, and it must be remembered that this acid is a perfectly normal constituent during the first half-hour or so of digestion; after *an hour there should only be the faintest traces.* It must not be forgotten also, that HCl is hostile to the action of bacteria; consequently, if lactic acid is found in excess two hours after a standard meal, we can at once conclude that HCl is deficient.

When it is desired to ascertain whether gastric digestion is normal, a test meal is first given; then a portion of the gastric contents are pumped out at various intervals through an elastic œsophageal tube, and chemically examined.

Unfortunately we have no reliable test for free HCl, *but if we assume that HCl is the only mineral acid in the stomach,* then the aniline dye, Congo-red, forms a splendid test.

Method.—Take the filtered gastric contents, add to it a solution of Congo-red, the reagent immediately turns blue. Wash in æther, and if the red colour be restored, the acid is *organic;* if not, *the acid is mineral,* and therefore HCl is present, as we assume it to be the only mineral acid present in the stomach.

Uffleman's Reagent is a most convenient one for general practice. The reagent is made by colouring (violet) a one per cent. solution of carbolic acid, by the addition of tinct. ferri perchlor.

METHOD.—Take filtered gastric contents, add an equal quantity of the reagent.

If HCl. is normal—colour is discharged.

If lactic acid be present—canary yellow.

If butyric acid be present—dirty grey.

An alcoholic solution of vanillin and phloro-glucin is also a good test for mineral acids, for if it be added to a sample of the gastric contents, and then evaporated, a brown-pink residue is left, if a *mineral acid be present.*

Tests for Peptone—

Characters.—

1. Like all proteids it is—
   (1) Insoluble in strong alcohol.
   (2) Gives xanthoproteic reaction.
   (3) Gives a pink colour reaction with copper sulphate.
2. Unlike all other proteids peptone is diffusible.
3. Heat does not coagulate peptone (*unlike albumin*).
4. Nitric acid does not precipitate peptone (*unlike albumin*).
5. Pure neutral ammonium sulphate does not precipitate peptone, while it precipitates albumin, globulin, and albumose.

Albumose or Propeptone is intermediate between albumin and peptone. Gives the typical reactions of proteids. Like albumin it is non-diffusible. Like albumin it is precipitated by nitric acid in the cold, but the precipitate *dissolves on heating and returns on cooling.* Neutral ammon. sulphate precipitates it as it does albumin.

Rennin is the milk-curdling ferment of the gastric juice. Pancreatic juice contains a similar ferment.

The curdling of milk is due to the coagulation of the alkali-albumin casein. Any acid will curdle milk, but rennin does something more than mere curdling—it renders the curd more digestible. It is a remarkable fact that rennin fails to "curdle" milk if lime salts are not present.

Presence of Pepsin.—The presence of pepsin can be inferred by the following process:—Take a filtered solution of the gastric contents, add to it 0·2 per cent. of HCl; place in this solution boiled fibrin—the fibrin will be dissolved as in normal gastric juice.

Vomited Matter may be tested, instead of drawing off the contents of the stomach by the œsophageal bougie, but the results are likely in such cases to lead to erroneous opinions.

Power of Absorption can be tested (so it is said) by observing the rapidity with which KI (which has been swallowed in a gelatine capsule) appears in the saliva.

Motor Power of the Stomach is estimated by testing with ferric chloride for salicyluric acid in the urine at various intervals after a certain quantity of salol has been taken by the mouth. Salol should not be decomposed by the stomach contents which are acid, but only when it meets with an alkaline fluid—*i.e.*, in the small intestines. If the urinary reaction (a violet colour) is delayed beyond half-an-hour, the motor power of the stomach is held to be deficient.

I have placed the foregoing tests, etc., before the reader, because the student is *expected to know such statements*, and the practitioner will find them useful in case of a consultation; but the writer has the courage to assert, that we must admit these tests are of more theoretical than practical importance if we duly consider their many fallacies, and the difficulty of carrying them out in routine practice.

Alkalies are said to increase the secretion of HCl if given before food, but the use of alkalies should not be continued for an indefinite time as is only too often done.

DYSPEPSIA.

It is difficult to draw a satisfactory distinction between gastritis and dyspepsia, for dyspepsia is often symptomatic of gastritis. If we were obliged to draw a hard and fast line of distinction, we might say — Gastritis means a diseased stomach—*i.e.*, one in which pathological changes have taken place; dyspepsia, on the other hand, means a stomach "on strike," a functional disturbance in fact, which may or may not be highly amenable to treatment.

The following Table may assist the reader in diagnosing the two conditions :—

	CHRONIC GASTRITIS.	DYSPEPSIA.
Pain	Often severe with local tenderness.	Less severe; tenderness is usually absent, or if present not localised.
Fever	Temperature is often raised.	Not raised.
Thirst	Often a marked symptom.	Absent.
Vomiting	Frequent, especially in the morning. Lactic, butyric, and acetic acids often present. Pain is not usually relieved by vomiting.	Vomiting is not frequent except after certain foods, then relief is obtained.
Causes	Usually the constant introduction of irritants, such as alcohol in excess, abuse of tea, morphia, etc.	See list of causes tabulated. Often there is no obvious cause and the best dietetic treatment often fails to cure.
Tongue, etc.	Is furred, red at the lip and edges. The lips are cracked, and the gums spongy and red.	Tongue broad, flabby, and indented by the teeth. Gums are soft and anæmic. Lips are not usually fissured.
Morbid Anatomy.	Stomach is much thickened, the mucous membrane is often much atrophied, and fibrous in structure. It presents a rough mammillated appearance with suppurating points, localised vascular areas, and hæmorrhagic erosions. *Note.*—Though the membrane is thickened, the essential glandular character is atrophied.	In pure dyspepsia these changes are not present. The mucous membrane may be thickened and injected. The muscular fibres are pale, flabby, and relaxed.

Causes of Dyspepsia.—

Faults on the part of the organs—

1. Bad teeth.
2. General debility of the digestive organs after fevers, etc.
3. Deficiency in the quantity or quality of the gastric juice, pancreatic secretion, and bile.

Faults on the part of the patient—

1. Habitual use of the *same kind* of food.
2. Intemperance in eating and drinking.
3. Excessive use of tobacco, especially if attended with excessive expectoration of saliva.
4. Bolting food.
5. *Cold* drinks during meals.
6. Mental work immediately after eating.
7. "Dirty" teeth.
8. *Deficiency of food.*
9. Excessive use of tea, coffee, alcohol, etc.
10. *Sound* sleep after dinner.

By the above table it will at once be seen that it is most difficult to enumerate all the causes of indigestion, but it should be remembered that many persons may eat with impunity what would cause much distress in others.

The more common symptoms are pyrosis, flatulence, eructations of acrid matters, disagreeable breath, vertigo, etc.

Many forms of dyspepsia are described, and I append a table showing the principal points of the three more distinct forms; at the same time it must be remembered the table is only a guide, for the types usually overlap each other.

[Table

	ATONIC DYSPEPSIA.	BILIOUS DYSPEPSIA.	NERVOUS DYSPEPSIA.
Immediate Cause....	Want of functional power, both as regards gastric secretion and movements.	Constitutional "lithæmia," vitiated bile, hepatic congestion.	Mental strain from worry, over-study, always being in a hurry, etc.
Pain, etc.	Some hours after food; vomiting not common.	Pain not marked, but nausea and vomiting often intense.	Pain is severe, often relieved temporarily by eating, but pain recurs in two hours or so.
Eructations	CO_2 principally, but may be acid.	H_2S, Butyric acid, CO_2, etc.	CO_2 — generally odourless.
Urine	Normal or high-coloured from urates.	High-coloured deposits, "gravel," and oxalates.	Pale deposits, amorphous phosphates.
Testing Gastric Contents shows..	Deficiency of HCl. Excess of lactic acid.	Lactic or butyric acids in excess. Bile salts and pigment may be present.	HCl is sometimes in excess, but frequently the secretion is normal.
Tongue	Broad, flabby, papillæ raised, furred at the back, and tremulous.	Broad also, but usually covered with a thick yellowish fur.	Is usually clean, raw beef-like in character, pointed tip, firm not flabby.
Special points	Most common amongst young women. Apt to persist.	Most common amongst middle-aged people of generous build. Paroxysmal in character, migraine and mental depression marked during the attack.	Most common amongst literary workers, speculative, thin, wiry people. Mental irritability; desire for air; arteries are tense; localised hyperæsthetic points; sleeplessness.

Treatment. — Stock prescriptions and rigid diet charts account for the failures in treating dyspepsia. Dyspepsia is not a disease, it is symptomatic of an altered digestive tract, and it is our first duty to find out the most probable cause, then, having fully considered the constitution of the patient, advise careful dieting, fresh air, exercise, etc. It is at all times our duty to suggest the avoidance of those things we know are indigestible, but he is a conceited man who thinks he knows better than the patient what best agrees with him. What ought to agree is often the thing that does *not*. If the cause be obscure, and the treatment slow in producing benefit, a careful examination of the gastric secretion after a standard meal should be made. The main points to remember are—

1. First allay any undue irritability of the stomach by a restricted amount of food, and the administration of hydrocyanic acid, bismuth and morphine in an effervescing form.

2. Promote gastric secretion by giving alkalies before meals; or imitate the secretion by giving pepsin and hydrochloric acid after meals.

3. Promote healthy peristalsis by giving muscular tonics such as acid nitro-hydrochloric with liquor strychnine.

4. Keep the bowels open.

5. Avoid turning the patient's stomach into a chemist's shop.

6. If there be much fermentation and evidence of the collection of ropy mucus in the stomach, wash it out and give a pill containing a minim or two of carbolic acid twice a day for a short period.

Remember that a healthy co-operation on the part of the patient is necessary, and this can only be obtained by showing him that we thoroughly understand what is wrong. When the patient is highly neurotic, a blister over the epigastrium is of great value.

ACUTE GASTRITIS.

Acute inflammation of the stomach is due to the severe irritation set up by the ingestion of unripe fruits, decomposed tinned meats, shell-fish, etc. It is frequently a complication of the malignant fevers, low inflammation, etc.

Pathology.—The mucous membranes show the usual hyperæmia, exudation, and increased mucus secretion observed when mucous membranes are inflamed. The various epithelial cells of the numerous glands may become highly granular, undergo mucoid degeneration or desquamate. There may be minute extravasations of blood, hæmorrhagic erosions, pustules or aphthous patches.

The submucous coat is infiltrated and the whole stomach wall may be congested.

Treatment—

If the irritating food is still in the stomach give a brisk emetic, followed by gastric sedatives.

Ice, an effervescing mixture containing morphia, etc., will be found very grateful to the patient.

The stomach should have perfect rest for at least twenty-four hours. If it is desired to increase that rest nutrient enemata may be given.

DILATATION OF THE STOMACH.

The stomach may be dilated through atony of its walls, consequent on constant distension by excess of food, but it is much more frequently an accompaniment of pyloric obstruction as in cancer, etc., or of chronic gastritis.

Diagnosis.—1. By the combined method of percussion and auscultation, as suggested by Dr R. A. Fleming. He writes "In the cases tested I used both the finger and also an ivory pleximeter, and percussed with one finger. The stomach note could be at once detected by the auscultating ear, whenever the stroke was made over it, *even though the colon* overlapped."

The dilation is apt to assume a more or less U-shape, since the œsophagus and pylorus are more or less fixed points.

Other methods recommended are distending the stomach with carbonic acid gas, water, etc.

The vomited matter, when vomiting occurs, is said to be characteristic: it is large in quantity and contains sarcinæ ventriculi, yeast, and other ferments.

HÆMATEMESIS.

Vomiting of blood may be an important symptom in many diseases.

The source of the hæmorrhage may be either congested and distended venules or capillaries in the gastric mucous

membrane, or through the bursting of a large vessel; for example, the splenic artery in gastric ulcer.

Causes.—The more common are—

1. Erosion of vessels by gastric ulcer, cancer, chronic gastritis.

2. Congestion of the portal circulation from any cause, but especially cirrhosis of the liver, extreme backward pressure from cardiac disease, cancer and other growths in the liver.

3. The action of irritant poisons.

4. Alterations in the blood, and blood-vessels by which blood oozes through, as seen in malignant fevers, purpura, severe jaundice, etc.

Symptoms.—Of course depend on the extent of the hæmorrhage; when it is severe the symptoms of collapse are marked. The attack is usually sudden, and accompanied by a feeling of intense nausea and a feeling of weight in the stomach.

Diagnosis.—When the hæmorrhage is not profuse it is sometimes difficult to distinguish this condition from bleeding from the lungs.

I append a table showing the chief differences :—

HÆMOPTYSIS.	HÆMATEMESIS.
1. Previous history of pulmonary troubles.	1. Previous history of gastric disturbance.
2. Blood is coughed up.	2. Blood vomited.
3. Blood is frothy and bright red.	3. Blood is dark-coloured and not frothy.
4. Blood may be mixed with sputa.	4. Blood may be mixed with food.
5. Dyspnœa and pains in the chest.	5. Nausea, and weight in epigastrium.
6. Is not usually succeeded by malæna.	6. Often followed by malæna.

Whilst the above differences are of the utmost value, they are not always conclusive. For instance, in cases of

hæmoptysis the blood is frequently *not* frothy, or an amount of the pulmonary blood may be swallowed and *vomited afterwards.* Again, the hæmorrhage may be so severe, so sudden, and so inexplicable as regards its real cause, that a hasty judgment may be quite erroneous.

Treatment.— The first thing to be decided if possible is the source of the bleeding; and secondly, whether it is advisable to check the hæmorrhage, knowing that hæmatemesis is so often a safety valve in portal congestion. Having decided to check the hæmorrhage, absolute quiet, mentally and bodily, must be obtained, and no treatment is more efficacious than a full hypodermic injection of morphia, combined with the administration of ice and dilute sulphuric acid.

ATROPHY OF THE STOMACH

May occur in long continued exhausting diseases, cancer of the cardiac end of the stomach, bulbar paralysis, or malignant stricture in any part of the œsophagus.

DISEASES OF THE INTESTINES.

TYPHLITIS, PERITYPHLITIS, AND APPENDICITIS.

By typhlitis is meant, inflammation of the cæcum proper; by perityphilitis is meant a phlegmonous inflammation in the cellular tissue uniting the cæcum with the psoas and iliac muscles; and by appendicitis is meant inflammation of the appendix cæci.

It appears, however, that the two former conditions are merely an extension of appendicitis, and it is becoming more and more recognised that the terms, typhlitis and perityphlitis ought to be abolished and the term appendicitis substituted to include all three conditions. This may be scientific and even convenient, but I think such a course open to grave criticism. We shall first consider the anatomical peculiarities of this region.

1. The cæcum marks the junction of the small with the large intestine, the junction being guarded by the ileo-cæcal valve, which allows fæces, etc., to pass from the small to the larger gut, but at the same time prevents regurgitation. The ileum joins it on its inner aspect about two-and-a-half inches above its blind end.

2. The cæcum is not invested posteriorly by peritoneum, but by loose cellular tissue.

3. The close proximity of the appendix cæci.

The vermiform appendix is situated at the inner lower and back part of the cæcum, and usually looks towards the spleen. Unfortunately for clinical purposes, the exact position can scarcely be diagnosed during life, and *post mortem* it has been found in almost every region of the abdomen.

Solid bodies of all kinds may find their way into the appendix. Osler asserts that "foreign bodies rarely lodge in it, but that concretions of inspissated mucus and fæces in which lime salts are deposited forming enteroliths are common."

APPENDICITIS.

Usually occurs in young people.

Causes.—Foreign bodies, concretions, tubercular ulceration, malignant growths, constipation, habitual use of indigestible food, etc.

Pathology.—

Varieties.—1. The catarrhal form.

2. The ulcerative variety (apt to end in perforation).

In the catarrhal appendicitis the tube is thickened as a whole, the muscular walls are somewhat thickened or fibrosed, and the mucous coat is covered with a thick viscid mucus. Its serous coat may become adherent to the neighbouring peritoneum forming adhesive bands, which may cause a loop of gut to be strangulated or to kink over it.

The ulcerative variety shows in addition to the above changes, one or more ulcers; moreover, the ulcerative process is followed by most important results.

1. *Perforation may occur* AFTER *adhesions have formed*, and be followed by a circumscribed intra-peritoneal abscess. This localised abscess may after a time excite a most intense general peritonitis, and instead of bursting externally, open into the peritoneal cavity.

2. *When the appendix is* NOT WITHIN *the general peritoneum*, perforation produces a retro-peritoneal abscess; in such cases, the pus may burrow in the cellular tissue, between the gut and the iliac fascia, and may then extend in three directions—

(1) Upwards, forming a peri-nephritic abscess.

(2) Downwards to Poupart's ligament, where it is prevented from extending down the thigh, by the union of the ligament and fascia, and so bursts externally.

(3) Into the true pelvis, and then burst into the rectum, bladder, or through the obturator foramen.

3. *Perforation may occur* BEFORE *adhesions are formed*, the appendix hanging free in the peritoneal cavity. In such cases, a speedy death results from the violent septic peritonitis.

Symptoms.—In the *catarrhal variety*, the chief symptoms at first are localised pain in the right iliac fossa, local tenderness, elevation of temperature, furred tongue, and vomiting; later, if suppuration occurs, a tumour may be detected above Poupart's ligament, possibly occupying the whole of the right iliac fossa; it may be absolutely dull on percussion, or give a modified resonant note. The pain is often paroxysmal, and shoots down the right leg, which is flexed; the temperature becomes septic in character; constipation, is usually a marked feature, but there may be diarrhœa, painful micturition, etc. In the ulcerative form, when perforation occurs into the peritoneum, the symptoms are those of shock, or those described under general peritonitis.

Diagnosis.—The diagnosis is often very difficult, and the symptoms may simulate obstruction of the bowels very closely; but in appendicitis, the tumour is diffuse, and fæcal vomiting is rare, or according to some authorities never present. In children, the history, tenesmus, bloody discharge, and rectal examination under chloroform, makes a diagnosis of *intussusception* fairly easy, though it sometimes simulates appendicitis. Psoas abscess, and pelvic cellulitis in women are also conditions which must be eliminated before arriving at a diagnosis.

An attack of appendicitis after passing off, is extremely likely to recur under slight provocation.

Treatment.—Palliative, as detailed under general peritonitis. If the symptoms become alarming, surgical measures must be adopted.

INTESTINAL OBSTRUCTION

May be due to many causes, as the following Table taken from Dr Tanner's work shows.

1. *Inter-mural*, or those originating in, and implicating, the mucous and muscular coats of the intestinal walls.—

(1) Cancerous stricture,
(2) Non-cancerous stricture, comprising—(*a*) Contraction of cicatrices, following ulcerations. (*b*) Contraction of walls of intestine from inflammation, non-cancerous deposit and injury.
(3) Intussusception.
(4) Intussusception, associated with polypi.

2. *Extra-mural*, or those causes acting from without, or affecting the serous covering—

(1) Bands and adhesions from effusion or lymph.
(2) Twists or displacements.
(3) Diverticula.
(4) External tumours or abscesses.
(5) Mesocolic and mesenteric hernia.
(6) Diaphragmatic hernia.
(7) Omental hernia.
(8) Obturator hernia.

3. *Intra-mural*, or obstructions produced by the lodgment of foreign substances—

Foreign bodies, hardened fæces, concretions having for their nuclei, gall-stones, etc.

As the subject of intestinal obstruction really belongs to the domain of surgery, only the main clinical points will be condensed here.

The obstruction may be of an acute or chronic nature.

General Symptoms of any Complete Obstruction.—The more common are pain, distension of the abdomen, and stercoraceous or fæcal vomiting, with complete constipation.

The pain is variable, but is usually intense, at first paroxysmal, but later, continuous. It is not always referred to the part obstructed.

The constipation is absolute, but the bowel *below* the stricture may pass fæces, or the masses may be removed by an enema.

The vomited matter consists at first of the stomach contents, then bilious matter, and finally fæcal matter, which may in addition be tinged with blood.

The patient, if unrelieved, falls into a typhoid state from intense peritonitis, etc., and usually succumbs in from four to six days after the onset of the severe acute symptoms.

Special Forms of Obstruction.—

I. INTUSSUSCEPTION.

Occurs most frequently at the ilio-cæcal valve. Usually the ileum and cæcum preceded by the valve, pass into the larger colon. The valve forms the apex of the intussuscepted gut, and is an important aid to diagnosis on rectal examination.

Causes.—Severe and sudden peristalsis, especially in young children.

Special Symptoms.—A sausage-like tumour felt through the abdomen, glairy mucoid and bloody discharge, and tenesmus. Spontaneous reduction may take place, or the gangrenous invaginated portion may slough and pass per anum.

Treatment.—Inversion of the patient, gentle manipulation under chloroform, and copious injection of hot water into the rectum may be tried prior to laparotomy being performed.

II. VOLVULUS.

Is a twisting or bending of a coil of intestine in such a manner that its calibre is obliterated at that spot. Is most common in the sigmoid flexure, and towards the back of the abdominal cavity.

Special Symptoms.—Volvulus furnishes the most typical symptoms of acute obstruction.

Treatment.—If it can be diagnosed with a fair amount of precision, palliative measures may be tried for a longer period than in strangulated hernia, as the twist is very likely to recur after being replaced.

III. IMPACTION OF FÆCES, AND OTHER FOREIGN BODIES.

Treatment.—Try clearing out the lower bowel with copious enemata, breaking up hardened fæces, massage under chloroform, etc., prior to laparotomy.

It will be seen from the foregoing statements, that as the relief of intestinal obstruction usually resolves into surgical measures, the reader should look for details of these important conditions in a work on surgery. As, however, these cases frequently first come under the notice of the physician, and also as the patient and his or her friends frequently object to operative measures, the physician must take a determined stand or attitude; for he must remember every hour of delay in obtaining relief is fraught with extreme danger, and he should therefore lose no time in advising laparotomy the moment he is convinced that the obstruction is complete. We may, however, temporise in cases of fæcal accumulation, and in the more chronic forms of intestinal obstruction met with in old people, in malignant disease, etc., where surgical measures are little less than hopeless.

When we decide to temporise, we must put the patient under the most favourable circumstances we can—alleviate the pain by opium, the distressing vomiting by ice, and apply hot fomentations to the tender abdomen. No cases require graver thought than intestinal obstruction, and if there be the slightest chance of only temporary relief by surgical procedure, the patient should have his choice.

ENTERITIS.

It is somewhat difficult to define what is meant by enteritis, for the mucous membrane of the intestines is inflamed in very many diseases, especially in cholera, typhoid fever, dysentery, etc. But we may assume at present, that by enteritis is meant an inflammation of the alimentary tract, due to irritation by indigestible food, or a general catarrhal condition following exposure to cold, etc.

Like all other inflammations of mucous membranes, we may get the following varieties—

1. The ordinary, or catarrhal inflammation.

2. The croupous or fibrinous form, when the inflammatory exudation is unusually rich in fibrin, and tends to coagulate.

3. The phlegmonous type; when the sub-mucous coat is deeply involved, and presents a number of suppurative points which burst and form ulcers.

INTESTINAL CATARRH.

Acute Catarrh is most frequent in the hot summer months, possibly because of the ingestion of large quantities of unripe or decomposed fruit, sour milk, etc. We must, however, admit, that excessive decomposition in the intestines forms a favourable nidus for the multiplication of bacteria, which otherwise would lie dormant.

Pathology.—The more marked changes are observable *in the ileum* as hyperæmia of the valvulæ conniventes, a swollen condition of the solitary glands which project like small shots, and frequently present ulcerated surfaces. The mucous secretion is much increased, and a slight amount of pus may be formed.

Chronic Catarrh most frequently is due to alcoholism, portal congestion, atony of the gut from constitutional diseases, habitual constipation, etc.

Pathology.—The more marked changes are observable in the *large* gut. The mucous membrane is much thickened, often pigmented, and shows extensive small follicular ulcers, which after healing give a peculiar worm-eaten character to the gut on holding it up to the light. The superficial veins are distended, piles are often prominent in the rectum, and polypoid outgrowths may spring from the mucous surface.

Symptoms of Acute Enteritis. The chief symptoms are—

1. Diarrhœa.
2. Abdominal pain, especially around umbilicus.
3. Nausea, anorexia, and vomiting.
4. Pyrexia.

The above are the so-called cardinal symptoms, but it does not need a genius to at once grasp that the inflammatory condition, if severe, is likely to be complicated with peritonitis. Taking a typical case of enteritis, the diarrhœa is the chief symptom, and as in such cases excessive decomposition takes place in the intestines, we naturally expect the stools to be offensive, and to consist principally of undigested food, epithelial debris, triple phosphates, biliary pigments, and mucus. A word of caution is necessary, however, as regards bile pigments. The fæces may appear to contain an excessive amount of bile through the haste with which the duodenal contents are hurried on; or on the other hand, the same catarrh that caused the enteritis may block up the common bile duct and prevent the bile from entering the bowels, in which case the fæces will be pale and clay-like, and a subsequent jaundice may develop. Without a careful examination of the stools, it is impossible to diagnose whether the morbid process is confined to the duodenum, jejunum, or ileum.

Treatment.—First clear out any offending matter with castor oil. Relieve pain with an hypodermic injection of morphia, and the application of hot poultices or turpentine

stupes to the abdomen. An effervescing mixture containing hydrocyanic acid and bismuth is useful in allaying undue irritability of the stomach. Salol or β-napthol are our best intestinal disinfectants.

Symptoms of CHRONIC Enteritis.—

1. Hæmorrhoids.
2. Constipation, alternating with diarrhœa.
3. Discharges of blood and mucus from the anus.

The fæces do not present the same "undigested food" character as in acute enteritis, but there is more tenesmus or violent straining at stool. The fæces often resemble the chronic dysenteric stools—viz., boiled sago streaked with blood; but, unlike dysentery, the diarrhœa is easily provoked by hot liquids, and only too often by *any* meal; this shows that the irritable state of the gut is by no means localised to the large gut, though the more marked pathological changes occur in that part of the alimentary canal.

Treatment.—Remove any obvious cause. Salol in 15 grain doses nightly, with the addition of 3 minims of liq. arsenicalis and 10 minims of tinct. opii before each meal, occasionally act like a charm. If these measures do not succeed, treat according to the principles laid down under "Chronic Dysentery."

DIARRHŒA.

We have already seen that the frequent passage of loose motions from the bowels occur in many diseases, but whilst diarrhœa is often associated with grave lesions of the intestines, such as cancer, typhoid, tubercular ulcers, enteritis, etc., it may come on without any apparent cause, or after slight nervous disturbance, fright, etc.; in other words, diarrhœa may be merely a functional disorder, or symptomatic of a grave disease.

Causes.—Increased discharge of fæces can be induced by—

1. Increasing the intestinal secretions.

2. Exciting peristalsis, directly through irritants, or indirectly through the nervous system.

The more common causes independent of actual disease of the intestines are—eating indigestible food, unripe fruit, etc.; drinking cold liquids; certain nervous states, especially at the climacteric period. I once had a patient who had an attack of diarrhœa every Friday night after making his weekly visit to his bakehouses; the change from cold to heat was evidently the cause in this case.

Diagnosis.—It is not always easy to say whether the diarrhœa is a mere temporary disturbance, or the evidence of a disease, acute or otherwise. In all cases note—

1. How the looseness commenced.

2. The consistency, colour, and odour of the fæces. Look for blood, fat, calculi, etc.

3. Pain and abdominal tenderness.

4. The amount of pain at the time and immediately after defæcation.

Treatment.—Having satisfied ourselves that the diarrhœa is due to an irritant or irritating products still in the gut, clear out the bowels with a full dose of castor oil, then we may give opium in various forms, a full dose of the tincture, Dover's powder and bismuth, chalk mixture, etc.

Broadly speaking, *acute* diarrhœa is best treated with alkalies and opium; *chronic* diarrhœa with astringent minerals, or acids, with opium. Hot fomentations to the abdomen, diffusible stimulants if the patient feels chilly, and a restricted diet, are the main points to be remembered.

In that form of diarrhœa which comes on immediately *after* food in nervous people, nothing succeeds better than two

minims of liq. arsenicalis with five minims of laudanum, given just *before* meals.

Diarrhœa in young children or infants, if not associated with other diseases, such as rickets, tubercular conditions, etc., is nearly always due to improper feeding, or irritation from worms.

DISEASES OF THE PERITONEUM.

ASCITES.

Ascites or dropsy of the peritoneum is not a disease of itself, but merely symptomatic of some condition which causes an increased transudation of fluid into the peritoneum. Dropsy means some abnormal changes in the blood-vessel walls, either the result of inflammation, vaso-motor paralysis, or obstruction; and since we know that the venous blood of the intestines is returned *via* the portal circulation, we naturally look for the more common causes of ascites in morbid states of the portal vein; but it must be remembered that inflammatory changes of local blood-vessels in connection with morbid processes set up in the peritoneum and various abdominal organs, tend to bring about dropsy of the peritoneum. It is highly important to remember that the causes of ascites are *not confined to obstruction* of the portal vein, sometimes the dropsy is of local inflammatory origin, and the fluid is then frequently collected into localised pools—*i.e.*, within the mesh-work of inflammatory adhesions. The typical signs of ascites are often masked by the large deposits of inflammatory lymph between the intestines, etc.

Causes.—The more common causes of ascites tabulated are—

1. *Portal obstruction*, either within or outside the liver.

2. Phlebitis of the portal vein.

3. Disease (usually malignant or tubercular) of the peritoneum itself.

4. Morbid states of the blood associated with renal disease, leucocythæmia, etc.

It must be remembered that obstruction of a vein does not of itself produce dropsy *until some retrograde change is set up in the walls of the vessels.* Obstruction must of course produce such changes in time.

Under portal obstruction, we may mention cirrhosis of the liver, neoplasms of the liver, pancreas, spleen, etc.; malignant disease of, or growths in connection with the intestines, and other abdominal organs.

When ascites is caused by cardiac or pulmonary disease, probably it is due to two factors, viz., portal congestion, and a *deteriorated character of the blood.*

Diagnosis.—The cardinal symptoms are—

1. Enlargement of the abdomen: the skin is tense and shiny with dilated superficial veins on the surface.

2. Fluctuation, and vibration on palpation.

3. Percussion yields a dull note, which alters on the patient assuming a new position of the body.

4. The above conditions are associated with anasarca, and other symptoms of dropsy elsewhere.

When the patient lies on the back, the dulness on percussion is most marked in *the flanks;* whilst the note is *resonant* at the umbilicus. Turn the patient on the side, the fluid moves to the most dependent part, and consequently a resonant note may be obtained in the *opposite* flank.

Caution, however, is necessary as regards this resonance; it may be absent through adhesions binding the intestines

down to the posterior wall; or the fluid may not gravitate, *because of being hemmed in by inflammatory lymph.* On the other hand, if the fluid be small in amount and the distension of the intestines marked, the *dulness* may be difficult to demonstrate.

Treatment.—Seek the primary disease, and direct the treatment to improving the *general* health of the patient on the principles already laid down under hepatic disease.

If the fluid causes much dyspnœa, or other symptoms, the fluid should be evacuated by Southey's tubes, or by tapping. It must not be forgotten that in old cases, the vessels of the abdomen may be so far devitalised, that a sudden removal of the support afforded them by the dropsical fluid causes them to become engorged with blood, and set up fatal syncope.

DIAGNOSTIC TABLE.

OVARIAN TUMOUR.	PREGNANCY.	ASCITES.
1. Growth begins on *one* side.	1. Begins in median line.	1. Fluid moves on altering position. Swelling is uniform.
2. Slow growth.	2. Uniform and definite rate.	2. May be rapid or not.
3. Fluctuation general over the tumour.	3. Fluctuation absent except in hydramnios.	3. Fluctuation general over the whole abdomen.
4. No signs of pregnancy and health deteriorated.	4. Signs of pregnancy and health normal.	4. No signs of pregnancy, and health much impaired.

In all cases an examination should be made of the ascetic fluid, both microscopically and macroscopically.

Ordinary Ascetic Fluid.—Is light yellow or straw coloured, of generally 1010 sp. gr.; contains albumin. In *chylous* ascites associated with disease of the pancreas and lacteals, the fluid is turbid and milky, exhibiting oil globules. In malignant ascites, the fluid is often dark from the presence of blood. "Cancer" cells may be discovered under the microscope.

PERITONITIS.

Inflammation of the peritoneum may be either of an acute or of a chronic nature. Recent and extensive observations in the mortuary go to show that primary or idiopathic peritonitis is seldom or never seen. In order to understand this, I cannot do better than borrow Dr Fagge's words: "The peritoneum is a huge areolar space or lymph sac, and its most intimate pathological relations are *not* with skin or mucous membrane, *not* even with joints, or the so-called arachnoid space, but with pleura, pericardium, and tunica vaginalis; all of which parts are embryologically parts of the great body cavity, formed by the splitting of the mesoblast into somato-pleuræ and splanchno-pleuræ. The diseases of these three divisions of the same original cavity are the same: *acute* inflammation, serous or purulent, traumatic or septic, chronic irritative effusion and passive dropsical effusion, hydrothorax, hydropericardium, and ascites; chronic adhesive inflammation with hypertrophy. All three are liable to be invaded by tubercle and also by cancer. All three are prone to follow the pathological fate of the viscera which they cover; they are all apt to suffer in the course of Bright's disease, and, lastly, they are often all affected together by inflammation, by tubercle, or more rarely by cancer. (Second edition of Fagge's Medicine.)

A peritonitic effusion differs, however, from pleuritic effusion in showing a strong tendency to become purulent. Possibly this purulent state is to be explained by the close proximity of the septic intestinal contents.

Varieties.—1. Acute—divided into

(*a*) Local
(*b*) General } peritonitis.

2. Subacute—in which form, attacks are nearly always due to the action of local irritation.

3. *Chronic.*—This form is usually due to irritation from specific, infective, or malignant growths—*e.g.*, chronic dysentery, tuberculosis, syphilis, cancer, etc.

Symptoms.—The symptoms of a general acute peritonitis present a most characteristic clinical picture, the main features of which are—

1. Great pain and tenderness over the abdomen, which is usually tense from tympanites.

2. Quick, wiry and incompressible pulse.

3. Facies Hippocratica—*i.e.*, "a sharp nose, hollow eyes, collapsed temples; the ears cold, contracted, and their lobes turned out; the skin about the fore-head being rough, distended and parched; the colour of the whole face being brown, black, livid, or lead colour." Face is also anxious.

4. Constipation.

5. Vomiting.

6. Dry, small red tongue.

7. High fever. (?)

The abdominal tenderness is sometimes so marked, that the slightest touch or even the respiratory movements—sneezing, passing flatus, and coughing—causes exquisite agony. Possibly the constipation, and the small wiry pulse, may be due to paralysis of the splanchnic nerves. Whilst the above are the cardinal symptoms of a general acute peritonitis, it must not be forgotten that the temperature may be sub-normal, diarrhœa be present instead of constipation, and the patient's face may be actually *apathetic* instead of anxious. This latter state is probably more frequent when the peritonitic fluid speedily becomes purulent, as seen in some cases of puerperal fever, typhoid fever, and strangulated hernia.

Causation.—Nearly always secondary to perforations of some viscus, gastric ulcer, typhoid ulcer, obstructed bowel, etc.; or to extension of inflammation of neighbouring parts, such as ovaritis, salpingitis, parametritis, etc.; or to blood poisons, such as malignant fevers, especially puerperal fever; or to morbid state of the blood, as seen in Bright's disease, etc.

Pathology—

1. Hyperæmia with loss of lustre: the hyperæmia is most marked where the intestinal coils *are not* in close contact with one another.
2. Exudation of lymph, giving a more or less shaggy appearance.
3. Effusion of fluid, which may be highly fibrinous and coagulates easily, forming extensive adhesions; or which may become
4. Rapidly purulent

Prognosis.—Depends largely on the cause. When the result of perforation and extravasation of blood into the peritoneal cavity, the condition may be fatal in a few hours. It is of course much more favourable when the peritonitis is localised.

Treatment.—Give full doses of morphia or opium, one to two grains of the latter may be given every four hours until pain and peristalsis are quite subdued. Hot fomentations, *if they can be borne,* to the abdomen; ice or leeches are advised by some. As the knees are usually drawn up to relax the abdominal walls, we may add to the patient's comfort by placing a soft pillow under the hams. Ice to swallow is often most grateful. "*Avoid purgatives*"—this is a golden rule; but the *lower gut* may be often emptied with advantage by an enema of *hot* water. When the acute symptoms subside, and the danger of collapse is diminished, if there be reason to suspect pus formation, laparotomy should be performed, and the whole cavity treated as a huge abscess.

Chronic Peritonitis includes at least four well marked types—*i.e.*,—

1. Peritonitis, as a result of the invasion and subsequent breaking down of tubercles.

2. A form associated with, and due to irritation of malignant growths.

3. A localised form associated with chronic inflammation, diseases of certain organs—*i.e.*, the uterus and appendages—chronic constipation, and other affections of the lumen of the intestines.

4. A form which tends to occur at more or less definite and regular periods without any obvious cause.

Symptoms vary with the cause, the extent of the effusion, and adhesions.

Constipation alternating with diarrhœa, colicky pains, loss of flesh, exacerbations of fever, and localised tenderness, are the chief symptoms complained of.

Though ascites is often prominent, much more frequently the fluid is collected into small pools in the meshes of the bands of lymph.

The mesentery is usually much shortened, and the calibre of the gut diminished.

Physical Examination.—May reveal hard masses of tubercular or malignant growths, etc. The abdomen may be either flat and dull, or distended and resonant.

Treatment.—First seek the cause, and if possible remove it, but obviously, when dependent on tubercle, etc., all that can be done is to allay painful symptoms. Massage, with the administration of the iodides and tonics, may be of use. It must not be forgotten that chronic peritonitis is often fatal through an acute exacerbation of inflammation.

TABES MESENTERICA.

Is a disease due to tubercular degeneration of the mesenteric glands. Though associated with tubercular peritonitis, it may be a primary disease, especially in infants and young children.

Pathology.—The glands are much increased in size, and all stages of tuberculosis may be seen; in some cases, the glands are soft from fatty or caseous degeneration; others, again, show a great increase of the fibrous tissue elements, and may consequently be harder than normal.

Symptoms.—A condition which so seriously interferes with the lacteal absorption, must of necessity cause grave symptoms of mal-nutrition; and indeed, the cardinal symptoms of tabes mesenterica may be summed up—as great wasting of the limbs, thorax, and face, with a *large* abdomen, unhealthy action of the bowels, and general exhaustion. The abdomen may be (as pointed out in Chronic Peritonitis) either resonant or dull. The enlarged glands can be distinctly felt, and ascitic fluid is often present. Diarrhœa usually alternates with constipation. The stools are slimy and pasty in character; they often contain undigested milk, fat globules, and sometimes much brown pigment.

Diagnosis.—Care must be taken not to diagnose, as tabes mesenterica, those cases of mal-nutrition with large abdomen, the result of improper feeding, congenital syphilis, and rickets. A careful examination of the abdomen should prevent such mistakes.

Treatment.—Everything must be done to put the child under the most favourable hygienic conditions, as detailed under Rickets. Iodide of iron is often most useful. Iodoform applied at night to the abdomen, in the form of an ointment spread on strips of calico, followed by gentle massage in the morning, sometimes does a great deal of good.

PARASITES AND PARASITIC DISEASES.*

Definition.—Parasites are low forms of organisms—animals or vegetables—which infect other animals or plants, lodging upon them or within them, and deriving their nourishment from the tissues and juices of their host.

Division.—Parasites are divided into two great classes—

1. Animal Parasites.
2. Vegetable Parasites.

ANIMAL PARASITES.

General Characters.—Animal parasites belong to the lowest types of the animal kingdom and are characterised by the greater simplicity of their structure as compared with that of the allied nonparasitic forms.

Life History.—

1. *Metamorphosis.*—Most animal parasites exist in two or more forms; the immature, larval, or embryonic form, and the mature form. Many of them undergo a series of changes—alternation of generation—before reaching maturity; first, by sexual union producing ova which give rise to embryoes or larva, which ultimately develop into adult forms, or by a process of budding, produce colonies of the mature organisms.

2. *Host.*—The animal infected by the parasite is called the host.

* I am much indebted to my friend Dr RYLAND WHITAKER for valuable help in this portion on Parasites and Parasitic Diseases.

3. *Intermediate Host* is the organism in which the immature forms are lodged.

4. *Habitat.*—Is the part of the body of the host in which the parasite, or its immature form, takes up its abode.

CESTODA.

(Κεστος—a girdle.)

The Cestoda are all endoparasitic worms and infect the intestinal canal of vertebrata.

General Characters.—The Cestodes differ from the Trematodes in being multiple in character. The tape-worm is not a single individual, but a multitude of organisms arranged in a chain, thus forming a compound jointed colony.

1. *Shape.*—The Cestoda are compound, flat, parasitic worms.

2. *Size.*—Varies much: some forms measure $\frac{1}{4}$ of an inch, others 24 feet in length.

3. *Structure.*—The adult worm or Strobilus consists of a number of complete sexual individuals arranged in a chain. We have—

(1) The Head or Nurse, which is usually small in size, pyriform in shape and has one or two suckers surrounded by a ring of chitinous hooklets, to enable the worm to cling to the intestines of its host. It has neither an alimentary system nor sexual organs.

(2) The Proglottides.—These are a series of segments produced one behind the other by a process of budding from the head or nurse. Each segment or proglottis resembles its neighbours except in size and degree of maturity. The segments furthest from the head are the oldest, the largest, and most mature; the segments next the head being immature and having no sexual organs. In an ordinary tapeworm there may be as many as 1200 of these segments.

Each proglottis has a complete water-vascular system, composed of parallel canals running on each side of the body, and united at the hinder end of each segment by cross branches. The proglottides have no digestive organs of any kind, being nourished by imbibition. They are hermaphrodite, and produce ova by sexual union with the proglottides of other cestodes. A single proglottis may contain as many as 35,000 eggs.

Life History.—As above stated, the ova of the cestoda are produced in the proglottides, which when ripe break off from the rest of the chain and are cast out by the body of their host. Within these ripe proglottides the ova are already partially developed, and when ejected are full of active embryoes. These embryoes are enclosed in a membrane to protect them from injury, and consist of a head furnished with three pairs of silicious spines or hooklets. By the decomposition of the proglottides the embryo-bearing ova are set free, reach water, and thence find their way into the stomach and intestines of their host.

The membrane enclosing the embryo is now ruptured mechanically, or digested by the gastric juice, and the embryoes are liberated. They are called PROSCOLEXES (*scolex*, a worm), and consist of a small vesicle with three pairs of silicious spines. By means of these hooklets the proscolex fastens itself to the intestinal wall, bores through it, and makes its way to the liver or other organ of its host. Here it becomes encysted, loses its hooks, and from its hinder end develops a small vesicle full of fluid.

It is now called a SCOLEX which in some tæniada are known as *hydatids*, in others as *cysticercus*. When thus encysted, the scolex is composed of a vesicle united by a narrow neck to a head similar to that of the adult tape worm, being armed with a circlet of hooklets, and having four oscula or suckers. It has no reproductive system, nor, in fact, organs of any kind, and can undergo no further

development unless it gains entrance into the intestinal canal of man or other host. This is effected by an animal eating flesh, etc., containing the scolices, when the cysts are digested and the scolices set free. They at once lose their caudal vesicle, attach themselves to the intestinal wall of their host but do not perforate it, and in this situation soon become the head of the future tape-worm and begin to produce proglottides which again pass through the cycle of development above described. Thus we have—

1. The *Ova* discharged from the ripe proglottis.

2. The *Proscolex.*—The minute embryo liberated from the ova when taken up from water, etc., by some animal.

3. The *Scolex.*—The more advanced, but still sexually immature, embryo into which the proscolex develops when it has become encysted in the tissues.

4. The *Strobilus* or adult tape-worm, infecting the alimentary canal of its host, and composed of a head, neck, and proglottides.

Division—The Cestodes which infest man are—

1. *Tæniada*—
   - (1) Tænia solium.
   - (2) Tænia mediocanellata.
   - (3) Tænia echinococcus.
2. *Bothriocephalida*—Bothriocephalus latus.

I.—TÆNIA SOLIUM.

Synonyms—Tænia cucurbitina, Tænia humana armata, Tænia lata, Tænia vulgaris.

Larva—Simple scolex, Measle, Cysticercus cellulosæ.

General Characters.—1. Size—The adult worm or strobilus measures about 2 to 10 feet long.

2. Shape—It has a small head, long narrow neck, and transversely segmented body.

3. Structure—

The *Head* is small, rounded, about the size of a pin-head, and consists of rostellum or beak with twenty-six hooklets, and of a wider part on which are four suckers.

Body.—Next the head comes a long, narrow, thread-like neck, followed by a series of larger segments—the proglottides. At first the segments are broader than they are long, and are immature—the remaining segments are the reverse, longer than broad, and are sexually mature proglottides. These proglottides are hermaphrodite, the genital orifices being placed alternately on each side of the body, and the male and female organs open by this common genital pore. They have a complete water-vascular system.

The *Ovary* consists of a central stem, with a number of lateral branches, each of which again branches. The testes appear as clear, white, convoluted tubes, with vesicles.

Ova are nearly spherical in shape, about $\frac{1}{750}$ inch in diameter, and are surrounded by a dense capsule which encloses the partly-developed, six-hooked embryoes. These embryoes give rise to the scolices in the flesh of the pig, the scolex in this case being called a "measle," or cysticercus cellulosæ, or bladder-worm.

Life History—As above described.

Intermediate Host.—The pig, which gets the embryoes from water—the scolices forming the "measles" of pork. Within these measles or cysts, the hooklets, which do not decompose, are often found after the scolex has perished. They are short, broad, hook-shaped bodies, with a small knob at their base.

Host.—Man; owing to eating imperfectly cooked, measly pork. It infects man, not only as the mature worm, but as cysticerci. The adult worm is found in man only.

Habitat.—The *immature* worm is found in subcutaneous tissues, muscle, brain, eye, liver of the pig; the *mature* form, in the small intestine of man.

II.—TÆNIA MEDIOCANELLATA.

Synonyms—Tænia saginata, Tænia dentata, Tænia inermis, Beef tape-worm.

Larva—Cysticercus bovis.

General Characters—

1. SIZE.—Larger than tænia solium, both in length and breadth, often measuring from 14 to 24 feet.

2. STRUCTURE—

The *Head* has four suckers, but no rostellum nor hooklets. Following the head is a narrow neck, and then the several segments or proglottides.

The *Ovaries* consist of many lateral processes, but these do not, as in the case of tænia solium, again branch, a character by which the proglottis of the one can be distinguished from that of the other.

Ova—Similar to those of tænia solium.

Life History—Similar to that of tænia solium.

Intermediate Host.—Cattle.

Host.—Man.

Habitat.—Immature form, in the muscles of cattle : as many as 300 having been found in a pound of flesh taken from psoas muscles.

The *mature* form occurs in the intestine of man.

III.—BOTHRIOCEPHALUS LATUS.

Synonyms—Broad tape-worm, Tænia lata, Tænia grisea, Dibothrium latum.

General Characters—

1. SIZE.—The largest known human tape-worm. It measures from 16 to 25 feet long, and about 1 inch broad, and consists of three or four thousand segments.

2. Structure.—The *Head* is small, oval or club-shaped, with a longitudinal groove or slit on each side. It has neither proboscis, nor suckers, nor hooklets.

The *Proglottides* are about 4000 in number, the largest being in the middle of the chain. They are each bi-sexual. The uterus consists of a simple, coiled-up tube, and the genital orifices are placed along the middle line of the ventral aspect—not on the sides, as in the last group.

The *Ova* are oval in shape, and about $\frac{1}{350}$ of an inch long. They have an operculum and a brown coloured shell.

Life History—The ova are set free in the body of the host, and on reaching water are there hatched. The proglottides themselves are not discharged from the intestine as is the case with tænia solium. The embryo, which has a ciliated envelope, swims about in the water till the envelope bursts and liberates six-hooked embryoes. These make their way into the muscles of some fresh water fish, and there develop into the asexual larval worm.

If the fish be eaten by man, the larva develops into the sexual form above described.

Intermediate Host.—Probably certain fish, as pike, turbot, etc.

Host.—Man, dog.

Habitat.—Intestinal canal.

Effects—Intestinal catarrh in children, but no serious effects in the adult.

Distribution—Germany, Russia, Poland, Sweden, Holland, Belgium, Ireland, England, France.

Symptoms due to either of these three worms.

Frequently they give rise to no symptoms: there are in fact no pathognomonic symptoms; but certain reflex disturbances are common, such as itching of the nose or anus, colicky pains, constipation alternating with diarrhœa, mental trouble, such as

melancholia, epilepsy, etc., voracious appetite, and painless vomiting which may simulate brain disease. I have known all the symptoms of stone in the bladder to arise in a case dependent on reflex irritation from tapeworm. *If the above symptoms are present without any obvious cause or reason, always examine the fæces for segments of these worms.* A discharge of the segments is of course conclusive.

Treatment.—Having diagnosed the presence of the worm, then administer something which will kill the worm and expel its carcase. The following treatment may be tried.—Give at night-time for six nights running, four grains of ipecac. with one grain of pil. hydrarg.; the seventh morning on an empty stomach give ʒj of the liquid extract of male fern suspended in mucilage, and four hours afterwards give half-an-ounce of Epsom salts and a drachm of ammon. chloride in a tumbler of effervescing water. I tried this course in eighteen consecutive cases of tapeworm, and in only two cases was the treatment unsuccessful in expelling the whole worm. Turpentine, pumpkin seeds, etc., have their advocates.

Caution.—Be sure the head is expelled.

TÆNIA ECHINOCOCCUS.

General Characters—

1. Size.—Tænia echinococcus is a small worm, about $\frac{1}{8}$ to $\frac{1}{4}$ inch long.

2. Structure.—This worm consists of only four segments, including the head. The *Head* is pointed, has four suckers and a double circlet of hooks. These hooks are about 30 to 40 in number, and are shaped like those of tænia solium, but are much smaller.

The last proglottis, when mature, is equal in size to the rest of the body, and contains the reproductive organs. The genital pores are placed on the lateral aspect of the body. The

ovaries are complicated, and the ova are small but exceedingly numerous, and in them are developed the six-hooked embryoes.

Life History.—When ripe the proglottides drop off, pass out of the body of the host—the dog or wolf. The embryoes are now liberated on the ground, on plants, or in water, and thus gain access to the stomach of man. They then perforate the intestinal walls, and getting into the circulation, are by this or other means carried to the liver or other organ, where they become encysted and develop a spherical vesicle which may reach a great size. They are now called HYDATIDS.

Hydatid Cysts—

1. STRUCTURE.—These cysts when fully formed are composed of three parts—

(1) The false cyst formed by the tissues of the part.

(2) The ectocyst—an opaque, gelatinous membrane of great thickness, white in colour, smooth, glistening, and laminated.

(3) The endocyst—a more opaque, granular layer, composed of nucleated cells, and covered by small white spots—brood capsules.

(4) Inside the cyst there is a colourless watery fluid which contains salts, but no albumin, a point of value in diagnosis.

Hydatid cysts are, moreover, much larger than those of cysticercus cellulosa.

2. DEVELOPMENT.—Within these cysts the scolices—echinococcus heads—are developed in the following manner:—

The inner wall of the cyst forms small vesicles—called *brood capsules*—which project into the cavity of the cyst. From the walls of these brood capsules small cup-like buds or hollows are formed, each of which gradually elongates and becomes a cæcum with its cavity opening outwards—

i.e., it communicates with the cavity of the brood capsule. Within these depressions or hollow buds the echinococcus head is developed, and, when mature, turns itself inside out —*i.e.*, everts itself, so that the head now projects into the brood capsule. These heads are similar to those of the adult worm, having a double circlet of hooklets and four suckers.

Development cannot proceed further than this in the human body, but if the cysts gain access to the dog, etc., then the adult tape-worm is formed in the intestine.

Secondary Cysts are often found in connection with the primary cyst. This may occur in one of three modes—

1. By a process of budding out of the wall of the ectocyst, thus giving rise to a number of daughter-cysts, side by side—*exogenous cysts.*

2. Again, the daughter-cysts may be formed inside the primary cyst—these are called *endogenous cysts.*

3. Or, the cysts may be *multi-locular—i.e.*, composed of many separate alveoli divided from each other by dense fibrous tissue. They occur as hard, firm tumours in the liver.

Intermediate Host.—The cystic form is alone found in man.

Host.—The adult worm in the dog and wolf.

Habitat.—The *cystic* form is found in the liver, lungs, brain, heart, muscle; the *mature* form, in the intestine.

Diagnosis and Treatment—See Tumours of the Liver.

NEMATODA.

Synonym—Thread worms.

General Characters. — The thread worms are a very large and well-known group of helminths. They are simple,

not compound, and do not form colonies. They closely resemble the common earth-worm, being round and thread-like without segmentation or appendages. They undergo no metamorphosis, the sexes are distinct, and there is a marked difference between the male and female — the male being smaller than the female.

STRUCTURE.—The Nematoda have a distinct alimentary canal with a mouth furnished with soft horny lips, an œsophagus, stomach, intestine, and anus. There is a thick elastic ectoderm or cuticle and a well-developed muscular system. The genital pore placed on the ventral aspect is, in the female, situated about the middle of its length; in the male, near the anus, where there is a chitinous prehensile investment.

The most common Nematoda are—

1. Trichina spiralis.
2. Filaria sanguinis hominis.
3. Filaria medinensis.
4. Dochmius duodenalis.
5. Ascaris lumbricoides.
6. Ascaris mystax.
7. Eustrongylus gigas.
8. Trichocephalus dispar.
9. Oxyuris vermicularis.

I.—TRICHINA SPIRALIS.

Synonyms—Flesh worm, Pseudalius trichina.

Larva—Muscle trichinæ, Encysted trichinæ, Flesh worms.

General Characters.—Trichina spiralis is a very minute worm, the male and female being distinct.

1. SIZE.—Male, $\frac{1}{18}$ inch; female $\frac{1}{8}$ inch long.

2. Structure—

The *Head* is narrow, pointed, unarmed, with a simple central oval aperture.

The *Body* is thread-like, bent upon itself, thicker behind than in front, and in both male and female the hinder part of the body is straight. In the male, however, it has a short, bilobed caudle appendage, between the lobes of which is the anus. The testes are convoluted tubes. The female is about $\frac{1}{8}$ of an inch long, rounder and shorter behind than the male. There is an ovary, vagina, uterus, and the genital orifice is near the head.

The *Ova* are $\frac{1}{170}$ inch long, and are hatched within the parent (ovoviviparous).

Larval Form—the trichina of muscle—is a very small worm, about $\frac{1}{30}$ inch long, coiled up in a spiral manner within a fibrous capsule or cyst, the long axis of which lies in the long axis of the muscular bundles. A single capsule may contain two or more larvæ and there may be as many as 325,000 of these capsules in an ounce of meat. They are especially common in the abdominal and thoracic muscles and appear as whitish spots from the cyst being often calcified towards the poles. This small worm has a digestive system and an imperfect sexual apparatus.

Life History.—When a piece of meat affected with trichinæ is eaten by an animal, the capsules are dissolved, and the embryo parasites which they contain are liberated. These mature in a day or two in the intestinal canal of the host. The sexes unite and give birth to ova and embryoes; a single ova producing over 1000 embryoes, and a single female discharging over 16,000 ova.

The embryoes migrate from the intestine to the striped muscles, passing through the intestinal walls, but it is not clear how they reach the muscles—possibly through the peritoneal cavity or through the blood and lymph.

Once in the muscles the embryoes penetrate the primitive bundles, reduce their contents to debris and soon become mature muscular trichinæ, forming cysts, part of which is made up of a chitinous secretion of the parasite, part by a wall of fibrous tissue formed of the perimysium of the muscle bundles. These cysts, as above stated, may become partly calcareous, giving rise to white shining spots in the muscle. They may remain quiescent for years.

Intermediate Host.—The trichinæ are found in pigs, rabbits, sheep, dogs, rats, mice. The pig gets them from the rat, which acquires them from human fæces.

Host.—Man, finding their way into his body through eating uncooked pork.

Habitat.—The adult worm only inhabits the intestine and only lives for a few weeks.

Symptoms of Trichinosis. — Unless there be a large number of embryoes eaten definite symptoms may not arise, but in well-marked cases of trichinosis the symptoms are very characteristic. A few days after eating the infected flesh, symptoms of gastro-intestinal irritation appear, there may be vomiting, diarrhœa, and abdominal pain.

Towards the second week, great soreness and stiffness of muscles develop, the temperature runs up, and may be remarkably remittent in character, a characteristic œdema sets in at first in the face, but becomes specially marked in the affected muscles. In protracted cases the patient becomes emaciated, exhausted, and a typhoid condition may supervene and carry off the sufferer.

It will be easily understood that the more important muscles such as the diaphragm may be early implicated, and a fatal issue is then speedily brought about.

The disease occurs in epidemic form in some countries.

Diagnosis.—Examine stools, or pieces of muscle that have been harpooned. The more important diagnostic features are—

1. Severe pains in the joints without marked swelling.
2. Œdema.
3. Dyspnœa.
4. Marked muscular pain.

Prophylaxis.—All meat to be thoroughly well done. Avoidance of pork in an infected district.

Treatment.—Purgatives at first, combined with twenty grains of salol every night. Later, give five grains of quinine suspended in two drachms of glycerine thrice daily. Treat symptoms as they arise. No specific remedy. Morphia may be required in large doses to relieve the intolerable pain.

II. FILARIA SANGUINIS HOMINIS.

Synonyms—Filaria Bancrofti, Filaria cystica, Trichina cystica.

General Characters—The mature forms are rarely seen. The sexes are distinct. The male is said to be smaller than the female and lives in the same vessels.

"The female is described as a small slender hair-like worm with a club-shaped head, a narrow alimentary canal, a two-horned uterus usually full of embryoes. These are discharged through the vagina, which opens near the mouth."

The embryoes measure about $\frac{1}{70}$ inch long, have a rounded head, a tapering tail, and are enclosed in a fine membrane which does not burst, but which elongates as the embryo uncoils itself, thus forming a delicate sheath to the embryo.

Life History.—The adult worms inhabit the lymphatics. The embryoes gain access to the stomach of their host through drinking water; they thence make their way to the vessels.

Here the sexes unite, and the embryoes are discharged into the lymphatics. In the daytime the embryoes are found in the lymphatics, but at night they crowd the blood stream. They cannot, however, undergo further development in man, and hence are taken up by a species of mosquito, in the body of which the embryoes are matured. When the insect dies, they find their way to water, thence to the stomach of their host, and, on reaching the blood and lymphatic system, form the mature filaria.

Intermediate Host.—Mosquito.

Host.—Man.

Habitat.—The embryo, called filaria sanguinis hominis —in the blood and urine; the mature form, called filaria Bancrofti—in the lymphatics.

Symptoms.—The passage of an opaque milky urine tinged with blood, is the most prominent symptom in the bulk of cases; *but the enormous lymph scrotum and certain forms of elephantiasis are also supposed* to be due to the presence of this parasite. Sometimes a blood-clot may form either in the bladder or pelvis of the kidney giving rise to troublesome symptoms.

Treatment.—At present very unsatisfactory. Iodide of potassium, turpentine, carbolic acid, etc., have their advocates.

III.—ASCARIS LUMBRICOIDES.

Synonyms—Round worm, Lumbricus teres hominis.

General Characters—These parasites closely resemble the common earth worm.

Size.—The male measures about 4 to 6 inches; the female, 10 to 12 inches long.

Structure.—This parasite is a broad, smooth, fusiform, translucent, brown or red coloured worm, with fine circular

striæ. Its anterior extremity has a three-lobed mouth. The tail is bluntly curved in the male, and has a double spicula near its end.

Ova, oval shaped, $\frac{1}{500}$ to $\frac{1}{150}$ inch in diameter, and have a hard shell and an albuminous envelope.

Life History—Not fully known.

Host.—Man, pig.

Intermediate Host.—Not required.

Habitat.—The ileum, colon, also mouth and nose. They are passed by the fæces, or are vomited.

Symptoms.—Very indefinite, in fact there are no characteristic symptoms. Picking of the nose, grinding the teeth, foul breath, etc., are the popular symptoms of the presence of this worm, but obviously any irritation of the intestinal canal may cause such symptoms. Perhaps we regard the presence of worms as a source of peripheral and intestinal irritation too lightly at present. Our forefathers distinguished a "worm fever" and prescribed successfully for that ailment.

Treatment.—Santonin four grains followed by three grains of calomel.

TREMATODA.

Synonyms—Flukes, Flat-worms, Suctorial-worms.

The only parasite under this class that we shall consider is

DISTOMA HÆMATOBIUM.

Synonyms—Bilharzia Hæmatobia, Thecosoma, Schistosoma, Gynæcophorus.

General Characters.—They are diœcious, the sexes being distinct.

1. Male—
   (1) Size.—About $\frac{1}{2}$ inch long.
   (2) Shape.—Cylindrical, with a canal or groove—gynophoric canal — at the posterior end of the body in which the female is lodged.
2. Female—
   (1) Size.— $\frac{4}{5}$ inch long.
   (2) Shape.—Thread-like.

Both male and female have two ventral suckers, and the reproductive orifice is below the ventral sucker.

Ova.—Oval in form, $\frac{1}{180}$ to $\frac{1}{160}$ inch in diameter, with a spine at the ends or at the sides of each egg.

Life History.—Not known.

Intermediate Host.—Not known.

Host.—Man and monkey ; got by drinking water.

Habitat.—The blood. Is especially found in the inferior vena cava and portal veins, and in the vesical and hæmorrhoidal veins.

Effects—The sexes unite in the blood—the ova are discharged, and pass through the walls of the bladder and ureter by means of the ulcerated surfaces caused by the parents. If in large numbers, they give rise to inflammation and hæmorrhage from the affected mucous membrane, causing endemic hæmaturia—or if the large intestine be affected, a special form of diarrhœa is the result. The ova pass out of the body by these channels, and can be found in the urine of the patient. If placed in warm water, the ova give rise to the free ciliated embryoes.

Distribution.—This parasite is rarely met with in England, but is common in Egypt, Cape, Natal, also in Brazil.

DISEASES OF THE MOUTH.

THRUSH OR PARASITIC STOMATITIS.

Is a *specific* disease dependent on the development and multiplication of a special fungus termed the *odium albicans;* is frequently met with in weakly children and infants.

By some the fungus is regarded as belonging to the yeast tribe, by others it is looked upon as a mould; whatever its nature is, it causes the formation of milk-white or greyish adherent patches on the mucous membranes of the mouth and pharynx of the child attacked.

Microscopically these patches consists of epithelium united into a membrane by twisted filaments (the fungus) which are often branched and composed of long cells joined end to end, and constricted at the joints.

The patches may extend to the œsophagus, stomach, and cæcum.

Symptoms.—The mouth is *dry*, and tender or painful; there is usually much debility and gastric disturbance. (This affection must not be confounded with aphthous stomatitis, presently to be described.)

Treatment.—Absolute cleanliness of the feeding bottles, the avoidance of stale milk, and the frequent swabbing out of the mouth with a solution of boracic acid or permanganate of potash, are the principle indications for successful treatment. Everything must be done to improve the debilitated condition generally.

STOMATITIS.

An inflamed condition of the mouth is common during dentition, certain fevers, and morbid states of the blood.

Such a condition, however caused, is likely to be attended with the following symptoms—

1. *Pain and difficulty* in mastication, articulation, and deglutition, in proportion to the severity of the disease.
2. Increased salivation.
3. Fœtor of breath.
4. Constitutional disturbances.

Like all inflammations of mucous membranes, the condition varies much in severity, and accordingly the following types are described :—

The simple or erythematous stomatitis, a mild form which readily yields to the local application of glycerine and borax.

Aphthous stomatitis, characterised by the formation of small vesicles or yellowish patches of a diphtheroid nature. The vesicles after rupturing leave small greyish ulcers. They appear on the gums, tongue, lips, etc., but seldom upon the pharynx ; they spread by contact.

This type has assumed an epidemic form in localities where cattle affected with foot-and-mouth disease were present. The relation between the two conditions have, however, not yet been determined. Some authorities term this epidemic form ulcerative stomatitis, or putrid sore throat.

Treatment.—Touch the ulcers with solid nitrate of silver. Improve the general health of the patient with tonics. A mixture of hydrochloric acid, chlorate of potash and glycerine, freely swabbed over the mouth, often acts like magic.

NOTE.—True aphthous stomatitis is more common *after* the period of dentition.

CANCRUM ORIS.

This is a form of gangrenous stomatitis, happily now rare, but sometimes seen in debilitated children between the ages of two and five years.

Pathology.—At first there appears a small diphtheroid patch of necrosed tissue with a general inflammation around it. This inflammatory zone extends and becomes brawny in character, the slough separates, and the ulceration goes on until the cheek is perforated. The disease frequently kills the patient before necrosis is well marked, but sometimes the gangrene extends to the jaw, malar bones, tongue, etc., before a fatal termination is brought about.

Symptoms.—The constitutional disturbance is very great, and usually the typical typhoid state rapidly ushers in a fatal ending.

Treatment.—Highly unsatisfactory, for it has been fairly well demonstrated that the virulence of the affection is not so dependent on the poison as much as the peculiar debilitated state of the patient, and the unhealthy surroundings in which he exists.

The application of the Paquelin cautery, with the administration of quinine and stimulants are the best measures.

SUPPURATIVE TONSILLITIS OR QUINSY.

Inflammation of the tonsils is not only a common complication or symptom of many fevers, but is also a common primary affection; but when the inflammatory process goes on to suppuration (as it frequently does) it is termed quinsy.

Ætiology.—Quinsy occurs most frequently in young people, but may occur at any age. There seems to be some relation

between the poison of quinsy and acute rheumatism, as the latter frequently begins as the former, and moreover, the temperature is remarkably high at times in cases of suppurative tonsillitis, and the joints are often very painful. Is quinsy a local affection, or is it a constitutional fever with marked local symptoms? These questions are not easily answered, but there can be no doubt that—

1. Suppurative tonsillitis is often epidemic in character.
2. It tends to recur at certain seasons in the same individual.
3. Once begun, the inflammation is not easily aborted until suppuration commences.
4. The fever and other constitutional symptoms are often out of all proportion to the local affection.

Pathology.—The crypts or follicles become much inflamed. Their epithelium proliferates and their lumen becomes filled with yellowish pus-like debris. These points may coalesce and form a dirty patch. The whole tonsil becomes swollen, intensely injected and œdematous, and in three or four days the interior of the tonsil becomes converted into an abscess. Usually one tonsil only is attacked, but the other frequently becomes similarly affected as the first gets better.

Symptoms.—First there is a slight soreness at one side of the throat, and a desire to swallow more frequently; this soreness increases as the tonsils become more inflamed. The angle of the jaw is also swollen, and any movement of the jaw or deglutition is attended with great pain. The temperature tends to run up rapidly, and I have notes of nine cases where the temperature exceeded 106° F. There is often intolerable headache rendered worse by movement, especially on rising from the horizontal position. The tongue is foul and the breath fœtid.

Diagnostic Points.—The rapid onset—the one tonsil affected—great tendency to suppurate—high temperature.

Treatment.—Open the bowels freely by saline purges. Subdue fever and relieve headache by antipyrin or salicylates. Gargle the throat with hot milk, or brush it with a hot solution of soda and morphia. Don't use astringents in the acute stage, they only irritate the inflamed part. In the *low subacute* forms, frequently seen in over-worked patients, nothing succeeds better than a good swabbing once for all of the liq. ferri perch. fort. It is not a pleasant application, but is very efficacious. Nourishing diet and tonics are absolutely required during convalescence.

MISCELLANEOUS DISEASES.

RAYNAUD'S DISEASE.

For a description of this peculiar disease we are indebted to the researches and writings of Dr Raynaud, and also, to a large extent, to Dr T. Barlow's able translation of Raynaud's work (1888). Raynaud's disease may be more or less correctly defined as a vascular disorder, dependent on vaso-motor disturbances which cause constriction of the peripheral arterioles, and so induce changes of various degrees conveniently termed — (1) Local syncope; (2) Local asphyxia; (3) Local and symmetrical gangrene.

1. *Local Syncope.*—This condition resembles the dead fingers or toes produced by intense cold—one or more fingers, *or the whole hand*, may be affected. Often a severe reaction sets in, and the parts numbed before now become intensely red, hot, and painful.

2. *Local Asphxia.*—This condition is characterised by the fingers, ears, and certain patches of skin on the arms and legs, becoming intensely congested, livid, and the capillary circulation arrested. The congestion gives rise to swelling,

stiffness, and pain; the latter is often succeeded by marked anæsthesia.

These attacks may occur at winter time for many years as "chilblains."

3. *Local or Symmetrical Gangrene.*—The parts asphyxiated become cold, insensible, and black in colour—*i.e.*, changes identical with necrosis elsewhere. Usually a line of demarcation forms and limits the necrotic condition.

Complications—

1. Hæmoglobinuria is so frequently present, that it is more like a symptom than a complication of Raynaud's disease.
2. Uræmia.
3. Œdema of the glottis and lungs.
4. Heart failure.
5. Inflammation of serous sacs.
6. Cerebral symptoms, independent (apparently) of uræmia.

Pathology. — Unknown. Vaso-motor constriction, vaso-motor dilatation with subsequent venous engorgement, altered conditions of the blood, etc., have been advanced as the causes. Better be frank and say we don't know anything about the pathology of this disease at present.

Treatment.—Improve any obvious condition of ill-health. Locally, make the circulation more brisk by gentle friction, warmth, and gentle galvanism. Soothe pain by sedatives such as belladonna, morphia, conium, etc.

SCURVY.

Is a constitutional affection characterised by great debility, a spongy condition of the gums, a tendency to hæmorrhages, and cachexia.

Ætiology. — Is much less common now compared with former times. The disease is usually associated with improper

and insufficient food, unhealthy hygienic surroundings, etc. Sailors afford the best examples, as a rule, of this disease.

Pathology. — There are three theories as to the causation of scurvy—viz., that it

1. Depends on the presence of a specific organism.
2. Deficiency of *potassium* salts in the blood.
3. Reduction of the alkalinity of the blood, not necessarily through the absence of potash, but a reduction in quantity of all the salts that tend to keep the blood alkaline.

Moreover, as citrates, malates tartrates, etc., become changed into carbonates in the blood, we may assume at present that scurvy is really due to an inadequate supply of organic acids, salts, etc., which are to be found in fresh vegetables, fruits, etc.

Symptoms.—Are insidious at first; the patient becomes weak, breathless, drowsy or languid, with more or less aching of the bones and joints generally. The gums become soft and swollen, bleeding easily on the slightest pressure.

As the disease becomes marked, the teeth may come out, the mouth becomes ulcerated and emits a frightful fœtid odour. Petechial hæmorrhages around the hair follicles, may be followed by large subcutaneous extravasations of blood on the extensor aspects, into the conjunctivæ, popliteal spaces, etc. The patient assumes a cachectic appearance, and is rendered quite unfit for mental or physical exertion. In some cases a peculiar form of night blindness develops; this condition is not dependent on permanent ocular changes, though optic atrophy has been observed in more than one case.

Treatment—

1. General hygiene must be attended to.
2. Diet should consist of good soup, fresh milk, cream, etc. Effervescing drinks made with fresh lemon juice are of special importance.
3. Medicinal.—Quinine dissolved in citric acid.

HÆMOPHILIA.

Is a peculiar diathesis characterised by a tendency to excessive and uncontrollable bleeding. It differs from purpura in being always present throughout the patient's life.

Many varieties have been described such as—

1. Those in which a slight traumatism is followed by excessive hæmorrhage.

2. Those which exhibit the condition after injuries of certain regions only.

3. Those in which the hæmorrhages take the form of attacks of spontaneous bleeding from the nose, uterus, mucous membranes, etc.

Pathology.—Unknown, as no *constant* condition or changes have been found post mortem either in the blood-vessels or blood; probably the fault lies in some structural changes in the walls of the capillaries. The diathesis is marked by being hereditary and is transmitted through the female line.

Symptoms.—Are sufficiently obvious, and need no detailed description. It must be remembered that in these cases death has followed the extraction of a tooth, cutting a corn too deeply, snipping a wart off, and other usually trifling operations. Often the first severe attack of bleeding occurs in infancy, or, on the other hand, it may first appear after adult age.

Treatment.—When we suspect the presence of this diathesis, a careful watch should be made over the patient, and on the slightest appearance of hæmorrhage, energetic treatment with styptics must be at once employed. Obviously, the bleeder must live on a non-stimulating diet, lead as even and placid a life as possible, avoid excesses of all kinds, especially the

ingestion of liquids and other compounds which tend to raise the blood pressure. No female of the hæmophilic stock should marry.

PURPURA.

Purpura is not a disease, but symptomatic of some grave change in either the blood or blood-vessels, whereby extravasations of blood into various tissues occur, producing red or blue patches which do not disappear on pressure or after death. Being symptomatic in character, we may expect several varieties, such as—

1. A form associated with the malignant fevers—*i.e.*, those fevers in which the "rashes" become hæmorrhagic, as already described under typhus, black measles, etc.

2. A form associated with grave *constitutional* changes—*i.e.*, syphilis, chronic Bright's disease, cardiac diseases, scurvy, etc.

3. A form associated with the circulation of certain poisons, to wit, snake poison, quinine, ergot, mercury, etc.

4. A form associated with arthritis—*i.e.*, (1) Peliosis rheumatica, already referred to under rheumatic fever; and (2) Febrile purpuric œdema—a condition in which many joints become inflamed from other causes than rheumatic poison.

5. A form probably neurotic in character, as seen in cases of myelitis, locomotor ataxia, severe neuralgia, purpura urticans, etc.

6. Purpura hæmorrhagica, a form which occurs without any apparent reason, and not associated with any particular condition. It differs from hæmophilia in not being a constant possession or factor, but a temporary condition which may or may not be highly amenable to treatment.

Symptoms.—The presence of petechiæ in the skin, etc., are sufficiently obvious to demonstrate the condition. When not

associated with any apparent cause, the more common symptoms are—marked anæmia, sallow complexion, local œdema or general anasarca, and more or less severe muscular pains. Death may occur from progressive exhaustion, or from internal hæmorrhages. Unlike scurvy, the gums are not swollen, no history of want of fresh air, vegetables, etc.

Treatment.—As we know nothing about the pathology of this disease, the treatment must be entirely emperical, or experimental. Aim at removing any apparent cause of ill-health, restoring tone of blood-vessels, etc. Ergot, iron, turpentine, strychnine, gallic acid, opium, etc., may be tried.

ACROMEGALIA.

Is a peculiar disease characterised clinically by an excessive growth chiefly of the face and extremities, and associated pathologically with enormous hypertrophy of the pituitary body. In some cases the thyroid gland has been found enlarged, and a persistence of the thymus glands noted, but these two changes are looked upon as accidental occurrences.

Symptoms.—Rarely is a more characteristic clinical picture presented than a confirmed case of acromegalia. The face is egg-shaped, with its broad end downwards; the lower jaw-bone particularly is much increased in size, and may cause the lower teeth which are wide apart to project, giving anything but a pleasing appearance; the tongue is somewhat enlarged and may cause a similar leathery speech to that of myxœdema. The extremities are markedly altered, the hands being huge, spade-shaped, and the nails broad and large. The lower extremities show similar abnormal overgrowth, and may render the gait peculiarly clumsy. The spine is often of the kyphotic type, and the abdomen being thrown outwards at the same time causes a characteristic "double hump."

There is much general lassitude and weakness, and usually *marked headache* and anæmia.

The menses in women may be arrested. Defects of vision are often present, the more common being either single or double hemianopsia; such conditions, however, are not always permanent. Polyuria and glycosuria may be present in some cases. The skin often presents pedunculated tumours and in marked contrast to myxœdema often is bathed in sweat.

Pathology.—At present is very obscure, though, from the constant presence in these cases of a much hypertrophied pituitary body, the condition seems to be dependent on changes in that gland. This, however, must be regarded at present as purely speculative, for cases of *atrophy* of the pituitary body have apparently been attended with no particular changes. The persistence of the thymus gland rests upon the slender evidence of increased dulness in its normal area.

Treatment.—Arsenic, strychnine, massage, galvanism, and the injection of extract of thyroid gland, or extract of pituitary body may be tried.

ACTINOMYCOSIS.

Is a chronic inflammatory affection set up through the irritation caused by the presence and multiplication of the ray fungus.

General Characters (Macroscopic)—

1. In Cattle.—The disease usually begins in the lower jaw, and causes sarcomatous-like growths—bulky tumour-like masses—connected with the bone. The bone is softened, eroded, leading to the formation of an abscess. The tongue is also affected, hard indurated nodules being produced which give a gritty, woody feel; hence the name, Woody Tongue. The virus is carried by grain. It does not affect carnivora.

Sites of the lesion—

1. Jaws—tongue—neck—glands beneath the jaw.
2. Larynx—lung—alimentary tract.

2. In Man.—The disease also affects the jaw, and leads, as in cattle, to the formation of nodular masses, of deep-seated abscesses (retro-pharyngeal) in the region of the spine, etc.

Nodules also occur in the lungs—patches of chronic induration, abscesses, and cavities.

The liver, intestines, the kidneys, and ovaries are also affected.

The masses tend to suppurate, *not to caseate.*

Structure (Microscopic)—

The nodules are seen to consist of masses of granulation tissue. They have a sponge-like arrangement, and contain small yellowish granules. They have a structure very similar to that found in tubercle, being composed of—

1. The fungus—ray fungus in the centre.
2. Round this there are the giant cells.
3. Epithelioid cells.
4. Masses of granulation tissue.

Mode of Inoculation—

1. Through the mouth by carious teeth, etc.
2. By the respiratory tract.
3. Through the intestinal tract.
4. Through the intestinal vaginal orifice.
5. In many cases the mode of inoculation is unknown.

Ray Fungus—

1. *Characters.*—The exact botanical position of the ray fungus is not known. By some it is regarded as a mycelium, by others as a leptothrix or cladothrix.

2. *Structure.*—The fungus consists of short threads or rods, often club-shaped, dotted, branched, sometimes calcified,

arranged in a radiating manner round a common centre, composed of fine filamentous fibres. They form the globular masses already spoken of. The central part of the mass is the living cladothrix, the rays are *degenerated filaments.*

3. *Cultivation.*—Actinomycosis has been cultivated but not with uniform success. It has also been inoculated, but here, also, the results have not been altogether very satisfactory.

Symptoms.—The symptoms of this remarkable disease depend entirely upon its mode of entrance, and the site of its most rapid multiplication; thus its pernicious influence may be most marked in—

1. The alimentary canal.
2. The pulmonary organs.
3. The brain.
4. The skin, etc.

Professor Grainger Stewart, in conjunction with Dr Robert Muir, gives, in "Edinburgh Hospital Reports," vol. 1., a most exhaustive account of a case which began in the ovaries.

Treatment.—It is at present unsatisfactory, because the condition, in the first place, is not easily diagnosed; and secondly, when it is diagnosed in man, the extensive inflammatory changes, the numberless adhesions set up, etc., give little chance of even successful surgical treatment. Medicinal measures are certainly less effective. The cases, indeed, require similar treatment to that of chronic pyæmia.

ANTHRAX.

Anthrax—also called charbon, malignant pustule, wool-sorters' disease, splenic fever, splenic apoplexy—is a disease which especially attacks cattle, horses, sheep, deer; less

commonly swine, dogs, etc.; but it also occurs in man, and is due to the agency of a specific bacillus—bacillus anthracis.

Division.—Anthrax, as it occurs in man and animals, may be—(1) Local; (2) General.

In man the disease manifests itself chiefly in the local form; in cattle, etc., the general form is more common.

Characters.—(1) In animals; (2) In man.

1. Anthrax in Animals—

1. There is but little local lesion. The chief changes are in the *spleen*, which becomes like a mass of blood-clot—hence the name, splenic apoplexy.

2. Hæmorrhages occur in such organs as the lungs, wall of heart, cortex of kidney, brain and its membranes.

3. There are areas of inflammatory exudation—cellulitis, effusions into serous cavities.

4. The blood has a dark colour, and is crowded with bacilli.

5. Lymphatic glands are also affected—more especially in animals, not so much in man.

2. Anthrax in Man—

Anthrax occurs in persons working amongst hides, hair of cattle, etc.; hence in tanners, wool-sorters. The poison, however, can be carried by flies.

1. Mode of Inoculation—

(1) By wounds.
(2) By the respiratory tract.
(3) By the alimentary tract.

2. Division.—There are two forms in man—

(1) *Local Form.—Malignant Pustule*—due to inoculation; hence it occurs on exposed parts—on the face, neck, lips,

hands, arms. It commences as a small pimple surrounded by a bluish inflammatory zone. The pimple may or may not burst, but in two or three days it enlarges and fresh vesicles form—bullæ follow, and we get local gangrene and necrosis. General blood-poisoning usually ensues. The bacillus is found in the superficial and subcutaneous lymphatics of the affected area, and afterwards in the blood, etc.

(2) *General Form.*—Rare in man, but may affect—

(*a*) The *Respiratory Tract.*—Wool-sorters' disease. The primary lesion is usually in the lower part of the trachea and larger bronchi, where there are patches of intense swelling of the mucous membrane, with hæmorrhages and ulcerations. Great swelling of mediastinal glands, which, from hæmorrhages, look like blood-clots.

(*b*) The *Gastro-intestinal form* gives rise to diffuse inflammation, with partial detachment of mucous membranes. Hæmorrhages also occur in this situation. In these cases there are few bacilli in the blood, but they are found in great numbers in the bronchi and in lymphatic glands.

BACILLUS ANTHRACIS.

1. Characters—

(1) *Shape.*—As it occurs in the blood of affected animals, bacillus anthracis consists of straight rods joined end to end, with blunt, slightly curved extremities. Motionless — thus differing from bacillus subtilis. There are no spores in the blood; these have been seen in the kidneys only.

(2) *Size.*—They vary considerably—from 5 to 10 or 20 m. in length—1·2 m. broad.

2. CULTIVATION. — Bacillus anthracis will grow on gelatine, agar-agar, potatoes, in hay infusions, in aqueous humour. It liquefies jelly, and sinks to the bottom of the vessel in whitish masses. It requires oxygen for its growth. When cultivated in potatoes, it gives rise to dry, creamy, yellow-coloured masses. When grown at a temperature of 15° to 42° C., it forms long filaments full of spores.

Treatment.—The site of inoculation should be at once incised or touched by the actual cautery.

If seen after marked symptoms have developed, support the patient with stimulants and quinine. Locally, carbolic acid may be injected into the brawny induration around the seat of inoculation.

HYDROPHOBIA.

Is a disease due to the inoculation of a specific poison generated in animals suffering from rabies.

Symptoms.—The wound by which the poison was introduced, as a rule, rapidly heals, and for a time nothing happens to attract the patient's attention to the scar. In about nine weeks or so, the scar may become painful and nervous disturbances manifest themselves. The patient becomes sleepless, peevish, irritable, and experiences a choking sensation about the throat. When the disease is fully developed the spasms become intense, the respiratory muscles and those of deglutition are specially involved; but a more or less tetanoid condition may be observed of nearly all the muscles. The features may be horribly contorted or wear an aspect of extreme terror; the saliva is not swallowed, and the collection of this in the mouth, causes the so-called foaming of the mouth.

The peculiar hissing character of the inspirations has been magnified by attendants to barking like a dog. The face is usually flushed or livid during the attacks, and there may be raving delirium, delusions, and hallucinations. It should be noted that, though the patient is very thirsty, he is afraid to drink, as any attempt at swallowing brings on the spasms at once; even the sound of running water will excite the attacks. The disease is usually fatal, if left alone, in from two to ten days after the development of the characteristic symptoms.

Treatment.—The wound when inflicted should be at once sucked, if the mucous membrane of the mouth be unbroken; ligature when possible *above* the bite, cauterise the wound or excise the injured part entirely. Watch for symptoms. When practicable send patient to Pasteur's Institute.

Pasteur's Method.—He obtains a strong virus by successive cultivations of specially inoculated rabbits. When he has succeeded in getting a virus which will cause madness in *the shortest possible period*, he next exposes certain portions of the prepared or inoculated spinal cord to the air. The air has the power of destroying the virus if it be long enough exposed, consequently the virulence or strength can be graduated by the length of time exposed. Pasteur has found that by inoculating with an extremely weak virus at first, he can by successive inoculations of stronger but graduated virus, render the patient incapable of being affected with this disease. In other words—*he uses up the necessary soil* for the development of rabies, by injections of graduated strengths of the virus. As this can be done during the ordinary incubation period after a bite from a rabid animal, *by the time the symptoms of rabies are due as it were, the soil is no longer suitable for the poison to develop, hence no bad results follow the wound first inflicted.*

TETANUS or LOCK-JAW.

It is an infectious disease dependent on the presence and multiplication of a special bacillus, and characterised clinically by severe tonic spasms of the muscles, especially those of the jaw.

Ætiology.—It most often occurs after trivial injuries to the hands or feet. It has been known to occur in epidemic form amongst newly-born children. When idiopathic in its origin, there is generally a history of sleeping on damp and infected soil. The organism is known as the "bacillus of Nicolaïer," who has cultivated it from certain putrefying fluids and surface soil. The bacillus has one end knobbed like a drumstick, grows into long threads, is mobile and anaërobic. A ptomaine "tetanine" obtained from a body after death from tetanus will, if inoculated into a healthy animal, produce lock-jaw (Briegor).

Symptoms.—Usually within ten days of the injury, the patient complains of stiffness in the neck and muscles of the jaw. Gradually lock-jaw is produced by the tetanic spasm of the muscles; the eyebrows at the same time become elevated, and the angles of the mouth drawn out, causing the so-called *risus sardonicus*. Soon all the muscles may be affected with paroxysms of tetanic spasms. The body may be arched, the patient resting on his head and heels (opisthotonus); pleurosthotonus or emprosthotonus are also common conditions. The pain amounts to agony during the paroxysms. The patient may be bathed in sweat, and the temperature reaches sometimes as high as 110° or 112° before death.

Diagnosis.—A diagnosis must be made from poisoning by strychnine. It is difficult to confound tetanus with strychnine poisoning, for the latter never causes the jaws to *remain* tetanically closed between the spasms.

Treatment. — Darkened room — absence of any source of irritation. During the severe paroxysms, give chloroform; between, administer hypodermic injections of morphia, hyoscine, curara, or atropine. The patient may be fed, per rectum, with pancreatised meat extract, etc.

RUSSIAN INFLUENZA.

It is extremely difficult to define what condition is exactly meant by the above heading, but possibly the following description will convey the main or essential points. Russian influenza is a convenient name given to an epidemic disease which has of late swept over Europe, and characterised clinically by—

1. Sudden onset.
2. Severe prostration or nervous exhaustion.
3. Tardy convalescence.
4. Serious sequelæ.

The disease has but few of the characteristic features of ordinary influenza cold. It is impossible to present a clinical picture that will embrace even the majority of cases. Three distinct types are met with—

1. *Nephritic Type.*—When the lumbar pain is intense, general aching of the joints, etc. The urine is scant, high coloured, but contains no albumin.

2. *Respiratory Type.*—In which form the thoracic organs tend to become most involved, pneumonia being very common.

3. *Cerebro-Spinal Type.*— A form which simulates cerebro-spinal fever, even unto the petechial rash on the wrists.

Kitasato claims to have discovered the organism that causes this disease: he describes it as consisting of "small rodlets strung together in threads. These bacilli are found in great numbers in the saliva and sputa of influenza patients."

"The history of the recorded epidemics of La Grippe is marvellously complete for centuries. Every country and every climate in the world is subject to it, yet it appears to find a permanent home nowhere as a constant or endemic resident, but to disappear from the face of the earth *for a series of years.* This ubiquitous disease has attacked humanity in its own violent fashion at short intervals from probably the earliest ages." (A. B. GRIFFITH'S "Bacteriology.")

Whatever the poison may be, several things are certain :— It carries off the aged and the feeble, and renders ordinary pulmonary diseases much more fatal; for instance, a patient may be recovering from some pulmonary attack, but influenza asserts its presence, and in such cases the patient only too frequently dies in spite of all attention and skill. Blindness, insanity, deafness, and a host of other severe conditions have been reported as results of this peculiar disease. In spite of the many monographs written on this subject, we may as well confess we know *little* about it; indeed, it is difficult to get accurate details. The *mild* form attacks those who can be laid up conveniently—*i.e.*, those whose salaries continue during illness. One is tempted to ask, "Is there a mild form of this disease?" To account for the extreme nervous depression in the real disease, the following theories are suggested—*i.e.*, that

1. The poison paralyses the cardiac ganglia.
2. The poison paralyses the cardiac muscle.
3. The poison paralyses the chief centres of the medulla.

Treatment.—Stimulants seem absolutely necessary in the bulk of cases. Of drugs, hydrobromic acid, quinine, antipyrin, salicylates, etc., have their advocates, but each case must be considered in detail and treated accordingly.

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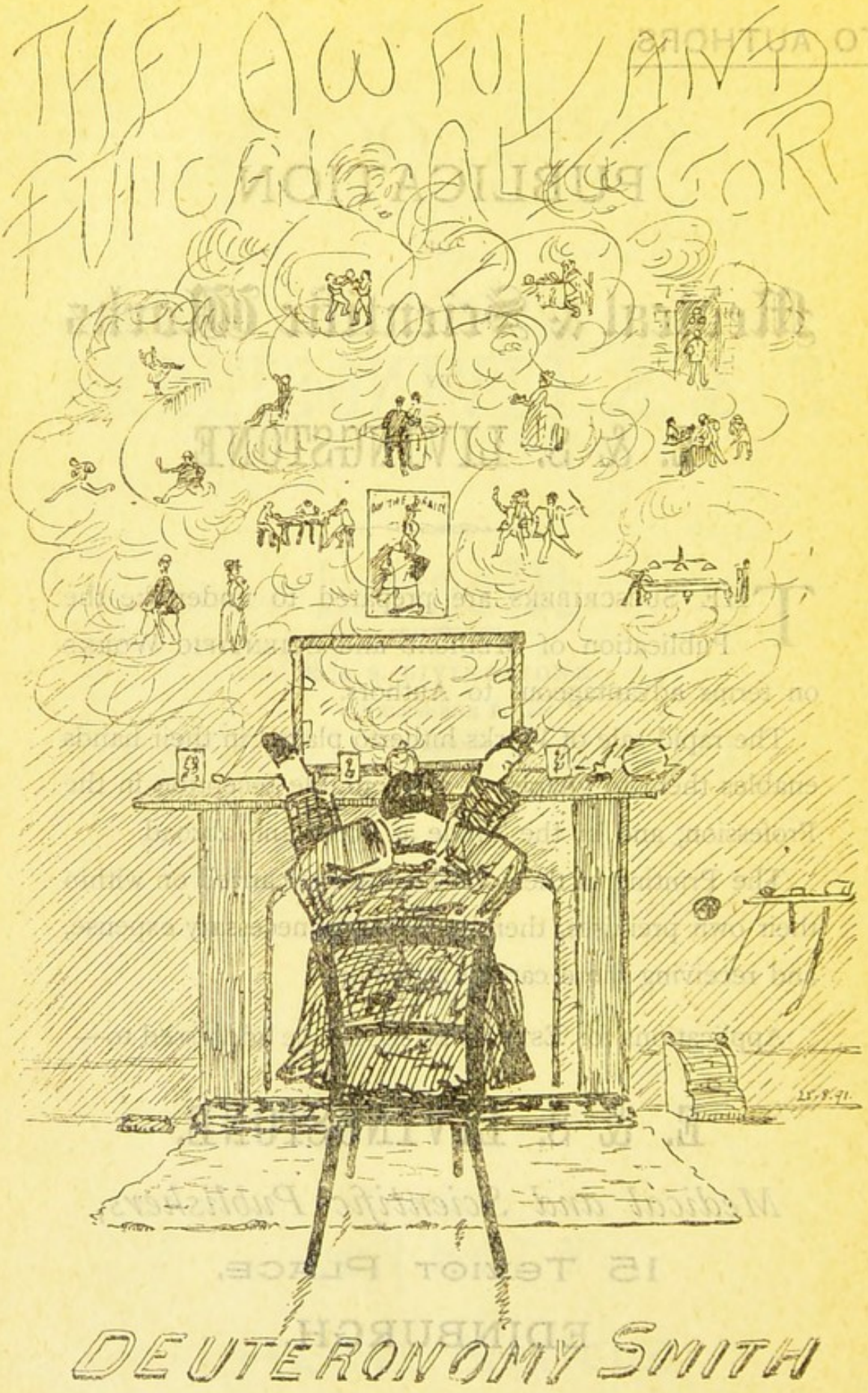
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