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ESSENTIALS OF PRACTICE OF MEDICINE

HENRY MORRIS, M.D.

QUESTIONS AND ANSWERS

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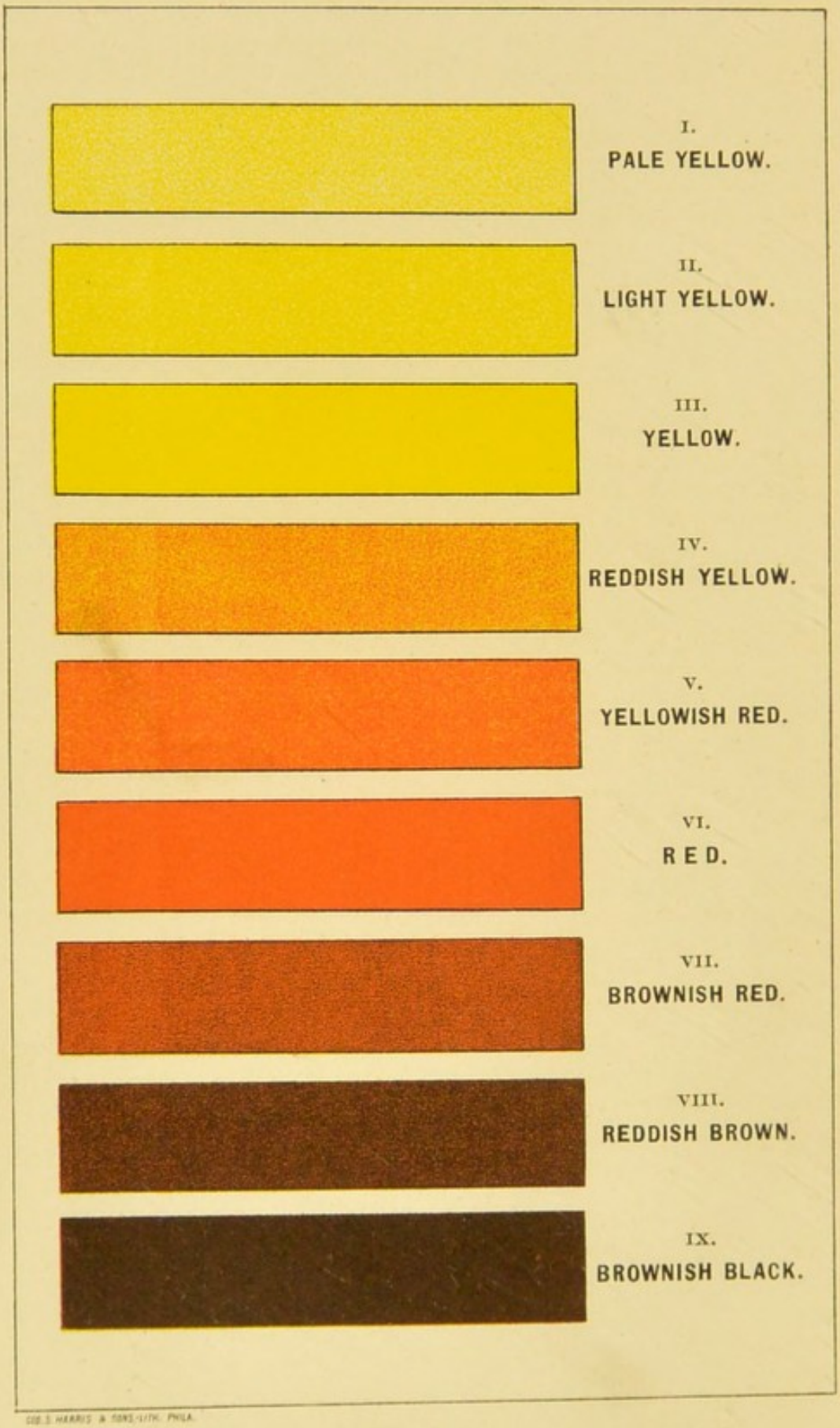
Scale of Urinary Colors, according to Vogel.



GEO. S. HARRIS & SONS LITH. PHILA.

Saunders' Question-Compends, Nos. 8 & 9.

ESSENTIALS OF PRACTICE OF MEDICINE.

ARRANGED IN THE FORM OF

QUESTIONS AND ANSWERS.

PREPARED ESPECIALLY FOR

STUDENTS OF MEDICINE.

BY

HENRY MORRIS, M.D.,

LATE DEMONSTRATOR JEFFERSON MEDICAL COLLEGE, PHILADELPHIA; VISITING PHYSICIAN TO ST. JOSEPH'S HOSPITAL: FELLOW COLLEGE OF PHYSICIANS, PHILADELPHIA; CO-EDITOR BIDDLE'S MATERIA MEDICA, AUTHOR OF ESSENTIALS OF MATERIA MEDICA, ETC. ETC.

WITH A VERY COMPLETE

APPENDIX, ON THE EXAMINATION OF URINE,

BY

LAWRENCE WOLFF, M.D.,

DEMONSTRATOR OF CHEMISTRY, JEFFERSON MEDICAL COLLEGE.

COLORED (VOGEL) URINE SCALE AND NUMEROUS ILLUSTRATIONS.

THIRD EDITION,

REVISED AND ENLARGED BY SOME THREE HUNDRED ESSENTIAL FORMULÆ, SELECTED FROM THE WRITINGS OF THE MOST EMINENT AUTHORITIES OF THE MEDICAL PROFESSION.

COLLECTED AND ARRANGED BY

WILLIAM M. POWELL, M.D.,

ATTENDING PHYSICIAN TO THE MERCER HOUSE FOR INVALID WOMEN AT ATLANTIC CITY, N. J.

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PREFACE TO THIRD EDITION.

THE fact that this small Manual of the Practice of Medicine should have passed through three editions since its issue, in the latter part of 1890, is sufficient guarantee of its popularity and that the Author's plans have not miscarried.

Great care has been exercised in the revision not to increase its size, and thus rob it of its usefulness to the student, who has but little time for large and exhaustive works. All obsolete and useless matter has been omitted, and the very latest physical diagnosis and treatment substituted, thus bringing the work thoroughly abreast with the times.

HENRY MORRIS.

313 So. SIXTEENTH ST.
Nov. 1, 1893.

PREFACE.

This little volume is intended as an *aid* to the advanced student of medicine who is preparing for his degree, or to the young practitioner in diagnosing affections or selecting the remedy for them.

The author has endeavored to place in as concise language as possible the *essentials* of the principles and practice of medicine, and in order to do this in few words has been obliged in some instances to make slight sacrifices of grammar at the altar of brevity.

He hopes it will be distinctly understood by the student that this book is not intended to, nor can it, replace the larger text-books in general use.

He dedicates it to the many students who have honored him with their attention during the past fifteen years, and acknowledges his indebtedness to most of the recent works on the Practice of Medicine, and especially on Neurology; and also to his friend, Dr. John M. Eager, recently of Baltimore, who has kindly undertaken the index for him.

In conclusion he would thank the medical critics for the kindness which they have shown him in the past, and invite any suggestions which they or other of his readers may feel disposed to make.

THE AUTHOR.

313 S. Sixteenth St., Philadelphia,

CONTENTS.

	PAGES
INTRODUCTION,	17
Definitions,	17–20
General Symptomatology,	20–25
Pain,	20
Physiognomy of Disease,	21
Pulse,	21–22
Tongue,	22–23
Temperature,	23–25
GENERAL DISEASES.—A. (*Exanthematous and Zymotic Diseases*),	25–98
Fever,	25–26
The Essential Fevers.	26–85
Continued Fevers,	27–47
Simple Continued Fever,	27–28
Catarrhal Fever,	28–30
Typhoid Fever,	30–39
Typhus Fever,	39–42
Relapsing Fever,	42–43
Cerebro-spinal Fever,	43–47
Periodical Fevers,	47–63
Intermittent Fever,	48–50
Remittent Fever,	50–53
Hemorrhagic Malarial Fever,	53–55
Congestive Malarial Fevers,	55–58
Malarial Cachexia,	58–59
Yellow Fever,	59–63
Eruptive Fevers,	63–85
Scarlatina,	63–69
Measles,	69–72
Rubella,	72–73
Smallpox,	73–78

PAGES

Inoculation and Vaccination, ... 78–79
Varicella, ... 80
Erysipelas, ... 81–83
Dengue, ... 84–85
Diphtheria, ... 85–89
Parotitis, ... 89–90
Submaxillitis, ... 90
Pertussis, ... 91–92
Cholera, ... 92–98

General Diseases.—B. (*Diathetic Diseases*), ... 99–123
Acute Rheumatism, ... 99–103
Muscular Rheumatism, ... 103–104
Chronic Rheumatism, ... 104
Rheumatoid Arthritis, ... 105–106
Acute Gout, ... 106-108
Chronic Gout, ... 108
Lithæmia, ... 109
Diabetes, ... 109–112
Polyuria, ... 112-113
Diseases of the Blood, ... 113–123
Anæmia, ... 113–115
Essential Anæmia, ... 115–116
Leukæmia, ... 116–118
Pseudo-leukæmia, ... 118–119
Addison's Disease, ... 119–120
Scorbutus, ... 120–122
Purpura, ... 122–123

Diseases of Special Organs, ... 123–366

Diseases of the Digestive Organs, ... 123–188
Diseases of the Mouth, Fauces and Pharynx, ... 123–130
Stomatitis, ... 123–124
Muguet, ... 125
Glossitis, ... 125–126
Acute Tonsillitis, ... 126–128
Chronic Tonsillitis, ... 128
Angina and Pharyngitis, ... 128–130
Diseases of the Œsophagus, ... 130–133
Œsophagitis, ... 130–131

PAGES

Stricture of the Œsophagus, ... 131–133
Diseases of the Stomach, ... 133–146
Acute Gastritis, ... 133–134
Acute Gastric Catarrh, ... 134–135
Chronic Gastritis, ... 135–137
Gastric Ulcer, ... 137–138
Gastric Cancer, ... 138–140
Hæmatemesis, ... 140–142
Dilated Stomach, ... 142–143
Gastralgia, ... 143–144
Dyspepsia, ... 144–146
Diseases of the Intestinal Tract, ... 146–169
Colic, ... 146–148
Constipation, ... 148–149
Cholera Morbus, ... 149–151
Acute Diarrhœa, ... 151–152
Chronic Diarrhœa, ... 152
Duodenitis, ... 152–153
Catarrhal Enteritis, ... 154
Membranous (Croupous) Enteritis, ... 154–156
Enteritis, ... 156–157
Colitis, ... 157
Acute Dysentery, ... 157–161
Chronic Dysentery, ... 161–162
Typhlitis and Perityphlitis, ... 163–165
Proctitis, ... 165–166
Intestinal Obstruction, ... 166–169
Diseases of the Peritoneum, ... 169–173
Acute Peritonitis, ... 169–172
Chronic Peritonitis, ... 172–173
Diseases of the Liver, ... 173–188
Icterus, ... 173–174
Hepatic Hyperæmia, Acute, ... 174
Chronic, ... 175
Acute Hepatitis, ... 175–176
Chronic Hepatitis, ... 176
Acute Catarrh of the Bile Ducts, ... 176–177
Passage of Gall-stones, ... 178–179
Hepatic Abscess, ... 179–180

PAGES

Interstitial Hepatitis, ... 181–182
Acute Yellow Atrophy, ... 183
Fatty Liver, ... 183–184
Albuminoid Liver, ... 184–185
Cancer of the Liver, ... 185–187
Hydatid Cyst of the Liver, ... 187
Functional Disease of the Liver, ... 188
Diseases of the Pancreas, ... 189–190
Pancreatitis, ... 189
Cysts of the Pancreas, ... 189
Cancer of the Pancreas, ... 190
Diseases of the Spleen, ... 190–192
Acute Affections of the Spleen, ... 190–191
Splenic Hyperæmia, ... 191
Acute Splenitis, ... 191
Splenic Abscess, ... 191–192
Chronic Diseases of the Spleen, ... 192
Diseases of the Kidneys, ... 193–212
Renal Hyperæmia, Acute and Chronic, ... 193–194
Acute Parenchymatous Nephritis, ... 194–196
Chronic Parenchymatous Nephritis, ... 196–198
Chronic Interstitial Nephritis, ... 199–200
Albuminoid Kidney, ... 200–201
Cystic Degeneration of the Kidney, ... 201–202
Passage of Renal Calculi, ... 202–203
Formation of Renal Calculi, ... 203–205
Pyelitis, ... 205–206
Hydronephrosis, ... 206–207
Hæmaturia, ... 207–209
Tubercular Disease of the Kidney, ... 209
Cancer of the Kidney, ... 210
Perinephritis, ... 210–211
Floating Kidney, ... 211–212
Diseases of the Chest, ... 213–305
Physical Diagnosis, ... 213–222
Inspection, ... 213
Palpation, ... 213–214
Mensuration, ... 214
Percussion, ... 214–216
Respiratory Percussion, ... 216

PAGES

Auscultation, ... 216–222
Combination of Physical Signs (Table), ... 222

Diseases of The Respiratory Organs, ... 223–272
Diseases of the Larynx, ... 223–229
Acute Laryngitis, ... 223–225
Chronic Laryngitis, ... 225–226
Croup, ... 226–229
Diseases of the Bronchial Tubes, ... 229–237
Acute Catarrhal Bronchitis, ... 229–231
Acute Plastic Bronchitis, ... 231–232
Capillary Bronchitis, ... 232–234
Chronic Bronchitis, ... 234–235
Asthma 235–237
Diseases of the Lung Tissue, ... 237–265
Emphysema, ... 237–239
Pulmonary Hyperæmia, ... 239–241
Pulmonary Œdema, ... 241
Hæmoptysis, ... 241–244
Acute Lobar Pneumonia, ... 244–250
Broncho-Pneumonia, ... 250–252
Chronic Pneumonia, ... 252
Pulmonary Gangrene, ... 253
Tuberculosis, ... 254–264
Acute Tuberculosis, ... 255–257
Chronic Tuberculosis, ... 257–264
Pneumonic Phthisis, ... 264
Fibroid Phthisis, ... 265
Diseases of the Pleura, ... 266–272
Acute Pleurisy, ... 266–269
Chronic Pleurisy and Empyema, ... 269–271
Pneumothorax, ... 271–272

Diseases of the Circulatory System, ... 273–305
Physical Examination of Heart, ... 273–276
Inspection and Palpation, ... 273
Percussion, ... 274
Auscultation, ... 274–276
Heart Sounds (Table), ... 274
Endocardial Murmurs, ... 274–275
Pericardial Murmurs, ... 275–276

PAGES

Diseases of the Heart, ... 276–297
Cardiac Hypertrophy, ... 276–279
Cardiac Dilatation, ... 279–282
Fatty Heart, ... 282–284
Endocarditis, ... 285–293
Acute Endocarditis, ... 285–288
Chronic Endocarditis and Valvular Disease, . . 288–293
Angina Pectoris, ... 293–294
Exophthalmos, ... 294–295
Cardiac Palpitation, ... 296–297
Diseases of the Pericardium, ... 297–301
Pericarditis, ... 297–301
Diseases of the Vessels, ... 302–305
Thoracic Aneurism, ... 302–305
Diseases of the Nervous System, ... 305–367
Diseases of the Brain, ... 305–330
Cerebral Hyperæmia, ... 305–306
Cerebral Anæmia, ... 306–307
Meningitis, ... 307–309
Tubercular Meningitis, ... 309–310
Apoplexy, ... 311–314
Cerebral Embolism and Thrombosis, ... 314–315
Aphasia, ... 315–316
Acute Cerebral Softening, ... 317–318
Chronic Cerebral Softening, ... 318
Abscess of the Brain, ... 319
Cerebral Tumors, ... 320–321
Cerebral Syphilis, ... 321–322
Acute Hydrocephalus, ... 322–323
Chronic Hydrocephalus, ... 323–324
Insolation, ... 324–327
Delirium Tremens, ... 327–329
Chronic Alcoholism, ... 329–330
Diseases of the Spinal Cord, ... 330–358
Spinal Hyperæmia, ... 330–331
Spinal Meningitis, ... 331–332
Pachymeningitis, ... 332–333
Myelitis, ... 334–335
Spinal Hemorrhage, ... 335–336
Acute Poliomyelitis, ... 336–338

PAGES

Chronic Poliomyelitis, ... 338–340
Spinal Sclerosis, ... 340–342
Antero-lateral Sclerosis, ... 340–341
Posterior Sclerosis, ... 341–342
Disseminated Sclerosis, ... 341–342
Paralysis Agitans, ... 343
Bulbar Paralysis, ... 343–344
Varieties of Tremor, ... 344–345
Writer's Cramp, ... 345–347
Convulsions, ... 347–348
Paralysis, ... 349–352
Tabular View of Paralysis, ... 349–350
Facial Paralysis, ... 351
Syphilitic Paralysis, ... 352
Neurasthenia, ... 352–353
The Neuroses, ... 354–358
Chorea, ... 354–355
Epilepsy, ... 355–358

DISEASES OF THE NERVES, ... 358–367
Neuralgia, ... 358–362
Cerebral Neuralgia, ... 359–360
Neuralgia of the Fifth Pair, ... 360
Cervico-occipital Neuralgia, ... 361
Cervico-brachial Neuralgia, ... 361
Dorso-intercostal Neuralgia, ... 361–362
Intercostal Neuralgia, ... 362
Lumbo-abdominal Neuralgia, ... 362
Sciatica, ... 363
Neuritis, ... 364
Multiple Neuritis, ... 364–367

INDEX, ... 369–374

THE ESSENTIALS

OF THE

PRINCIPLES AND PRACTICE OF MEDICINE.

INTRODUCTION.

What is meant by the principles and practice of medicine?

By this term is meant all the knowledge which has been acquired of the *cause*, *pathology*, *symptoms* and *cure* of disease.

What is pathology?

Pathology relates to the study of disease, or diseased action during life, and is divided into general and special pathology.

What is meant by these terms?

By *general pathology* is meant the study of groups of disease processes, *e. g.*, the pathology of fever. By *special pathology* is meant the disease process occurring in one affection, as the pathology of typhoid fever.

What is pathological anatomy?

Pathological anatomy is the study of diseased processes as they are found after death.

What is pathological chemistry?

This is the study of the changes produced by disease in the excretions, secretions or tissues of the body.

What is disease?

Disease is a deviation from the normal standard of healthy functions, or from the standard tissue changes. A *functional disease* is one in which the tissues do not properly perform their function, but

in which there is no structural alteration to be found. An *organic disease* is one in which the functions are not properly performed, owing to structural lesion of the tissues at fault. Every disease has a local anatomical beginning.

What is meant by the incubation period?

The *incubation period* is that time elapsing between exposure to a disease and the manifestation of the first symptoms. The time during which the disease may be said to lie latent in the system.

What is the prodromic period?

This is the stage of early development of disease, the time elapsing between the incubation period and the full manifestation of diseased action.

What are symptoms?

Symptoms are the language of diseased nature, and may be either objective or subjective. *Objective symptoms* are such as are evident to the senses of the observer. *Subjective symptoms* are such as are felt and complained of by the patient.

How do diseases terminate?

All diseases terminate in health or in death. They may end by (1) *Lysis*, a gradual withdrawal of diseased action; (2) by *Crisis*, a sudden change for better or worse; or (3) by *Metastasis*, a sudden shifting of the disease. The first process is most common; the last is rarely seen.

What is death?

Death is a complete cessation of the bodily functions and of reconstructive tissue change. Death always comes through one of three channels, the *heart*, the *brain* or the *lungs*. It may be due to *asthenia*, or increasing debility with depression of the vital function, to *anæmia*, or an insufficient quantity or quality of the blood, to *apnœa*, or non-aëration of the blood, and consequently of the tissues, or to *coma*, an abolition of the function of the brain.

What is meant by ætiology?

This is the study of the causes producing disease.

How may the causes of diseases be divided?

They may be divided into predisposing and exciting causes. *Pre-*

disposing causes may be *inherited*, or *acquired* by debilitating influences. Our habits, age, sex, occupation, race and previous diseases all act as predisposing causes. *Exciting causes* may be food (too little or too much, or of unhealthy quality), drink (impure water or milk, or other beverages), heat, cold, noxious substances in the atmosphere (such as chemical poisons or disease germs), barometric changes, different electrical conditions, absence of light, or substances or individuals with whom we come in contact producing infectious or contagious diseases.

What is an infectious disease?

An *Infectious Disease* is one which is due to a disease-germ introduced into the body from without, but not capable of being reproduced in the body, hence, not capable of being communicated from one individual to another (*e. g.*, influenza, malaria, tubercle).

What is a contagious disease?

This is a disease due to a specific cause, capable of being reproduced in the economy, and of being transmitted from the sick to the well (*e. g.*, typhus fever, smallpox).

How does contagion spread?

Contagion spreads principally by absolute contact with the poison, and to a slight extent, also, by atmospheric diffusion. The atmosphere, however, dilutes and weakens the poison.

What is meant by a sporadic disease?

A *Sporadic Disease* is one which occurs in isolated cases (*e. g.*, rheumatism).

What is meant by an endemic disease?

A disease is *endemic* when a number of cases occur in a limited locality, due to the same cause (*e. g.*, typhoid fever in a limited locality, from drinking water of a contaminated well).

What is an epidemic disease?

When a disease is widely spread over a community it is said to be *epidemic* (*e. g.*, cholera or smallpox). Epidemics are usually contagious or infectious.

What is diagnosis?

Diagnosis is the discovery of disease by means of its symptoms.

What is differential diagnosis?

Differential Diagnosis is the art of discovering the disease which is present by a careful comparison of its symptoms with those of other diseases which may closely simulate it.

What is prognosis?

Prognosis is the art of foretelling the issue of any disease (it may be *general*, as the general prognosis of typhoid fever, or *special*, as the special prognosis of any given case).

What is meant by treatment?

Treatment is the art of preventing disease, or of taking care of the sick, and alleviating their sufferings, or of aiding and hastening their cure. It may be *Prophylactic* when the aim is to prevent the spread of disease; *Abortive*, where the disease is cut short and prevented from running its regular course; *Palliative*, where it tends to allay suffering; *Expectant*, where the disease is allowed to run its course, the symptoms being treated as they require it; *Restorative*, where it aims to build up the constitution, and restore to the system such materials as may be wanting in the economy (*e. g.*, iron in anæmia, or the phosphates in rickets); or *Radical*, where a rapid impression is made upon the system and the course of a disease cut short.

GENERAL SYMPTOMATOLOGY.

What is meant by general symptomatology?

By *general symptomatology* is meant the study of such symptoms as pain, the expression of the face, the pulse, the tongue and the temperature, not in connection with any special disease, but with diseased processes generally.

Describe the varieties of pain.

(1) *Pain* may be *sharp*, *acute* and *lanceolating* (usually intermittent). This kind of pain is seen in peripheral nervous affections, as neuralgia; also in the inflammations of serous membranes.

(2) It may be *dull*, *gnawing* and more or less *continuous*. This kind of pain is found in chronic tissue changes generally (*e. g.*, in hepatic or splenic affections and in inflammation of the mucous membranes or of bone).

Exception.—In *malignant diseases* (cancer), even when characterized by chronic tissue changes, the pain is sharp.

The character of the pain will vary also according to the tissue involved (*e. g.*, in inflammations of the *skin* it is *burning* and *itching*, in the *bone*, *dull* and *boring*, in *mucous membranes*, *aching*, etc.).

Describe the physiognomy of disease.

Various diseases have their own characteristic physiognomies, which cannot be described accurately, and a knowledge of which can only be acquired by experience (*e. g.*, malaria, cancer, scrofula, tubercle, respiratory and cardiac diseases, and various fevers).

Describe the pulse of disease.

The *normal pulse in adults* varies from 65 to 75 per minute; *in infants*, from 112 to 120; *in old age*, about 60, rising in extreme old age until it approaches that of the infant. It is more rapid in females, more rapid during waking, more rapid in the erect posture, and more rapid during digestion. The *pulse of disease* should be studied with regard to its frequency (rapid or slow), its volume or strength (full or small), its resistance or tension (hard and incompressible, soft and compressible), and its rhythm.

A *moderately rapid pulse* is found in acute sthenic diseases.

A *very rapid pulse*, in marked anæmia, or in low fevers with great debility, therefore it shows great weakness.

The pulse is *very slow* in brain effusions, in disintegration of the cardiac muscle, as the result of great cold, in jaundice and in shock.

It is *full* in health, in states of high arterial tension from any cause (*e. g.*, powerful ventricular contractions).

A *hard pulse* is usually associated with a full pulse, and is found in the early stages of acute diseases.

Exceptions.—(1) The *receding pulse* of certain cardiac diseases is full but not hard. (2) *In atheroma of the vessels*, the pulse is hard because the vessels are rigid, but is not full.

A *small pulse* is usually soft and compressible and is seen in debility.

Exception.—In peritonitis and other inflammations below the diaphragm, the pulse is small but hard and wiry.

Rhythm.—The rhythm is altered in certain brain diseases, as tubercular meningitis; in organic diseases of the cardiac muscles, in func-

tional disturbance from perverted innervation of the heart (as from indigestion), from the abuse of tobacco, tea or alcohol, or from uterine disorders. In children the rhythm may be disturbed from the changes occurring at puberty, or from certain medicines, as quinine.

Describe the appearance of the tongue in disease.

The *tongue in health* is small, smooth, and often slightly coated, particularly in the morning. These characters may be altered by disease of the organ itself or by disturbances of the system at large.

It is *red*, *swollen* and *superficially ulcerated* in acute glossitis, and may be characteristically altered by other local disease, as ulcer, cancer, syphilis, etc. When the tongue is *heavily coated* (due to the accumulation of epithelium) it shows that a similar condition exists in the alimentary canal, especially the stomach and the upper part of the small intestines. This is seen in all acute catarrhs of the alimentary tract. The coating is usually white. It may be yellow in hepatic disorders or it may be artificially colored from food.

It may be *denuded of epithelium*, and studded with prominent papillæ, as in scarlet fever, dysentery, and sometimes in pneumonia.

When the tongue is *very moist* it indicates an increased secretion of the mouth, and generally a similar condition of the upper part of the alimentary tract. It is often *swollen* and *indented* by the teeth. This condition may result from ptyalism, or as a part of a general hyper-secretion of all the glands. It is not a very common condition.

The tongue is *dry* in persons who sleep habitually with their mouths open, as when the nares are obstructed It is also seen when the secretions generally are arrested, which in acute disease shows a condition of lowered vitality, hence a condition of danger. According to Dickinson the tongue is usually dry just before death. In coma, no matter what the cause, the tongue will be found dry. If in the course of a *chronic disease* the tongue should be *dry*, sugar will probably be found in the urine.

A *livid tongue* is seen when the circulation is much interfered with.

It is *extremely red* in great irritation of the stomach and bowels,

but may be red only at the tip and edges, the remainder of the tongue being concealed by heavy coating.

A *glazed tongue, smooth and glossy, with prominent papillæ*, discolored, often brownish, and frequently cracked or fissured, is only seen in cases of great prostration. The glazed appearance is due to fungi developed between and on the papilla.

A *fissured tongue*, if neither glazed nor brown, indicates nothing of importance, frequently being seen in perfectly healthy individuals.

How is the temperature of the body ascertained?

The *temperature* is taken by a clinical thermometer, placed either in the mouth, axilla or rectum, the mouth being the most convenient, the axilla the most accurate. A self-registering thermometer is used, and should be gently waved to and fro by swinging the arm, in order to set the index. It is then placed in the axilla carefully, in such a manner that it is in contact with the skin on all sides, and is allowed to remain for at least six minutes, when it is removed and the temperature read. It should be taken at least twice daily.

What is the normal temperature of the body?

The *average temperature* is 98.6°. There may be a *normal variation* of from .5° above to .5° below the average, which is compatible with perfect health. These variations are even greater in children. The *temperature of the mouth* is about .5° lower, that of the rectum 1.° higher, than the axillary temperature. There is a certain relation between the temperature and the pulse, the latter increasing about ten beats per minute for every 1° rise in temperature above the normal.

Are greater variations than the above compatible with life?

The temperature in fever may rise to 107° with recovery (rare). A temperature of 111° has been observed in cerebral rheumatism, the patient recovering (very rare). Mr. Teale has reported a case of injury to the nervous system which recovered, in which the temperature rose to 117°. Another case has been reported, of 120° (?), with recovery. When the temperature is very high the thermometer is usually inaccurate, or the patient is playing some trick with the instrument (malingering cases). In hysterical patients under

great excitement the temperature may be very high for a short time.

The temperature is *depressed* in debility (97°). After severe cholera morbus, or in cholera collapse, it may even be as low as 92°. In the insane it sometimes falls to 80°, or even below it.

What temperature indicates a fever?

A temperature of over 100° is a febrile temperature. From 100° to 101° is a slight fever; 101° to 103° a decided fever; 103° to 105° a high fever; when over 105° it indicates great danger.

What is meant by the period of defervescence?

The decline of the fever is thus spoken of. The temperature then often falls below the normal.

Do special diseases have characteristic temperatures?

Certain diseases have peculiar temperature records which are characteristic of them, *e.g.*, typhoid fever, measles, etc. In cancerous affections the temperature is below the normal. In all cases where the tubercular process is active the temperature is above the normal.

Is the temperature of any prognostic value?

In fevers a steady or falling temperature is a favorable sign, while a rising temperature indicates danger. If it remains high when it should fall it is a bad sign. In acute tuberculosis when the fever subsides the active process is arrested.

What therapeutic indications may be deduced from the study of the temperature?

A high temperature calls for antipyretics. A low demands stimulants and tonics.

Is the thermometer ever used to take the local temperature of individual parts of the body?

As increased tissue change is accompanied by elevation of temperature, which will be higher in the neighborhood where it is produced, the thermometer over an inflamed part will indicate a greater elevation of temperature than over other parts of the body. For the purpose of taking the surface temperature, thermometers are used the bulbs of which are arranged in a flattened coil. The

surface temperature is always several degrees lower than that of the axilla, due to evaporation and the radiation of heat. The temperature of the head and abdomen is nearly 3° lower, and that of the chest 2° lower than that of the axilla.

GENERAL DISEASES.—(A.)EXANTHEMATOUS AND ZYMOTIC DISEASES.

Fever.

What are the characteristics of a fever?

Fevers are characterized by (1) an *elevation of temperature;*

(2) *Increased rapidity of tissue change;*

(3) As a rule *increased rapidity of the circulation*, and

(4) *Alteration in the secretions*, which are usually diminished.

What are the general indications for their treatment?

(1) *Reduce the temperature* by cold sponging, cold baths, quinine (gr. xx), or if the temperature is very high, by antipyretics, as antipyrine.

(2) Give *easily digested food* in small amounts frequently repeated, to counterbalance the waste of the tissue.

(3) *Regulate the circulation* by alcohol, ammonia, or perhaps digitalis, if the pulse is small, weak and rapid, or by aconite, veratrum viride, or antimony, if the pulse is full and strong.

(4) *Keep up the secretions* by diuretics, diaphoretics or purgatives, as the case may require.

Mention some of the general laws governing fevers.

(1) They are all due to *special poisons.*

(2) They are mostly *contagious* or *infectious.*

(3) They are mostly *self-limited diseases*, *i. e.*, they run a definite course and terminate spontaneously.

(4) *One attack* usually *protects* the individual from a recurrence of the disease.

(5) With few exceptions there are *no specifics.*

(6) With few exceptions fevers have *no characteristic pathological lesions.*

What is meant by fever diet?

Fever diet consists in giving the patient plenty of milk, arrowroot or broth, therefore a light, easily digested, fluid diet *every two or three hours, day and night.* If milk alone is used the patient can take from three to five pints in twenty-four hours.

What is meant by fever treatment?

Fever treatment consists in sponging off the body of the patient under the bed-clothes with cool water, three or four times a day, keeping him lightly covered, the room well ventilated, and its temperature from 68° to 70°. He should be given plenty of cooling drinks, in small quantities, from fear of overloading his stomach, but frequently repeated, and he should be coaxed to take them. The secretions must be kept up by diuretics, diaphoretics or purgatives, as indicated.

The Essential Fevers.

Have the essential fevers definite periods of incubation?

The average period of incubation is *less than a week* in *catarrhal fever, cerebro-spinal fever, dengue, diphtheria, erysipelas, scarlatina* and *relapsing fever.*

It is *two weeks* in *smallpox.*

Two to *three weeks* in *typhoid fever.*

One to *four weeks* in *malarial fever.*

Twelve hours to *twelve days* in *typhus fever.*

Twenty-four hours to *twenty-four days* in *yellow fever.*

Eight to *twenty-eight days* in *measles.*

What are the days on which the eruptions of the essential fevers appear?

The eruptions appear on the *second day* in *scarlet fever* and *varicella.*

During the *third day* in *smallpox* and *rötheln.*

On the *fourth day* in *varioloid* and *measles.*

On the *fifth day* in *typhus fever.*

From the *fifth to the seventh day* in *dengue.*

From the *seventh to the ninth day* in *typhoid fever,* and is *early* or *uncertain* in *cerebro-spinal fever.*

How are the essential fevers classified?

They are divided, according to their chief characteristics, into the continued, the periodical, and the eruptive fevers.

CONTINUED FEVERS.

What are continued fevers?

Fevers of the continued type are those of which the chief characteristic is a temperature more or less continuous during the duration of the disease. They are *simple continued fever*, *influenza*, *typhoid fever*, *typhus fever*, *relapsing fever*, and *cerebro-spinal fever*.

Simple Continued Fever.

What is simple continued fever?

It is a short febrile disease without any very obvious cause, and having no constant pathological lesion.

What are the synonyms of simple continued fever?

It is called essential fever, ephemeral fever, irritative fever, febricula, ardent fever, synocha, etc.

What is its duration?

It lasts from four to seven days, and frequently terminates by a crisis.

What is its cause?

It is often due to fatigue, especially in children; to mental strain or anxiety in adults, or to exposure to heat.

What are its symptoms?

It begins with a chill, followed by fever and gastric disorder, lasting for a few days, and ending by a critical perspiration or diarrhœa. In adults, after mental anxiety or exposure to heat, there is often severe headache and even delirium.

How is this disease diagnosed?

The *diagnosis* is made by the cause, by the symptoms, the duration and the termination.

(1) From *cerebral softening* it is known by the cause and duration.

(2) From *typhoid fever*, by the absence of sufficient intestinal symptoms, by the absence of the eruption, and by the duration.

What is the prognosis?

The *prognosis* is favorable, the disease always ending in recovery.

How should it be treated?

It should be *treated* by rest in bed; fever treatment and diet; acting on the skin with diaphoretics, and giving quinine and tonics as convalescence approaches.

Catarrhal Fever.

What is catarrhal fever?

It is an acute epidemic disease, characterized by fever with catarrh, severe pains and great nervous depression.

What are the synonyms of this disease?

It is called epidemic catarrhal fever, epidemic contagious catarrh, influenza, dandy fever, la grippe, la coquette, la folette, etc.

Is this a new disease?

It is not. Epidemics of influenza were reported in the fifteenth century, and perhaps even prior to this period. It occurs in marked epidemics, sweeping rapidly over continents, about every twenty-five or thirty years, with numerous minor epidemics, more or less limited in extent, occurring in the intervals.

What is its cause?

It is due to a special atmospheric poison, probably a germ, and is infectious rather than contagious. If contagious it is but feebly so.

What are the symptoms of this disease?

It begins suddenly with chilly sensations, rapidly developed, with general hyperæsthesia of the surface, and severe shooting neuralgic pains all over the body, but particularly in the back and limbs. There is fever (100° to 103°), with catarrh of the nose, eyes, fauces, pharynx, larynx, and sometimes of the bronchial tubes; a strange, irritative, laryngeal cough, worse at night, and singular nervous depression, out of proportion to the local symptoms. It sometimes

ends abruptly by a critical discharge from the bowels or skin, and often leaves the patient much debilitated, with a feeble heart, with laryngeal cough, or with a lingering catarrh of the mucous passages.

What is the duration of influenza?

It lasts from three to ten days, often followed by a tedious convalescence.

What are its complications?

(1) *Pneumonia* occurs in about four per cent. of the cases. It is more frequent in some epidemics than in others, and may be either catarrhal or (not so commonly) croupous. It is the most frequent complication.

(2) *Gastro-intestinal Catarrh.*—In some epidemics the alimentary canal bears the brunt of the disease. This complication is seen in a small number of cases in any epidemic, and is occasionally very severe.

(3) A *cerebral complication*, resembling meningitis, characterized by severe headache, photophobia and violent delirium, lasting four or five days, is occasionally seen, but is not a common complication. It would appear to be due rather to an irritation of the brain resulting from the poison in the blood than from any definite lesion.

(4) *Stiffness of the joints*, without inflammation, lasting four or five days, is occasionally met with.

How may this disease be diagnosed?

It is recognized by being epidemic, by the fever with catarrh, by the hyperæsthesia and the pains, by the great nervous depression, and by the complications.

What is the prognosis of influenza?

It is one of the least fatal of diseases in itself, but owing to the great depression it induces, the death-rate is frightfully increased during its prevalence, for, when it attacks old persons, or people debilitated from any cause whatsoever (as teething children, or those affected by chronic cardiac diseases, or chronic bronchial or pulmonary diseases), it proves fatal.

How should this disease be treated?

The patient should be confined to bed and ordinary fever treatment and diet administered. Diaphoretics (as neutral mixture and

sweet spirits of nitre) should be freely given, and the bowels regulated. He should take quinine in tonic doses, from the commencement of the disease far into convalescence. Under no circumstances should the treatment ever be of a depressant nature. If any complication is present, or if there is much exhaustion, stimulants should be freely used; and, during convalescence, iron, quinine and other tonics must be administered.

How should special symptoms be treated?

If the *cough* is severe small doses of Dover's powder (gr. iij–v) may be given at night and repeated as necessary, or inhalations of compound tincture of benzoin in hot water may be used at bedtime, or paregoric or minute doses of morphine administered.

If the *stomach* is irritable, small doses of calomel with sodium bicarbonate or cocaine may be taken; or minute doses of morphine injected hypodermically.

If the *neuralgic pains* are very severe, antipyrine with quinine may be administered in capsules or solution.

For the *nasal catarrh*, a solution of cocaine may be sprayed or inserted on cotton into the nostrils, or the tincture of iodine inhaled from a wide-mouthed bottle.

Should *delirium*, *sleeplessness*, etc., be present and opium seem to aggravate rather than relieve it, urethan, chloral or the bromides may be given instead of Dover's powder.

Bronchitis and *pneumonia* should be treated as if no other disease were present, great care being taken not to depress the patient.

Should the *cardiac action* be weak, rest should be enjoined far into convalescence, and digitalis, strophantus or strychnine conjoined with the tonic treatment.

Typhoid Fever.

What is typhoid fever?

It is an acute infectious fever, due to a special poison characterized by an insidious beginning, a characteristic temperature record, characteristic eruption, peculiar intestinal symptoms with diarrhœa, and having characteristic post-mortem appearances.

What are its synonyms?

It is called enteric fever, gastro-intestinal fever, infantile remittent fever, slow nervous fever, autumnal fever, abdominal typhus, etc. etc.

What are the causes of typhoid fever?

It is due to a specific poison, probably never generated *de novo*, but favored in its development by decomposing animal matter, particularly the alvine dejections, as may occur in foul drains. The poison may be propagated by drinking water, or milk contaminated with it. It attacks persons in the prime of life.

What are the incubation period and duration?

The *incubation period* is about two weeks, sometimes three weeks or longer. The *duration* is three and a half weeks, followed by a tedious convalescence.

What are the symptoms of this disease?

(1) *Prodromic Period.*—It comes on gradually, with chilly sensations, malaise, perhaps some diarrhœa, the patient being feverish toward night, and in about half of the cases having irregular attacks of epistaxis; all this lasting from three days to a week.

(2) *The First Week of the Disease.*—The fever then becomes continuous, with evening exacerbations and morning remissions. Diarrhœa, if not present before, now comes on. There is great lassitude, headache, a soft, frequent pulse, and often nocturnal delirium.

(3) *Second Week.*—These symptoms all increase, an eruption appears, the patient is often in a condition of stupor, alternating with hallucinations, the tongue is dry, and the fever more continuous.

(4) *Third Week.*—If the case is doing well the symptoms now begin to abate. There is not so much delirium or headache, and there are marked variations in the fever, the temperature being much lower in the morning, but high at night.

If the case is not doing well the stupor deepens, the diarrhœa increases, there is picking at the bed-clothes, subsultus tendinum, the patient has a tendency to sink down in the bed, and he dies from exhaustion, or from some of the complications which may occur at this time.

(5) *Fourth Week.*—About the middle of the fourth week the fever ceases, the evening temperature being normal, and he enters upon a tedious convalescence, some irritability of the bowels remaining.

SPECIAL SYMPTOMS.

FEVER SYMPTOMS.—*Temperature* is characteristic. Commencing at 99° the first night it gradually ascends, being about 1° higher each evening than on the preceding evening, but declining nearly a degree in the morning from the temperature of the preceding night. This continues until the evening of the fifth to seventh day of the disease, when the temperature has reached 103°–104°. It then remains stationary, with a slight remission and evening exacerbation, until the thirteenth or fourteenth day, when it begins to decline, coming down every morning 1°, 1½° or even 2°, but going up again in the evening nearly as high as the night before. This continues until from the twenty-first to the twenty-fifth day, the evening temperature being normal. It occasionally happens (very rarely) in persons who have been subjected to great exhaustion and extreme nervous depression before they contracted the disease, that typhoid fever will run its course without any elevation of temperature whatever.

The *pulse* is small, soft, and compressible. It is frequent rather than rapid. Should it be very rapid it would indicate great debility.

The *heart* muscle is very weak, from granular degeneration. The first sound of the heart is consequently indistinct, and in bad cases may be entirely absent.

INTESTINAL SYMPTOMS.

Diarrhœa, if not present from the first, generally comes on by the end of the first week. There are three, four, or often many more large, fluid and very offensive stools, yellowish or brownish in color, resembling pea soup in consistence. In some epidemics the diarrhœa is not present, or only to a slight extent. Persons suffering from hernia frequently pass through the disease without diarrhœa.

Tympanites.—This comes on in the second week usually, and is often excessive.

Gurgling on pressure over the right iliac fossa is usually present in the second week, but is a symptom of little importance.

The *spleen* is found by palpation to be enlarged, softened, and tender, on touch.

The *tongue* is covered with a light yellow fur at first, becoming dry, devoid of epithelium, and glazed in the second week. Its tips and edges first lose the coating, giving rise to a peculiar V-shaped redness on the otherwise heavily coated tongue. If the tongue cracks, bleeds, and becomes covered with a dull varnish, it is a bad sign.

Sordes appear on the teeth, gums, lips and roof of mouth, and consist of collections of dried mucus, epithelium, bacteria, and sometimes blood.

ERUPTION.—This is only absent in about 12 per cent. of the cases. When present it is the most strongly diagnostic symptom. It appears from the seventh to the ninth day, in the form of a few rose-colored, slightly elevated spots, like flea-bites, generally on the abdomen chest or back, sometimes on the limbs, but never on the face. They disappear on pressure, and during convalescence, but reappear if a relapse takes place. They come in crops of four or five in number, lasting three or four days. They are not present after death.

The *face* is flushed, having a dull, stupid expression, and the eyes are heavy and listlesss.

There is *pain in the right iliac fossa*, griping in character, with *tenderness* on pressure in the same region.

The *urine* is deficient in chlorides, and, in bad cases, contains albumin, which is not necessarily indicative of renal disease.

Râles are heard over the chest, especially posteriorly, and are due to hypostatic congestion of the lungs, and effusion into the bronchi.

Ridges are found on the nails during convalescence, and if a relapse takes place it will be indicated by a second ridge.

NERVOUS SYMPTOMS.

The *headache* is very intense, being replaced by *stupor* in the second week.

Delirium is generally present in the second week, if not earlier. It is usually of a low muttering type. Occasionally fierce delirium is seen, in which patients usually manifest a great desire to jump out of the window.

The special senses often suffer, hearing being generally obtunded, and vision perverted.

Muscular System.—There is great muscular weakness, from granular degeneration.

What is the pathology of this disease?

Peyer's patches and the *solitary glands* become infiltrated by poison, and swell during the first ten days or thereabouts. Inflammation ensues, and from the tenth day ulceration occurs, and perforation of one or more of the coats of the bowels may result. The ulceration continues from the eighteenth to the twenty-fifth day, after which cicatrization takes place, and the ulcers are generally healed in six weeks. Cicatrization may occupy a longer time.

What is the pathological anatomy of typhoid fever?

Intestines.—Infiltration, inflammation and ulceration (the ulcers being in the longitudinal axis of the bowels) of the *solitary glands* and *Peyer's patches;* often more or less complete perforation, and partial cicatrization is found after death. The *mesenteric glands* are enlarged and swollen, but do not ulcerate. The *spleen* is always large and soft.

The *lungs* are congested, especially at the lower part, but pneumonia is rare.

The *heart* and other muscles have undergone granular degeneration.

What are the complications?

(1) *Hypostatic congestion of the lungs* is often present, to a greater or less degree, but pneumonia is rare.

(2) *Intestinal hemorrhage* from ulceration of a vessel of the bowel. If in the second week the temperature suddenly falls to or below the normal, it will indicate a hemorrhage from the bowel, even before the appearance of the blood in the stool.

(3) *Perforation with peritonitis* is generally fatal. The symptoms of this complication are sudden, severe pain in the right iliac fossa, rapidly followed by symptoms of peritonitis and collapse.

What are the sequelæ of typhoid fever?

(1) *Anæmia*, from an impoverished condition of the blood.

(2) *Dropsy*, usually from blood changes, but which may result from renal disease.

(3) *Phlegmasia dolens* (*milk-leg*).—This is due to phlebitis of

one of the larger veins, and is a tedious, but not dangerous complication.

(4) *Paralysis*, usually of the lower extremities, and probably due to spinal anæmia. The electro-muscular reactions are impaired. It is tedious, but not dangerous, and may be foretold by a tremor occurring during convalescence in the part about to be affected.

(5) *Dysentery.*—If in the third week the diarrhœa passes into dysentery, the large bowel has become ulcerated, and the prognosis is not so favorable.

(6) *Periostitis.*

(7) *Formation of abscesses*, and various other sequelæ indicating a lowered vitality and an impoverished condition of the blood.

Describe the period of convalescence.

Convalescence is always slow. The patient is always greatly emaciated, and the bowels remain irritable. If there is any latent disease in the system, as phthisis, cancer or scrofula, it may develop during this period.

Relapses are not uncommon, and are probably due to a further absorption of the poison which has remained dormant in the bowels. During a relapse all the symptoms, as eruption, fever, etc., return. The disease, however, runs a more rapid course, and usually ends favorably.

What is meant by abortive, and by walking typhoid?

Cases of abortive typhoid are usually, but not necessarily, mild. The eruption appears earlier, the disease runs a more rapid course, and ends in about two weeks.

Walking typhoid is usually milder in the beginning, but as the patient continues to attend to his usual business, and takes no care of himself, they are generally serious cases, and often terminate fatally.

How is typhoid fever diagnosed?

The *diagnosis* is formed from the history of the gradual beginning, from the characteristic temperature record, from the characteristic eruption, and from the peculiar intestinal symptoms, and nervous phenomena.

DIFFERENTIAL DIAGNOSIS.

From (1) *Simple Continued Fever.*—This disease ends about the

time the typhoid fever is beginning. There are no intestinal symptoms, no eruption, and no characteristic temperature record.

(2) *The Typhoid Condition in Other Diseases.*—In these cases the history, the absence of the eruption, of the characteristic intestinal symptoms, and of characteristic temperature, should render the diagnosis plain.

(3) *General Debility.*—The same remarks apply to this as to the preceding condition.

(4) *Acute Tuberculosis.*—Here we have an irregular fever, absence of eruption, presence of bacillus in the sputum, and the physical signs of the disease.

(5) *Cerebro-spinal fever* can only be mistaken for typhoid when the acute stage of the former disease is passed, and the patient has entered into a low asthenic state. But the history, the difference of the eruption, the absence of intestinal symptoms, and the presence of rigidity or stiffness of the muscles of the neck, would indicate the nature of the attack.

(6) *Typhus.*—This disease is differentiated by the sudden beginning, the difference in the temperature and eruptions, the constipation, and the greater prominence of the nervous system.

(7) *Meningitis.*—Cases of typhoid fever with severe headache and active delirium may simulate meningitis; but in the latter disease the pulse is hard, full and tense; there is no eruption, no diarrhœa, but causeless vomiting, with a clean tongue.

What is the mortality and prognosis in typhoid fever?

In hospital practice the *mortality* is about 18 per cent.; in private practice 10 to 12 per cent.

The earlier it is treated the better the *prognosis.* When a case runs a typical course the prognosis is favorable. All complications increase the danger. Unfavorable prognostic symptoms are a very high temperature, a pulse over 120, subsultus tendinum, picking at the bed-clothes (*carpologia*), and sinking down in the bed.

What is the treatment of typhoid fever?

(1) Prophylactic Treatment.

Disinfect the stools, water closets, etc., most thoroughly, with corrosive sublimate or other germicide.

(2) General Treatment.

Give fever treatment and diet, with special attention to the liquid diet. Good hygiene (cleanliness, cool air, thorough ventilation); good nursing; diluted nitro-hydrochloric or other mineral acids (gtt. x–xx, freely diluted, every two to four hours), or carbolic acid with tincture of iodine; stimulants generally in the second week, and treat any symptoms that may require it.

(3) Treatment of Special Symptoms.

(*a*) If the *fever is very high* antipyrine or large doses of quinine may be given, or cold baths or a wet pack employed if there is no tendency to intestinal hemorrhage.

(*b*) *Profuse Diarrhœa.*—If there are more than three or four stools a day, and the patient's strength is failing, opium should be given conjoined with bismuth (gr. x–xx), alone or combined with carbolic acid (gtt. j); or the sulphate of copper, nitrate of silver, acetate of lead, or other mineral astringent may be used with the opium.

(*c*) If there be *excessive tympanites*, turpentine injections (gtt. x; tr. opii deodor., gtt. j–v; in gum water every three hours), or turpentine stupes and turpentine internally, or injections of vinegar and water, should be employed. Nux vomica or strychnine, given internally, are often of service.

(*d*) If there be *much gastric disorder*, which is sometimes seen early in the case, especially in malarial districts, calomel (gr. v–x) will often give relief.

(*e*) Should there be *great oppression in breathing*, turpentine stupes and turpentine internally, with frequent change in the patient's position, and, in severe cases, dry cups to the chest, should be used.

(*f*) If the *heart is very weak* the patient should be stimulated with alcohol, strychnine, strophantus and quinine. During the height of the fever digitalis is not efficient as a cardiac stimulant. It is better to reserve it until convalescence.

(*g*) For *restlessness and sleeplessness* urethan (gr. xx repeated once), opium (gr. j of the extract in suppository), or morphine (gr. $\frac{1}{6}$ to $\frac{1}{8}$, hypodermically) should be given at night, and repeated if necessary. Musk is also good in these cases. Chloral, if used, should be given with care, because of the cardiac depression.

(*h*) If the *delirium* is excessive, the same treatment may be employed, but should be given during the day as well as night. Great delirium calls for an increased amount of stimulants. If it is associated with high temperature and all else fail, cold baths may be tried.

(*i*) The *headache* usually passes away without treatment. If it should continue and be excessive, the head may be shaved and ice applied to the scalp. Occasionally, if the circulation is languid, hot water bags give a better result. Should this treatment produce no result, morphine (gr. $\frac{1}{6}$) may be used hypodermically, or a blister applied to the scalp.

(*j*) For *stupor and coma* free stimulation must be employed, and caffeine or cocaine given.

(*k*) Great *debility or exhaustion*, which generally occurs in the second week, calls for the use of stimulants, the amount being regulated by its effect on the pulse and on the first sounds of the heart. As long as the pulse becomes steadier and less frequent, and the first sound of the heart more distinct, the amount of alcohol is not in excess of the requirements of the system. Usually not more than half a pint of brandy per day is required. Stimulate especially in the early morning hours, and nourish the patient well. Caffeine or cocaine (gr. $\frac{1}{6}$ every two hours) may be used as heart stimulants.

(*l*) *Subsultus tendinum* calls for chloral and opium; or better still, if there is much restlessness, camphor, asafœtida, musk, or spirits of chloroform, may be used.

(4) Treatment of Complications.

(*a*) For *hypostatic congestion* of the lungs. Frequent change of position, turpentine internally, turpentine stupes, and dry cups are the best means of combating this complication.

(*b*) For *intestinal hemorrhage* give less food, and, if possible, less stimulant; keep the bowels absolutely at rest by opium, and give ergot hypodermically and internally every half hour to an hour, until the blood disappears from the stools; or the oil of erigeron, or Monsel's solution well diluted, may be alternated with the ergot, or acetate of lead may be given. In some instances ice applied to the abdomen, or bandaging the abdomen as tightly as possible, appears to exert a favorable influence.

(*c*) For *perforation with peritonitis* give as little food as possible. The amount of stimulants must be regulated by the amount of collapse. The patient must be kept continually under the influence of large doses of opium, a great tolerance for which drug is present in these cases.

(*d*) All *other complications* should be treated on general principles.

Typhus Fever.

What is typhus fever?

It is an acute contagious disease due to a special poison, characterized by a sudden beginning with peculiar temperature record, a characteristic eruption, and constipation. No constant lesion is found after death.

SYNONYMS.

Jail fever, camp fever, ship fever, hospital fever, brain fever, or spotted fever.

CAUSE.

It is due to a specific poison, originating in or cultivated by filth or overcrowding. It occur in epidemics, and is very contagious.

INCUBATION PERIOD.

Twelve hours to twelve days.

DURATION.

About two weeks.

What are the symptoms of the disease?

It begins suddenly with a chill, high fever from the start (the temperature rising, however, until the eruption appears, then remaining stationary until the tenth day, when a slight remission occurs), grave nervous symptoms, a flushed, dull and muddy looking face, and costive bowels. It ends by a crisis about the fourteenth day.

SPECIAL SYMPTOMS.

(1) *Fever Symptoms.*—There is a chill, followed by fever, the temperature being high immediately, but continuing to rise until the second or third day, when it has reached 104°. It continues at

this height, with slight morning remissions, and evening exacerbations, until the tenth day, when there is a more marked remission; but it soon rises again, and continues high until the fourteenth day, when it falls rapidly to the normal, or even below it.

(2) *Eruption.*—The eruption appears on the fifth day of the disease, and covers the entire body except the face. It is scarcely influenced by pressure, and consists of coarse measly blotches, which soon become darker and purplish, and often petechial. It remains after death, but fades during convalescence.

(3) *Nervous Symptoms.*—These are like those of typhoid fever, but there is more headache, more marked delirium, often fierce in character in the early part of the disease, and more stupor.

What are the complications of typhus fever?

(1) *Pneumonia*, much more frequent than in typhoid.

(2) *Albuminuria.*—Renal complications are more frequent than in typhoid fever, and may result in uræmia.

(3) *Diarrhœa.*

(4) *Dysentery.*

(5) *Erysipelas.*

(6) *Parotitis.*—This is a dangerous complication, death frequently occurring from pressure of the enlarged glands on the jugular vein. Therefore, suppuration should be encouraged, and the pus evacuated as early as possible.

(7) *Phlegmasia dolens*, and other complications (not intestinal), such as occur in typhoid and other low fevers.

Describe the morbid anatomy in typhus fever.

The *blood* is dark and diffluent, and the *spleen* enlarged. No constant lesion is found.

How is this disease diagnosed?

It is known by its beginning suddenly with a high temperature record, a characteristic eruption, absence of intestinal symptoms, and grave nervous phenomena.

DIFFERENTIAL DIAGNOSIS.

(1) *From typhoid fever* by the sudden beginning, the temperature, the eruption, the constipation, and the duration.

(2) *From measles;* there is no eruption on the face, no catarrhal

symptoms, a different temperature record, and more marked nervous symptoms.

(3) *From meningitis* (sometimes, also, called brain fever), by the history the eruption, the rapid, compressible pulse, the coated tongue, and the absence of vomiting as a prominent symptom.

(4) *From relapsing fever*, by the eruption, the different temperature record, the different history, and the absence of spirillæ in the blood.

What is the prognosis and mortality of this disease?

The *mortality* is twenty-five per cent. The older the patient the more unfavorable the case. When an epidemic attacks armies it is very fatal. *Unfavorable symptoms* are a pulse above 120°; very profuse eruption; contracted pupils; sleeplessness, and very high temperature. When the pulse remains very rapid during convalescence it indicates the development of tubercle in the lungs.

What is the treatment of typhus fever?

(1) Prophylactic Treatment.

Thorough disinfection, ventilation and isolation.

(2) General Treatment.

Fever treatment and diet, with less attention, however, to a fluid diet than in typhoid fever. Regulate the bowels with calomel, podophyllin and salines. Give mineral acids, as in typhoid fever.

(3) Treatment of Special Symptoms.

Headache.—Leeches, and warm or cold applications to the head. Opium may be given if the pupil is not contracted, or bromides and chloral may be resorted to.

Sleeplessness.—For this symptom anti-spasmodics, as musk and camphor, are of service. Opium may be given, but not too frequently. Urethan, bromides and chloral are also of use.

For stupor and coma, stimulants, caffeine and cocaine are usually indicated.

(4) Treatment of Complications.

Every complication should be treated as if it were the only disease present. Pay special attention to the bladder, and be sure that retention of urine does not occur.

Pneumonia.—Stimulants and quinine are here indicated; and for the oppression in breathing the free use of turpentine internally and externally affords most relief.

Relapsing Fever.

What is relapsing fever?

It is an acute epidemic disease, usually limited to the seaboard towns in this country, characterized by a sudden beginning, with chills, fever, headache, muscular pains, vomiting and jaundice, ending suddenly in a week, and having a great tendency to recur.

SYNONYMS.

Februs recurrens, mild yellow fever, bilious relapsing fever, famine fever, seven days fever, etc.

CAUSE.

It is a contagious disease, occurring in epidemics due to the presence of a peculiar spiro-bacterium (the spirochæte of Ehrenberg) in the blood. These organisms are rarely absent during an attack, and are in constant motion at this time. The disease spreads more rapidly under bad hygienic conditions and in times of famine.

What are the symptoms of this disease?

It begins suddenly with a chill, followed by fever, violent headache and great muscular pains. After a few days vomiting and epigastric tenderness occur, and are prominent symptoms. Soon jaundice takes place, with hepatic and splenic tenderness, the fever increases and the pains in the limbs become more severe. This lasts for five to seven days, when suddenly the fever terminates, and all the symptoms pass away as rapidly as they came on. The patient is now apparently well, and remains so for a week, but generally on the fourteenth day from the commencement of the fever the pains and other symptoms recur, and the patient suffers a relapse as severe as the primary attack, but of shorter duration. After three or four days the disease again terminates, and the patient remains well for one or two weeks, when another relapse may occur.

PROGNOSIS AND MORTALITY.

The *prognosis* is favorable. The *mortality* in private practice is from 2 to 4 per cent.

MORBID ANATOMY.

The *spleen* is found invariably enlarged, and the *white blood corpuscles* are increased in number, and *spirochæte* are found in the blood.

COMPLICATIONS.

Pleurisy and *pneumonia* are the most frequent complications.

DIAGNOSIS.

The disease is known by the presence of an epidemic, by the sudden high fever, the jaundice and other hepatic symptoms, the short duration, abrupt termination, the relapse and the presence of spirochæte in the blood.

How is this disease treated?

(1) PROPHYLACTIC TREATMENT.

Thorough ventilation, disinfection and isolation.

(2) GENERAL TREATMENT.

Fever treatment and diet, rest in bed during the attacks and between them, if possible. Keep the bowels open with calomel and salines. Give diuretics, diaphoretics, and quinine, as a tonic.

(3) TREATMENT OF COMPLICATIONS.

Treat every complication as if it were the only disease present. Nourish the patient well.

Cerebro-spinal Fever.

What is cerebro-spinal fever?

It is an acute infectious fever, due to a special poison characterized by a sudden beginning, with intense headache and delirium, contraction of the muscles of the back, moderate fever, irregular pulse, frequently a petechial eruption, usually followed by grave sequelæ and having characteristic post-mortem appearances.

What are the synonyms of this disease?

It is also called cerebro-spinal meningitis, cerebro-spinal typhus, and spotted fever.

What is its causation?

It is due to a specific poison which is said to be most malignant

during wars, and to be found following the route of armies. It is rarely seen in summer—occurs in epidemics, and is not contagious.

What is its incubation period and duration?

The *period of incubation* is about one week. *It lasts* about a week, but the convalescence may be protracted for many months.

Describe this disease.

It begins suddenly with a chill, followed by intense vertigo and violent headache, vomiting, fever, petechiæ, irregular pulse, hyperæsthesia of the skin, and rigidity with spasmodic contractions of the muscles of the back. Delirium, stupor, and in bad cases intense restlessness, coma and death.

Deviations of Type (all of which may be followed by the same sequelæ) :—

(1) *The Fulminating Form.*—These cases occur in the beginning of the epidemic. They run a rapid and violent course, death occurring usually in twenty-four hours, from the intensity of the poison acting on the nervous system.

(2) *The Abortive Form.*—These are mild cases where the patient is not confined to bed, but suffers from obstinate rigidity of the muscles of the back of the neck, with intense headache and inability to apply the mind to any of his ordinary occupations which require thought.

(3) *The Hysterical Form.*—In this variety the delirium is of an emotional kind, resembling hysteria. Partial paralysis may take place, with stiffness of the muscles of the neck.

Special Symptoms :—

(1) *Premonitory Symptoms.*—A sense of weight in the back of the head and neck, with pain on pressure in the same region.

(2) *The eruption* is not characteristic. It may come early (a bad sign) or irregularly, and is found all over the body and, later in the disease, on the face also. It is not influenced by pressure and consists in petechial spots of extravasated blood, like purpura. It is absent in about half the cases.

(3) *The temperature* is usually not over 102°, except in the fulminating form, when it may be very high. It is apt to intermit or remit.

(4) *The pulse* is very irregular, and often does not correspond to the apex beat. It varies very much in twenty-four hours.

(5) *Vertigo* is often one of the first symptoms. It is very severe, but grows gradually less as the disease progresses.

(6) *The headache* is fearful, the patient complaining of it as long as he is conscious.

(7) *The delirium* is generally fierce and active, and is rarely absent. The restlessness is so great that the patient constantly wants to get out of bed and leave the house. Sometimes the delirium simulates hysteria.

(8) *Spinal Symptoms.*—There is shooting pain along the spine, and retraction of the muscles, as in tetanus. Clonic and tonic contractions occur, and hyperæsthesia of the surface is present.

(9) *Convalescence.*—If the patient is about to recover, about the ninth or tenth day the delirium ceases; there is less difficulty in swallowing; the stiffness lessens, and the patient slowly enters upon a tedious convalescence, passing into a low typhoid condition, which lasts many months. During convalescence the sequelæ usually appear.

What are the complications and sequelæ?

(1) *Pneumonia.*—The lung symptoms may be so severe as to mask those of the disease, should the fever be mild in character.

(2) *Joint affections* are sometimes seen, resembling acute articular rheumatism.

The Sequelæ are:—

(1) *Blindness* in one or both eyes.

(2) *Deafness.*

(3) *Idiocy.*

(4) *Epilepsy.*

(5) *Local or general paralysis.*

The first three are usually incurable. Epilepsy occasionally improves under treatment. Paralysis gives a more favorable prognosis.

What is the morbid anatomy of this fever?

There is an inflammation and exudation of plastic lymph on the *meninges of the brain and spinal cord*, the pia mater and arachnoid being especially affected. The exudation occurs into the ventricles, but particularly at the base of the brain, in the subarachnoid spaces,

hence the sequelæ. The pia mater is congested and adherent to the cord.

The *lungs* are engorged, and spots of catarrhal pneumonia are usually present.

How is cerebro-spinal fever diagnosed?

This disease is known by the presence of an epidemic, by the sudden onset, by the violent vertigo, the retraction of the head, the delirium, eruption, irregular pulse and temperature, and the various sequelæ.

(1) *From congestive fever* it is differentiated by the spinal symptoms, eruption, absence of remission, and the difference in the temperature.

(2) *From spinal meningitis*, by the cerebral symptoms and eruption.

(3) *From typhus fever*, by the spinal symptoms, the difference in eruption, which is sometimes absent, and which appears on the face.

(4) *From tubercular meningitis*, by the sudden onset without prodromes, and by affecting adults as well as children.

(5) *From typhoid fever;* no mistake is possible until the acute symptoms have subsided and the patient passes into a low typhoid condition. In these cases the history, the rigidity of the muscles of the neck, the absence of intestinal symptoms, the difference in eruption, and the sequelæ would indicate the nature of the disease.

What is the prognosis and mortality?

The *mortality* is very high, varying with the epidemic from twenty to seventy-five per cent. It is greatest in the early part of the epidemic. The disease is not so fatal among children. The least fatal age is from two to five years, the most fatal from eighteen to twenty-five.

How is this disease treated?

Give the patient fever treatment and diet.

Keep the head cool and the body warm.

Act on the skin with small doses of pilocarpine, and keep the bowels open with calomel, podophyllin and salines.

In the early stages give large doses of opium—enough to keep the patient under its influence—either alone or alternating with potas-

sium bromide. (The latter acts well in children when used alone.) Chloral may also be given with the same object in view (*i. e.*, to depress the reflex centres and diminish the amount of blood in the brain). Ergot may be used to contract the blood-vessels. *Locally*, local depletion by cup or leeches may be employed if the patient is young and robust, followed by ice to his head and spine. *In the latter stages* combine potassium iodide with the other treatment, apply warmth to the head, and blisters to the back of the neck and spine, to cause absorption of the exudate. *During convalescence* give quinine in tonic doses, feed the patient well, and stimulate if the heart is weak.

The complications and sequelæ should be treated on general principles.

PERIODICAL FEVERS.

Name the periodical fevers.

The fevers of this group are *intermittent*, *remittent*, *hemorrhagic malarial*, *congestive*, *typho-malarial*, and *yellow fevers*. All except the last are due to *malaria*.

What is malaria?

Malaria is a poison which is generated by heat and moisture. It is most likely due to microscopic germs of either vegetable (bacillus malariæ) or animal (plasmodium malariæ) origin. The latter view has been steadily gaining ground of late.

There can be no malaria without heat and moisture. It is generated in marshes where vegetable decomposition is going on, or in newly turned up earth, where the land has long lain fallow. It is said that it may be developed from the decomposition of rock. *It accumulates in low moist districts* and at the bases of mountains. *It drifts in the direction of prevailing winds.* It would appear to be a heavy poison, for it rarely rises higher than five hundred feet above the sea level, and trees growing in the vicinity of swamps appear to arrest its spread. *Certain trees*, as the eucalyptus, *absorb and destroy the poison. Water also absorbs it*, and salt water probably destroys it. Persons drinking fresh water impregnated with the poison are apt to become infected and suffer from malarial dysentery. It disappears under good drainage and the cultivation of the soil.

It enters our bodies with our food and drink, or through the air. Poisoning is most apt to take place *in the early morning hours or at night.* The poison is more readily absorbed during fasting than during digestion.

Malarial poison is the same all over the world, and manifests itself by its periodicity.

The period of incubation is very uncertain, usually from three to four weeks; although in some instances but a few hours have elapsed between the exposure and the development of the sickness.

Intermittent Fever.

What is intermittent fever?

Intermittent fever is a fever due to malaria, characterized by attacks of distinct febrile paroxysms, recurring periodically, with complete intervening intermission.

Each paroxysm consists of three stages—a cold stage, a hot stage, and a sweating stage. The length of the interval between paroxysms (called the apyrexia) gives the name to the type of the disease.

What are the types of intermittent fever?

Intermittent fever is of a *quotidian type* when the apyrexia lasts twenty-four hours, the attack recurring each day.

It is *tertian* when the apyrexia lasts forty-eight hours, the attack recurring every other day.

It is *quartan* when the apyrexia lasts seventy-two hours, the attack recurring every third day.

A *double tertian* is really a quotidian, but every other day the attacks are more severe.

When two attacks occur in one day it is spoken of as a *duplicated quotidian.*

Quotidian and *tertian intermittents* are the most common varieties in this part of the country.

What is the duration of this fever?

If not treated after ten or twelve paroxysms, the type may undergo a change, the fever becoming more continuous and gradually subsiding, to recur in a few weeks or months.

What are its symptoms?

(1) *Premonitory Symptoms.*—Preceding an attack, the patient

often complains of pain in the back and limbs, increased salivary secretion and a frequent tendency to yawn.

(2) *The symptoms of the cold stage* are, a chill, with blue nails, pale skin (the so-called *goose-flesh*), frequent urination, shivering and intense thirst. During this time a thermometer in the axilla will show that the internal temperature of the body is from 104° to 106°. This stage lasts from half an hour to two hours, and is followed by the *hot stage.*

(3) *The symptoms of the hot stage* are subjective and objective heat of surface. There is a full bounding pulse, flushed face, severe headache, nausea and vomiting. The axillary temperature rises to 105° or 108°. This stage may last up to four hours, and is followed by the *sweating stage.*

(4) *Sweating stage;* perspiration now breaks out, first on the brow and face, then over the whole body. The secretions are re-established. The headache disappears, the nausea subsides, the pulse falls, and the temperature goes down 1° or 2° every five or ten minutes, until it is below the normal, when the attack is over and the patient is well until the next paroxysm.

The *urine* is acid during the paroxysms, is often alkaline during the intermission, and may contain albumen.

How is this disease diagnosed?

This disease is recognized by the history of malarial exposure, by the stages, the high temperature, the complete intermission, and the recurring paroxysms.

(1) *From hysteria* it is differentiated by the characteristic temperature, the stages, the complete intermission, and the recurrence of the paroxysms. During an hysterical chill the temperature is sometimes very high, but only for a short time, with great and irregular oscillations.

(2) *From hectic fever* it is diagnosed by a physical examination, which shows the absence of cause for the hectic. In the latter disease, also, the pulse remains high in the intermission, and the chills usually occur in the afternoon.

(3) *From urethral or syphilitic fever* by the absence of the necessary history, and the fact that the temperature is much higher than in the latter diseases.

What is the treatment of intermittent fever?

(1) *If a chill is impending* and there is not time for the action of quinine, it may be warded off by a hypodermic injection of morphine (gr. $\frac{1}{6}$), or of pilocarpine (gr. $\frac{1}{8}$).

(2) *Treatment of the Cold Stage.*—Keep the patient as warm as possible by hot drinks, by hot applications externally, and by warm covering.

(3) *Of the Hot Stage.*—Fever treatment, cold drinks and ice, cold sponging to the surface, and a fever mixture with a little aconite if the fever runs high, are indicated. Antipyrine will reduce the temperature, but does not shorten the disease.

(4) *Of the Sweating Stage.*—During this stage do nothing except when it is prolonged and exhaustive, in which case the body may be sponged with alum or tannin in solution; and a few drops of chloroform in mucilage and water, or a minute dose of atropine may be given.

(5) *Of the Intermission.*—Purge the patient with calomel, followed by a saline to unload the portal circulation, if there is time. After this administer quinine (gr. xx–xxiv in 24 hours, in solution or freshly made pills, which should not be sugar coated) in two decided doses, the last from four to six hours before the expected paroxysm. If for any reason quinine cannot be given, cinchonidine or some other alkaloid of cinchona, may be substituted. Should these not be available, arsenious acid or Fowler's solution may be substituted. In obstinate cases Fowler's solution, with strychnine and iron, or small doses of quinine with whiskey early in the morning, may be successful. Vapor baths also are very valuable in these cases.

Remittent Fever.

What is remittent fever?

It is a fever due to malaria, characterized by periodical exacerbations and remissions, the patient never being free from fever until the attack terminates. It is found all over the United States, particularly in the South and Southwest, in Africa and parts of Europe, as Italy, Hungary, etc. It attacks men more frequently than

women, and children but rarely suffer from it. The black race is nearly exempt.

Each exacerbation at first consists of three stages (*vide* intermittent fever), but later on, the cold stage disappears.

It may be *quotidian*, *tertian* or *quartan*, but is usually quotidian.

What are the synonyms of this disease?

It is called bilious fever, bilious remittent fever, marsh fever, jungle fever, Roman fever, etc.

What are its symptoms?

There is a chill, at first recurring daily with the exacerbations, followed by quickened pulse and respiration, flushed face, intense headache, pains in the back and limbs, like yellow fever and smallpox, great gastric disturbance, a temperature of 105° to 107°, sometimes delirium, and frequently jaundice. All this lasts from 10 to 18 hours, when the sweating stage takes place, during which the temperature is rapidly lowered some 3° or 4° (but not to the normal); and a remission occurs in which the patient feels well, but the fever, although less, still continues.

After a time the exacerbation sets in again, and the symptoms of the first paroxysm are repeated.

After three or four paroxysms the chill disappears, and the fever becomes more continuous, with morning remissions (as a rule) and evening exacerbations. If the disease is not treated and the patient is about to recover, the fever gradually passes away in two or three weeks, or may end as an intermittent fever, or the patient may pass into a low typhoid condition known as *typho-malaria*.

The pulse is full, bounding, rapid and a little tense during the paroxysms.

The tongue is coated with a heavy yellowish fur.

Nausea and vomiting, or at all events gastric irritability, are usually present, and are sometimes very severe.

The bowels are generally constipated, sometimes stubborn diarrhœa is seen, and in rare cases the stools are dark and offensive. Toward the end of the attack there are black, tarry passages from the bowels, due to altered bile.

Jaundice is a frequent symptom.

The headache is usually severe, generally disappearing in the remission, but may be a prominent symptom throughout the attack.

Delirium is often present.

What are the sequelæ and morbid anatomy of remittent fever?

SEQUELÆ.

(1) *Persistent headache.*

(2) *Dropsy, and persistent anæmia.*

(3) *Malarial cachexia.*

MORBID ANATOMY.

The *heart* is softened and granular. The *blood* contains pigment, and the corpuscles of Laveran are usually present.

The mucous membrane of the *stomach* is softened and congested.

Bruner's glands are enlarged and of a dark slaty color, due to the deposition of pigment.

The *liver* is olive-colored or bronze.

The *spleen* is enlarged and slate-colored.

What is the diagnosis and prognosis of this disease?

DIAGNOSIS.

This disease is known by the history of malarial exposure, the paroxysms of fever, the constipation, remission, black, tarry stools, the frequent jaundice, and the recurrence of the paroxysms.

(1) *From intermittent fever* it is known by remitting, but never intermitting;

(2) *From jaundice* by the high fever and rapid pulse;

(3) *From meningitis* by the remissions, highly-coated tongue, pulse, high temperature, and jaundice; and

(4) *From typhoid fever* by the more marked remissions, the jaundice, constipation and absence of eruption.

PROGNOSIS.

Formerly this was a very fatal disease, and even now, under appropriate treatment, two per cent. die.

What is the treatment of remittent fever?

(1) GENERAL TREATMENT.

Fever treatment and diet (light, easily digested food, fluid in character) are called for, and, as a rule, stimulation must be avoided.

Quinine (gr. xx–xxx per diem) may be given at any time, or in any stage, without waiting for the remission; although, if of a quotidian type, the bulk of the remedy is preferably administered during this time. It is best to precede the administration of quinine by a mercurial purge, if there is time enough for it to act; otherwise do not wait for the purge before giving the quinine.

If the fever is not broken after six or seven days, reduce the amount of quinine one-half and continue the laxatives, treating the symptoms as they arise.

(2) Treatment for Special Symptoms.

For *irritable stomach* give calomel (gr. ij–v, with sodium bicarbonate), followed by a saline, as Rochelle salts. If this does not check it, give smaller doses of calomel (gr. $\frac{1}{8}$–$\frac{1}{5}$), with minute doses of morphine. Let the patient swallow small pieces of ice, or drink effervescent mineral waters; or employ small doses of cocaine hypodermically. As a last resort a blister may be applied to the epigastrium.

If the fever is high, with a bounding pulse, intense headache, etc., add tincture of aconite (gtt. j) to the fever mixture, every two or three hours. Apply cold to the head, use mustard foot baths, and mustard plasters to the nape of the neck, or give antipyrine.

If the head symptoms are very severe, and are not benefited by other means, local blood-letting may be employed.

Hemorrhagic Malarial Fever.

What is hemorrhagic malarial fever?

It is a remittent fever, of peculiar type, characterized by sudden jaundice and bloody urine, occurring just before the remission, and depending on the intensity of the malarial poison, which probably acts on the solar plexus of the sympathetic nervous system.

It is found especially in the South and Southwest. It is becoming a more common disease, and appears to be spreading over larger areas of territory. It is occasionally seen on the eastern shores of Maryland also.

Synonym.

It is sometimes called Yellow Disease.

Describe this disease.

It begins like an ordinary remittent fever, but the symptoms are more marked from the onset—more severe.

(1) There is a *chill*, which may be repeated in the second or third paroxysms, and is much more severe than in ordinary remittent fever.

(2) There is high *fever*, with severe pains, vomiting, headache, delirium, etc., as in ordinary remittent fever, but much more severe.

(3) This is followed by a *sweating stage*, at the beginning of which the patient suddenly becomes deeply jaundiced, and hemorrhages occur from the gums, fauces, lungs, stomach (rare), bowels (also rare), but especially from the kidneys, and continue until the remission, during which they disappear until the succeeding paroxysm. As the paroxysms continue to recur the renal hemorrhage becomes continuous, and does not intermit as at first. The urine under the microscope is seen to contain blood corpuscles (many of which are altered in form), hæmatoglobin (from decomposition of blood corpuscles) and blood casts. The urine is of a uniform black color. The blood is not coagulated, and no more albumen is found than can be accounted for by the presence of the blood. After from six to twenty-four hours the remission occurs and lasts a variable time, depending upon whether the disease is of the quotidian, tertian or quartan type. The disease may end fatally, the patient dying of exhaustion, or from anæmia due to the hemorrhages. More frequently, however, death results from uræmia, caused by the renal congestion.

What is the prognosis and mortality?

Although a very dangerous disease, it is very amenable to treatment. If not treated about two-thirds die. If not treated early half the cases die. If treated from the beginning not more than 10 or 15 per cent. die.

What is found on post-mortem examination?

The *liver*, *spleen* and *kidneys* are congested, the latter containing blood casts. The *blood* itself is dark and diffluent.

How is hemorrhagic malaria recognized?

This disease is known by its occurring in malarial districts, by

beginning as a severe remittent fever, by the early jaundice, the hemorrhage, the remission, and the return of the paroxysm.

(1) *From yellow fever* it is differentiated by the malarial exposure, by the high temperature, by the abrupt jaundice and bloody urine (rare in yellow fever) at the end of the paroxysm, by the recurrence and by the presence of no more albumen than can be readily accounted for by the blood.

(2) *From intermittent hæmaturia;* this disease is rare in this country, and consists of occasional hæmaturia without fever.

How should it be treated?

The *treatment* does not differ materially from that of remittent fever, except in—

(1) *The earlier, freer* and *more determined use of quinine* (giving gr. xxx–xl per day, not waiting for the stage of remission, but administering the remedy as soon as the patient is seized with the disease).

(2) *Keeping up the secretion of urine* during the disease and following convalescence (by washing out the kidneys with mild diuretic mineral waters, as Vichy, etc., or flaxseed tea and plenty of water. It should always be remembered that acute Bright's disease may supervene).

(3) *By treating the hemorrhage* with diluted sulphuric acid (gtt. v–x every hour, continued during the remission until the urine loses its smoky appearance. Ergot may be used for the same purpose, or tincture of the chloride of iron (not so good). Gallic acid, gr. x–xxx, repeated at short intervals, is much used in Louisiana).

(4) *By giving, during convalescence, a long-continued course of mineral acids, with tincture of ferric chloride* (or Basham's mixture), *and quinine in tonic doses*, given steadily and kept up for some time.

Congestive Malarial Fever.

What is this disease?

It is a fever due to a profound affection of the nerve centres by a concentrated malarial poison, and is characterized by symptoms of internal congestion. It is also called *Pernicious Malarial Fever.*

What are the varieties of this disease?

The principal *varieties* found in this country are the *cerebral*, the *pulmonary or thoracic*, and the *gastro-intestinal forms*, depending on the seat of the congestion. Another variety, the *sweating form*, is occasionally met with, but occurs especially in Italy.

Any of the varieties may be of the *quotidian*, *tertian*, or *quartan type*. They are all characterized by the *hot*, *cold*, and *sweating stages*, as are the other forms of malaria.

What are the symptoms of this fever?

It is never congested from the beginning, but starts as an ordinary remittent or intermittent fever, the symptoms, however, being much more severe than usual. In the second, third or fourth paroxysm, usually in the cold stage, it becomes congestive. In all the varieties there is marked prolonged chill, the surface being cold, but the internal temperature 106° to 108°. There is intense longing for fresh air, and great anxiety. This lasts for several hours, is followed by reaction and increased fever, followed in its turn by sweating, and a remission or intermission; or the cold stage or the hot stage may be followed by exhaustion, perhaps by death.

The symptoms of internal congestion come on usually in the cold stage, last through the hot stage, and pass away in the sweating stage, to recur with the next paroxysm.

In the cerebral form, in addition to the common symptoms, there is violent headache, fierce, active delirium, and coma.

In the pulmonary form there is gasping for breath, cough, and sputa tinged with blood. On percussion the resonance is impaired; on auscultation the vesicular murmur is almost suppressed, and râles are heard over both sides of the chest, with other symptoms and signs indicative of congestion of the lungs.

In the gastro-intestinal form there is violent vomiting and purging, often rice-water stools, and collapse with a cold surface, like Asiatic cholera.

In the sweating form the sweat is so severe and protracted that the patient appears to be wasting away before the eyes.

After an intermission or remission all the symptoms return, with increased severity, and this is repeated until the patient dies or the attack is broken up.

What is the diagnosis and prognosis?

The *diagnosis* is easy; the disease begins as an ordinary intermittent fever, symptoms of internal congestion being superadded to the third paroxysm.

PROGNOSIS.

The first paroxysm is not very dangerous. In the second, death usually occurs in the cold stage. A third almost always terminates fatally.

If treated from the beginning the prognosis is favorable; if not, it is bad.

How should it be treated?

(1) *To Prevent a Threatening Paroxysm.*—If the patient has had a severe chill and another is impending, morphine should be administered hypodermically at once, followed by pilocarpine. This will lessen the severity, if it does not arrest the approaching chill.

(2) *If not seen until the chill*, endeavor to bring on the reaction by frictions with hot turpentine, cayenne pepper, or hot brandy. Friction should be applied to the whole surface, but particularly to the spine.

In the cerebral form dry or wet cups may be applied to the nape of the neck, or even general blood-letting resorted to.

In the pulmonary variety wet cups or dry cups followed by hot turpentine fomentation should be applied to the chest.

In the gastro-intestinal variety dry or wet cups, or leeches should be used externally, and opium should be given internally, combined with blue mass, or with acetate of lead, camphor and capsicum.

(3) *When reaction takes place* and the fever is high, the temperature should be reduced by antipyrine and cold sponging, and the circulation regulated by aconite.

In the sweating variety atropine should be administered, and the surface sponged with whiskey and alum or other astringents.

(4) *To prevent recurrence* quinine should be given as soon as the patient is seen, preferably in solution and by the mouth, if not contraindicated, giving at least gr. xl before the next paroxysm. If there is much gastric irritability it may be given in suppositories by the rectum in double the dose; or if the bowels are also irritable, the tartrate, lactate or hydrobromate of quinine, or the muriate of

quinine and urea, may be injected hypodermically, the dose being half as large as when given by the mouth. In children the oleate of quinine may be applied with friction to the skin of the abdomen, thighs and axilla. The quinine should be continued for several weeks in small doses, the amount being increased at the septenary periods.

Malarial Cachexia.

Describe this condition and its treatment.

This occurs—

(1) After repeated attacks of any kind of malarial fever, or—

(2) After a long residence in a malarial district, in persons who have not suffered from an attack of fever, but who have gradually absorbed the poison.

SYMPTOMS.

Malaise, listlessness, attacks of dyspepsia, more or less acute, and sometimes slight jaundice, high-colored urine, malarial anæmia, and sometimes albuminuria (which may even prove fatal), sleepless or restless nights, and although there is no actual outbreak of fever, yet there is a certain periodicity manifested by the symptoms, as shown by their liability to recur at stated times. All this may last for months, and is particularly apt to recur in the Spring and Fall. The spleen is often much enlarged, and the patient presents a cachectic appearance difficult to describe, but easy of recognition.

POST-MORTEM EXAMINATION.

The *liver* and *spleen* are pigmented, the latter being sometimes enormously enlarged (so-called *ague cake*), glistens on section, and later undergoes waxy degeneration. The *blood* is deteriorated and contains pigment.

TREATMENT.

Change of climate, or at least change of scene for a time (mountains or seashore), is very essential. When this cannot be done, or as an adjunct, the secretions should be regulated by occasional saline laxatives, light diuretics, as Vichy or Poland waters, and the secretion of the skin kept up by systematic exercise and Turkish baths.

Antiperiodics.—The system is generally saturated with quinine, but small doses of it (gr. ij–iv) may be given on rising in the

morning, conjoined with Fowler's solution (gtt. ij–iv t. i. d.), after meals. Iron and strychnine are also of use.

For the ague cake, ergot should be administered hypodermically (in doses of ♏ xx, every second day), or internally (especially in children). This is better than the treatment by iodine.

Yellow Fever.

What is yellow fever?

It is an acute, infectious epidemic disease, due to a special poison, consisting of one paroxysm of three stages, characterized by an abrupt beginning, with severe pains, albumen in the urine, remission and jaundice, black vomit and collapse.

It is found in hot, low climates, as the West Indies, South America, the southern parts of the United States, Gibraltar, the Mediterranean, Siam, etc.

It is also called the Yellow Jack, El vomito, Typhus icterodes, Mediterranean Fever, etc.

What is its cause?

It is caused by a specific poison, not malarial in nature, but probably a vegetable germ. It is epidemic, is very feebly contagious from the bodies of the sick, but spreads with great rapidity through merchandise, clothes, hair, etc. It does not seem to spread through infected stools, or water. It attacks strangers especially, and is self-protective. It is spread, not caused, by bad hygiene; is destroyed by frost, or by heat exceeding 225°.

What is the incubation period and duration?

The *incubation period* is from twenty-four hours to twenty-four days. The *duration* is from three to nine days, the average being seven.

Describe a case of yellow fever.

It consists of three stages: first, *the fever stage*, lasting from a few hours to three days; second, *the remission or lull*, from twenty-four to thirty-six hours; third, *the stage of collapse and exhaustion.*

(1) *Febrile stage;* begins abruptly, with a chill, usually at night. There is headache, fever, brilliant eye, severe supraorbital pains,

and pains in the sockets of the eyes, back and joints. Nausea, great thirst, and albumen in the urine.

The muscular strength is well preserved, and the mind remains clear. Sometimes the patient is very restless, which is a bad symptom. After lasting from a few hours to a few days these symptoms all subside, and are followed by—

(2) *The stage of remission or lull*, during which the patient is perfectly well, except for a little gastric irritability and for the jaundice which now appears. The disease may end here, or pass into the third stage after twenty-four to thirty-six hours, never longer.

(3) *The stage of exhaustion or collapse;* the tongue and skin become dry; there is high fever; irregular respiration; constant vomiting, often of blood (the black vomit); hemorrhages from the nose, and sometimes from the bowels; occasionally petechial spots on the skin; the jaundice deepens; and, though recovery may take place, collapse and speedy death generally follow.

Deviations of Type.

(1) *Inflammatory cases* are those marked by high fever and severe symptoms in the first stage.

(2) *Congestive cases* are those in which there is congestion of the internal organs, with early and grave prostration.

(3) *Walking cases* are those in which the patient does not feel sufficiently ill to go to bed, but continues about his ordinary occupations, thus overtaxing a heart which is weakened by granular and fatty degeneration, and which usually speedily gives out.

Special Symptoms.

The pulse is rarely above 100, irregular, and often below the normal in the remission; it usually rises in the stage of collapse, although it may be either slow or rapid. It is very small and weak in bad cases.

The temperature in the first stage is usually from 100° to 102°, but may be as high as 105° or 106°, especially in inflammatory cases. It falls during the remission, but rises in the stage of collapse.

Black Vomit.—This consists of blood altered by the abnormal secretions of the stomach. It is found also in the intestines, and sometimes in the cavity of the pericardium. It generally appears in the third stage, after the skin becomes dry. It lasts until death

supervenes, although it is not invariably a fatal symptom. The vomited matters come up without effort. Bleeding may occur from any mucous surface or may take place under the skin, therefore there is a hemorrhagic tendency. Black vomit is sometimes seen in other diseases, as in typhus fever, the plague, etc.

Jaundice.—The skin is of a peculiar orange color. This is not true jaundice, but is due to disintegrated blood, as well as to bile in the circulation. It may be absent, and when present appears slowly at the end of the first and during the second stage.

The *urine* contains albumen, even in the first stage. This may account for the coma and convulsions which are sometimes seen.

Post-mortem Examination.

The *heart* is granular.

The *stomach* and *upper part of the intestines* are congested, their mucous membranes are softened, and ecchymotic spots of extravasated blood are formed on their surfaces.

The *liver* is yellow, having undergone acute fatty degeneration.

The *kidneys* are congested and granular.

Disintegrated blood is found in the alimentary canal, pericardium, under the skin, and sometimes in other cavities of the body.

How may this disease be diagnosed?

Yellow fever is known by being epidemic, by consisting of one paroxysm of three stages, by beginning suddenly, with severe pains, albuminuria, remission, jaundice, black vomit and collapse.

Differential Diagnosis.

(1) *From intermittent fever* it is known by being epidemic, by the albuminuria, by the different temperature records, and by its consisting of one paroxysm.

(2) *From remittent fever*, by its being epidemic, of shorter duration and consisting of one paroxysm. By the presence of albuminuria from the beginning, by the severe pains, by the absence of muscular prostration and by the black vomit.

(3) *From hemorrhagic malarial fever*, by its being epidemic; by the difference in the temperature record; by the slow supervention of jaundice at the end of the first stage; by the early albuminuria; by the black vomit and the absence of hemorrhage from the kidneys, and by the fact that the paroxysm does not recur.

(4) *From typhoid fever;* a mistake is only possible should the patient pass into the typhoid state during convalescence. The history of an abrupt beginning without intestinal symptoms or eruption; a knowledge of the epidemic, and the difference in the temperature record, should render the diagnosis clear.

What is the prognosis and mortality of yellow fever?

In *hospital practice* the mortality is high, being from 30 to 50 per cent., while in *private practice* only 10 to 12 per cent. die.

Walking cases are very dangerous. A very high temperature indicates a bad case.

How should this disease be treated?

(1) PREVENTIVE TREATMENT.

As this disease spreads by human intercourse, merchandise, clothes, etc., care should be taken, *first*, to destroy all clothing which comes in contact with the disease, or, if it cannot be destroyed, to disinfect it thoroughly and afterward to boil it. This is better than quarantine. *Second*, to disinfect the bodies of the sick, as it appears to be contagious from bodily contact; and *third*, to subject all vessels coming from an infected port to the action of superheated steam, as heat above 225° destroys the poison.

(2) GENERAL TREATMENT.

The patient should have plenty of fresh air and cooling drinks. Absolute repose in the recumbent position should be insisted upon, on account of the granular degeneration of the heart.

Diet.—During the first stage he should be given as little food as possible, and that little should be very bland. During the second stage the diet should still be somewhat restricted in very small amounts, and very easily digested. In the stage of collapse he should be fed frequently, and if the stomach is too irritable to retain the food, it should be given per rectum. Stimulants are often necessary in this stage.

Laxatives.—These should be given in the early stages, and continued in small doses throughout the disease.

Diaphoretics, as neutral mixture, with small doses of morphine, should be freely administered during the first stage.

The latest treatment consists in giving small doses of corrosive

sublimate with bicarbonate of sodium, in large quantities of water, frequently repeated.

(3) Treatment for Special Symptoms.

If the temperature is very high in the early stages, sponging with ice water, or other cold applications to the surface, is indicated.

If there is much nausea or vomiting in the early stages, carbonated waters, ice, cocaine, or a few drops of chloroform in iced gum-water, with mustard plasters to the epigastrium, is the best treatment.

For the black vomit tincture of ferric chloride (gtt. v-x on shaven ice every third hour), or Monsel's solution, or cocaine, do good.

As the kidneys are congested, plenty of cooling drinks, as cream of tartar lemonade, should be given throughout the disease, to prevent urinary suppression.

ERUPTIVE FEVERS.

Name the eruptive fevers.

The eruptive fevers are scarlatina, measles, rubella, smallpox, varicella, erysipelas and dengue.

Eruptive Fevers are all characterized by—

(1) Having an eruption.

(2) By being self-limited; by running a definite course.

(3) By being self-exhaustive and self-protective.

(4) By being contagious, and—

(5) By attacking children especially, with the exceptions of smallpox, erysipelas and dengue.

Scarlatina.

What is scarlatina?

Scarlatina is an acute fever, due to a special poison, characterized by a high temperature, a rapid pulse, a peculiar eruption, and a tendency to involve the throat.

It is found all over the world, independent of climate, although most prevalent in the cold and temperate regions. It appears in

epidemics, but is always present in cold climates, especially in winter.

The first epidemic is described about the middle of the sixth century.

SYNONYM.

Scarlet fever.

What is the cause of this disease?

It is due to a specific poison, which is highly contagious, and which resides partly in the breath, partly in the secretions, but more especially in the skin of the patient. The desquamated scales are highly contagious, and, clinging to the walls of rooms or to clothing, spread the disease. The disease is also inoculable by the blood or serum of the sick to the well. No special bacterium has been found.

What are its symptoms and varieties?

Scarlet fever begins suddenly with vomiting (sometimes with a chill, sometimes with convulsions). The pulse is very rapid from the start, ranging from 130 to 180, and the fever very high. At the end of twenty-four hours an eruption appears on the face and neck, sometimes on the breast, and spreads all over the body in twenty-four hours more. The vomiting now lessens and stops, and sore throat (if not present earlier in the disease) now appears and becomes a prominent symptom. Nocturnal delirium is also present. The disease remains at its height for about one week, when the eruption begins to fade and desquamation occurs. The fever now subsides, but does not entirely disappear before the third week. The other symptoms, however, abate, and the patient is convalescent.

VARIETIES.

(1) *Scarlatina Simplex.*—This is the variety just described.

(2) *Scarlatina Anginosa.*—In this variety the symptoms are similar to those of scarlatina simplex, but the throat symptoms appear earlier and are more marked. The tonsils and cervical glands are swollen, a grayish-white membrane is found on the throat, the mucous membrane of which is ulcerated, the breath is offensive, and frequently the thyroid gland is enlarged.

(3) *Scarlatina Maligna* is the gravest form of the disease, the

nervous symptoms predominating. There is delirium and recurrent convulsions, very high fever (even as high as 115°); the throat symptoms are generally, but not necessarily, severe; the eruption is irregular, often late in appearing, is livid, uninfluenced by pressure, and often interspersed with petechial spots.

(4) *Scarlatina Latens* is a variety with no marked symptoms; the eruption may or may not be present, and the sore throat, of which the patient generally complains, may be very slight. It is, however, liable to the same complications and sequelæ as are the most severe cases.

Special Symptoms.

(1) *The eruption* comes out at the end of the first twenty-four hours, and appears first on the face and neck, sometimes on the breast, as a bright scarlet rash. Appearing at first in minute dots, it soon becomes confluent, and spreads all over the body in twenty-four hours after its first appearance. It is most marked around the joints, and on close examination in these situations, minute spots are seen a little raised above the surface, and more vividly red than the surrounding skin. It disappears on pressure, except in malignant cases, when it is of a dusky or livid hue. It is associated with swelling of the skin, and is due primarily to congestion of the cutaneous capillaries. The superficial layers of the skin, when examined under the microscope, are seen to be infiltrated with embryonic cells, and superficial hemorrhages are found in the glandular structure of the mucous membranes. The eruption remains at its height for from three to five days, and then gradually fades in the order of its appearance.

(2) *Desquamation* comes on when the eruption fades, and occurs first on the face, afterward on the body, in large flakes. It may continue for weeks. As long as it lasts so long is there danger of contagion from the patient.

(3) *Throat Symptoms.*—These have already been described when speaking of the anginose varieties.

(4) *Fever Symptoms.—The pulse* is very rapid. This is so early a symptom that causeless vomiting, and a pulse of 130 to 160 per minute, in a child, should lead to the suspicion of this disease, even before the eruption appears. The pulse remains frequent during convalescence.

The temperature rises to a great height early in the case, and remains high even after the eruption appears. It falls when the eruption fades, but while the morning temperature may be normal in the second week, the evening temperature will remain high, and it is not until the third week that it reaches the normal.

What are the sequelæ of this disease?

(1) Inflammation of the glands of the neck.

(2) Enlarged tonsils and chronic sore throat.

(3) Post-nasal catarrh. (These three sequelæ are hard to cure.)

(4) Chorea.

(5) Scarlatinal rheumatism.

(6) Valvular disease of the heart. (These three sequelæ may appear long after the disease has subsided.)

(7) Otorrhœa, and deafness, from extension of the inflammation from the throat into the ear. It may give rise to pyæmia from absorption of pus, or to inflammation and abscess of the brain.

(8) Ulceration of the cornea.

(9) Chronic diarrhœa. (Peyer's patches, and the solitary glands are often enlarged.)

(10) Scrofulous throat affection.

(11) Persistent anæmia, with glandular enlargement.

(12) Inflammation of any of the serous membranes.

(13) Tuberculosis.

(14) Acute Bright's disease. This comes on as the disease disappears, about the tenth or twelfth day, rarely after the third week. Albumen is found in the urine in small amounts during desquamation in any bad case, but if *inflammation* of the kidneys supervenes, a large amount of albumen is found, and the urine will be scanty, high colored, and of a smoky appearance, from the blood which it contains. Under the microscope an abundance of red blood corpuscles and tube casts containing blood and desquamated epithelium are found. As the acute stage subsides, the secretion of urine becomes freer, the amount of blood diminishes, and then dropsy, which is rare in scarlet fever from any other cause, may come on, and rapidly increase to an enormous extent.

How is scarlet fever recognized?

It is recognized by the eruption appearing at the end of twenty-

four hours, and preceded by vomiting, high fever, and very rapid pulse. Should the patient not be seen until later, the course, desquamation and the marked sequelæ, would aid the diagnosis.

(1) *From roseola* it is diagnosed by the fever, the sore throat, and the eruption, which does not come and go as in the latter disease.

(2) *From dengue*, by being contagious and occurring in epidemics, by rarely affecting adults, by the absence of pain in the joints, and by the eruption, which does not come and go as in the latter.

What is the prognosis?

The prognosis differs in different epidemics and in different individuals. Excluding the epidemics of malignant scarlet fever, in which the mortality ranges from 50 to 60 per cent., it would probably average 13 to 19 per cent.

Unfavorable signs are convulsions, a temperature over 105°, and an irregular eruption. Should convulsions occur late in the disease, they are usually referable to uræmia, and give a better prognosis than when they occur early. Should this disease occur after surgical operations, or in pregnant or puerperal women, the prognosis is bad.

Prognosis of Complications.

When *deafness* occurs it is usually permanent.

The *prognosis of kidney complications* is unusually favorable, considering the nature of the disease and the gravity of the symptoms. By far the larger majority recover under appropriate treatment.

The *prognosis of heart complications* is as bad regarding the ultimate result as when they follow acute rheumatism.

How should this disease be treated?

(1) Prophylactic Treatment.

Isolation must be complete and entire, not only of the patient, but of all who come in contact with him. There should be only one nurse, and she must change her clothing before coming in contact with others. Only the necessary furniture should be allowed in the room, which should not be carpeted. The patient must not be allowed to come in contact with others until desquamation has entirely ceased.

Disinfection.—The body of the patient, and of all who come in contact with him, must be scrupulously disinfected. The desqua-

mated scales must be collected and burned. Such clothing as cannot be destroyed must be disinfected and boiled, and the room scrubbed with disinfectants, repapered and repainted after the disease is over.

(2) General Treatment.

Fever treatment and diet; anoint the skin; keep up the action of the kidneys with plenty of diluents, and, as desquamation begins, give quinine in tonic doses, and infusion of digitalis if albuminuria is present, substituting Basham's mixture for the latter during convalescence, and keep the patient in the room, or at any rate in the house, for three weeks after all desquamation has ceased, for fear of complications, as well as from fear of spreading the disease.

(3) So-called Specific Treatment.

Mercuric iodide has been lauded as a specific and to lessen the tendency to complications.

Sodium salicylate has been used every three or four hours, internally and by inunction (gr. xx to vaseline ʒj). Used in the latter manner, it allays the burning and heat of the surface.

Ammonium carbonate is good as a cardiac and nervous stimulant, if it does not produce gastric irritability.

Potassium chlorate with tincture of ferric chloride is often used when there is much throat complication.

(4) Treatment for Special Symptoms.

For *high temperature* antipyrine, quinine, or digitalis may be used, conjoined with sponging with tepid water and disinfectants.

If *cerebral symptoms are present with high temperature*, put the patient in a warm bath and pour cold water on his head, thus lessening the temperature of the bath rapidly.

For restlessness and nocturnal delirium occurring early in the case, bromide of potassium and chloral may be cautiously given.

To allay the irritation and burning and itching of the skin, any oleaginous preparation externally may be used, especially the sodium salicylate.

For the throat complications, keep the throat clean, and disinfected by boric acid in glycerine, potassium permanganate, thymol, or other disinfectant gargles. When the patient is too young to use a gargle spray the throat with an atomizer. Let the patient also have plenty of ice in the mouth.

In malignant cases reduce the temperature by cold baths, antipyrine, etc., and stimulate even though the temperature is high. Quinine should be given in decided doses in these cases.

During convalescence give quinine as a tonic, with tincture of ferric chloride.

(5) Treatment for Complications.

For ear complications the utmost cleanliness should be enjoined, and powdered boric acid blown into the auditory meatus.

For convulsions, if occurring early in the disease, reduce the temperature by cold water treatment and by antipyrine, and give potassic bromide and a brisk purgative. *When occurring later*, they are generally of uræmic origin. Large amounts of fluids should be administered internally; infusions of digitalis and chloral should be given, and cups should be applied over the kidneys.

For kidney complications, large amounts of diluents should be administered, and infusion of digitalis, with some alkaline diuretic, given until the blood disappears from the urine; at the same time dry or wet cups are employed externally. When the acute stage is passed, Basham's mixture should be substituted for the digitalis. A milk diet should be insisted upon until all trace of albumen has disappeared. When dropsy is present pilocarpine may be administered in small doses, frequently repeated, and vapor baths employed.

Measles.

What is measles?

Measles is an acute, contagious, exanthematous fever, due to a special poison characterized by marked catarrhal symptoms.

Synonyms.

Morbilli, Rubeola (this name is also applied to Rubella).

Causation.

It is due to a specific poison, which can be inoculated either by the nasal mucus or the blood. It is very contagious, especially among children. It usually attacks children from five to thirteen years of age, but may occur at any age, and when attacking adults is more severe. A second attack is exceedingly rare.

The Incubation Period is from eight to twenty-eight days.

Describe a case of measles.

It begins with chilly sensations, catarrhal symptoms and fever, sometimes convulsions. On the second day the fever declines, but the other symptoms persist. On the fourth day an eruption appears, the temperature again rises, and the catarrhal symptoms increase. After the eruption has lasted for from three to five days it begins to fade, the fever declines and a minute desquamation sets in, the patient convalescing by about the tenth day.

SPECIAL SYMPTOMS.

Eruption.—The eruption appears on the fourth day, first on the face and neck (from which it also first disappears), spreading slowly over the body, and consisting of papules arranged in crescentic patches, alternating with healthy skin. It lasts from three to five days, and gradually fades. Sometimes the eruption is delayed in appearance; in these cases there are apt to be grave nervous symptoms.

Catarrhal Symptoms.—The eyelids are swollen, there is a burning discharge from the nose, sore throat with cough, and laryngeal catarrh. Occasionally the mucous membrane of the alimentary canal suffers from the catarrhal process.

Fever Symptoms.—The temperature rises abruptly on the first day to 102° or more. On the morning of the second day a distinct remission occurs, the temperature falling almost to the normal, and then remaining stationary until the eruption appears, when it rises again to 102° or 103°, and continues there until the eruption has faded, when it returns to the normal.

VARIETIES.

Malignant or black measles occurs especially in adults, particularly among raw recruits, when it is called *camp measles.* The eruption is usually delayed, convulsions, delirium, or other nervous symptoms are present, and petechial spots occur under the skin and mucous membranes. It is a very fatal variety.

COMPLICATIONS.

(1) *Pneumonia.*—This is usually catarrhal, but in adults it may be croupous, and is a very fatal complication.

(2) *Pleurisy* is occasionally seen.

SEQUELÆ.

(1) *Chronic cough or chronic sore throat.*

(2) *Otorrhœa and deafness.*

(3) *Enlarged lymphatic glands.*

(4) *Development of the hereditary taints*, as scrofula, phthisis, etc.

What is the diagnosis and prognosis of measles?

DIAGNOSIS.

This disease is recognized by the abrupt rise in temperature, the remission, the catarrhal symptoms, the second rise in temperature at the time of the eruption, and the eruption, followed by minute desquamation.

DIFFERENTIAL DIAGNOSIS.

(1) *From scarlet fever* it is differentiated by the eruption appearing later, and in the form of crescentic patches, with intervening healthy skin, by the catarrhal symptoms, by the lower temperature, less rapid pulse, and the absence of pronounced throat symptoms.

(2) *From Typhus Fever.*—A mistake is only possible in malignant cases. Here the presence of an epidemic of either disease, with the catarrhal symptoms and appearance of the eruption on the face in measles, or the marked cerebral symptoms and prostration in typhus, should reveal the nature of the disease.

PROGNOSIS.

The prognosis is favorable. This disease is only dangerous through its complications and sequelæ. It is more dangerous in adults, and especially among troops is it more apt to be malignant, and to have serious complications.

What is the treatment of measles?

Give fever treatment and diet, and occasionally a laxative; act on the skin and kidneys by a fever mixture, and treat the catarrhal symptoms.

If the eruption is delayed and nervous symptoms are present, place the patient in a hot bath and pour cold water on the head, as in scarlet fever; at the same time administering quinine.

Should the fever be high add aconite to the fever mixture, or even give antipyrine.

In malignant cases reduce the temperature, give quinine, and stimulate boldly, to gain time.

For itching of the skin sponge with warm vinegar and water, or anoint the surface as in scarlet fever.

Should the cough prove troublesome, minute doses of opium or Dover's powder may be administered, or steam may be inhaled.

For pulmonary complications treat as if they were the only diseases.

Rubella.

Describe this disease.

It is an acute specific exanthem, due to a special poison, characterized by the peculiar eruption, somewhat resembling measles, and usually accompanied by very little fever.

SYNONYMS.

It is also called German measles, French measles, rubeola sine catarrhô, and rötheln.

SYMPTOMS.

There is usually very little fever, the eruption appears on the first or second day, generally coming first on the neck or chest, and spreading more or less over the body. It comes and goes. Catarrhal symptoms are usually absent, or but very slight. There is sore throat, swelling of the lymphatic glands of the neck, often sore mouth, with swollen gums, and a variable amount of headache and lassitude. The disease lasts for a week or ten days, and is followed by little, if any, desquamation. Sometimes the hair falls out after this, as after other fevers.

The eruption is not uniform. It is usually a coarse, measly rash, with large masses of intervening healthy skin. It is rose-colored, not crescentic, and frequently fades and reappears several times during the course of the disease.

COMPLICATIONS.

(1) *Pneumonia.*

(2) *Desquamative nephritis.*

PROGNOSIS.

It is not a dangerous disease, and the complications are but rarely seen.

TREATMENT.

Fever treatment and diet, light laxatives and diaphoretics are indicated.

For *the enlarged glands* the parts should be kept warm, and the neck rubbed with camphorated oil.

For *the sore mouth* a wash composed of borax (ʒj), carbolic acid (gr. j) glycerin and rose-water (*āā* ʒss) is very soothing and affords the patient much relief.

Other symptoms must be treated as in measles, should they require it.

Smallpox.

What are the varieties of smallpox?

There are *two varieties* of smallpox. *Variola*, or smallpox proper, and *Varioloid*, or modified smallpox. Both are produced by the same poison, and equally capable of transmitting the worst variety of this disease to the unprotected.

I. VARIOLA.

What is variola?

It is an acute, specific, eminently contagious and infectious fever, characterized by a peculiar eruption consisting of three stages, by a remission of the fever and other symptoms when the eruption first appears, and a secondary fever when the eruption attains its height.

What is the cause of this disease?

It is due to a specific poison, which is communicable from the time of the initial fever. It is highly contagious, and may be transmitted by the breath or other emanation from the body of the sick. The pus and scabs are most highly contagious. The poison clings to clothing. The black race are very susceptible. This is an old disease, having probably always existed in Asia, and having first been brought to Europe by the Crusaders.

What is the incubation period and duration?

The *incubation period* averages fourteen days.

Duration.—Four or more weeks must elapse before danger of contagion is over.

Describe the symptoms of variola.

It begins with a chill, moderate fever, intense headache and pain in the back and loins and legs, lasting for three days. Then a characteristic eruption appears, and the other symptoms abate or perhaps entirely subside. The eruption is at first papular, changing to vesicular, and finally becoming pustular. On the eighth day after its first appearance, the pus oozes out from the pustules, and a secondary fever occurs. The pus dries and forms scabs, the fever declines and the scabs fall off, leaving little depressed red spots, which heal by pitting.

Special Symptoms.

(1) *The initial or primary fever* is like any other fever, but the headache and pains in the loins and legs are much more intense. It lessens or disappears when the eruption comes out.

(2) *The eruption* comes out on the third day, first in the face, forehead and lips, but soon appears all over the body, and is papular. On the second day of the eruption, the papules change to vesicles. On the third, fourth and fifth days, the vesicles become pustules. On the fifth, sixth and seventh days the pustules increase in size, become indented in the centre (spoken of as "*umbilicated*") and surrounded by a distinct areola. On the eighth day of the eruption, usually, the pustules are at their height, and pus oozes from them at their base and forms scabs. This continues until the fourteenth day of the eruption, and is called *period of maturation*, after which the scabs begin to fall off and continue falling until about the twentieth or the twenty-eighth day. They leave little red or livid spots, which gradually become white indentations called pits.

The character of the eruption determines the type of the disease.

Variola is said to be discrete when the pustules are scattered; *confluent* when the pustules coalesce (and the symptoms are more severe), and *malignant* when extravasations of blood occur into the pustules, and grave symptoms of blood poison are present.

(3) *The secondary fever* usually occurs on the eighth day of the eruption (eleventh day of the disease), when the pustules maturate. It is caused by a slight degree of septicæmia, due to the absorption of pus into the circulation. During this time the face is immensely swollen, there is delirium, dyspnœa, and grave complications are apt

to occur. It is the period of greatest danger. The worse the case, the worse the secondary fever.

What are the varieties of variola?

(1) *Variola discreta* is the variety just described, each pustule being separate and distinct. It is the mildest type of this disease.

(2) *Variola confluens* is characterized by greater severity of symptoms and by the tendency of the pustules to run together in masses, forming enormous scabs, particularly in the face.

(3) *Variola maligna or black smallpox* is the most severe variety of the disease. The eruption may come out early, or may not appear until after death. Petechial spots occur on the skin, and blood is extravasated into the vesicles and pustules. There is active delirium passing into stupor and coma. The patient usually gets better or dies in the first week.

What are the complications of smallpox?

(1) Salivation.
(2) Inflammation or ulceration of the cornea.
(3) Pulmonary congestion.
(4) Pneumonia.
(5) Pleurisy.
(6) Formation of abscesses.
(7) Periostitis.
(8) Caries or necrosis, particularly of the lower extremities.
(9) And other complications, such as are seen after low fevers generally.

Describe the anatomy of the pustules.

Each pustule is subdivided into compartments (which do not communicate with each other), by little septa of connective tissue. The glands of the skin and the hair pass through them.

The pus does not apparently differ chemically or microscopically from ordinary pus.

Pustules are found not only on the skin, but on the broncho-pulmonary mucous membrane, and perhaps give rise to the laryngeal and pulmonary complication. They are sometimes found on the pleura, but rarely in the peritoneum. It is exceedingly rare to find them in the gastro-intestinal tract.

Describe the post-mortem appearances of variola.

With the exception of the eruption there is nothing characteristic. The blood is black and fluid, the heart is sometimes granular, and the kidneys are sometimes congested.

How is this disease diagnosed?

This disease is recognized

(1) *By the initial fever*, with intense pain in the back.

(2) *By the eruption* appearing on the third day on the face, being at first papular, changing to vesicular and becoming pustular.

(3) *By the initial fever subsiding* and its symptoms abating when the eruption first appears, and

(4) *By the occurrence of a secondary fever* when the pustules maturate.

What is the prognosis and mortality of variola?

The *prognosis* is favorable if the case runs a typical course.

It is unfavorable if the secondary fever is severe; if the kidneys do not act well; if there is marked salivation; if buboes and abscesses form; if it is complicated with pneumonia; if there are severe cerebral symptoms; or if it occurs in pregnant women.

The mortality in discrete smallpox is about four per cent., *in confluent smallpox* about fifty per cent.; *in malignant smallpox* nearly all die.

It is least fatal between the ages of fifteen and thirty years; it is most fatal before ten and after sixty years of age.

Is this a self-protective disease?

As a rule, one attack will protect forever from the disease. Should the patient have a second attack it is generally fatal. From a third, very few, if any, recover.

How should it be treated?

(1) PROPHYLAXIS.

Most thorough isolation should be practiced. There should be as little furniture in the room as possible, and the patient must be attended by but one nurse, who should be scrupulously isolated, and should never come in contact with any one without having previously changed her clothes and thoroughly disinfected herself. The attending physician should not stand nearer the patient than is neces-

sary to make a thorough examination, should not prolong his visit unnecessarily, and should never go to visit others to whom he may communicate the disease, without having changed his clothing and disinfected his whole person. The patient should be sponged with disinfectants, and the clothing, bedding, etc., destroyed when the disease is over. The room should be thoroughly disinfected, and ventilated for a long time before it is again occupied The scabs should be collected and burned, and the patient have a thorough disinfecting bath after all the scabs have fallen from his body, before he is allowed to come in contact with others.

(2) General Treatment.

Give ordinary fever treatment and diet, and feed liberally, and stimulate toward the period of maturation. Keep the room dark, and have it thoroughly ventilated. Treat such symptoms as require it, on general principles.

There is no specific.

Xylol may be given.

Carbolic acid or the sulphocarbolates have been recommended.

Thymol has also been used.

(3) Treatment of Special Symptoms.

If *diarrhœa* is present, sulphuric acid with a little morphine may be used.

If the throat symptoms prove severe, boric acid and glycerin, or potassium permanganate gargles are useful.

For delirium when fierce and active in the initial fever, bromides and chloral, with cold to the head, will generally give relief; *when low and muttering (usually in the secondary fever)*, camphor, opium, given cautiously, free stimulation, and nourishing food at frequent intervals, is the best treatment.

During convalescence give tincture of ferric chloride and quinine, as tonics.

To prevent pitting keep the patient in a dark room, and apply anything to the exposed surface which will keep out light and air. Corrosive sublimate lotions have lately been much employed.

(4) Treatment of Complications.

If salivation is profuse, astringent gargles, as a solution of tannic acid, are indicated.

For ulcers of the cornea, touch each ulcer with nitrate of silver in strong solution, or in the form of lunar caustic. Feed the patient well, and stimulate boldly.

For other complications, stimulate, nourish and treat as if it were the only disease present.

II. Varioloid.

What is varioloid?

Varioloid is variola modified by inoculation, vaccination, or occasionally by a preceding attack of variola.

How does it differ from variola?

It differs from variola,

(1) *In having an initial fever of less intensity*, and characterized by less violent symptoms.

(2) *By the eruption appearing one day later.*

(3) *By the eruption running a more rapid course*, many of the papules aborting without even reaching the vesicular stage, and the pustules which are present maturating sooner than in the unmodified disease.

(4) *By the absence of the secondary fever*—therefore the period of maturation is not particularly dangerous.

(5) *Ulceration of the cornea* and other severe complications never occur.

Means of Preventing Smallpox.

What means may be employed to prevent smallpox?

(1) *Inoculation*, with the virus of smallpox. This usually gives rise to a milder form of the disease, but as occasionally it is followed by the most virulent type of smallpox, and as each inoculated person is a centre from which the disease may spread, it should never be practiced.

(2) *Vaccination.*—This was first performed by Jenner, May 14th, 1796, and consists of introducing into an abrasion on the surface of a healthy person the lymph from an eruption on the udder of a cow affected with cow-pock, or, from the eruption produced in a human subject by lymph thus previously introduced. It affords an almost entire immunity from smallpox,—the few persons who take the

disease after a good vaccination suffering from it in a very modified form (mild varioloid).

Re-vaccination should be practiced at the age of puberty, or in the face of a spreading epidemic of smallpox.

How to Vaccinate.

How is vaccination performed, and what will occur should it be successful?

(1) *In vaccinating* use human lymph or scab, or the virus taken from the cow affected with true cow-pock, which is much better than that produced by inoculating the cow with human smallpox.

(2) *Scrape the surface* to be vaccinated until it is devoid of epithelium, and serum begins to exude from it. Make several slight incisions, and cross incisions, but not deep enough to draw blood.

(3) *Rub over this surface* the matter, previously moistened, and protect it until it is thoroughly dry.

(4) *Watch the patient carefully*, observing the vaccination to see whether it is a good one, or in other words *really protective.*

If The Vaccination Takes—

(1) *About the fourth day* a slight red papule appears.

(2) *On the fifth, sixth and seventh days* this papule becomes a distinct vesicle, and enlarges.

(3) *On the eighth day* the vesicle is at its height, and a distinct regular areola is formed.

(4) *By the ninth, tenth or eleventh day* it becomes a pustule with an enormous areola.

(5) *By the fourteenth day* the areola fades and a crust forms.

(6) *By the nineteenth or twentieth day* the scab comes off.

When animal virus is used the course of the eruption may be three or four days later.

If a patient is exposed to the contagion of smallpox it is better to vaccinate him immediately, as the vaccinia has almost run its course before the incubation period of smallpox is over, so that he will be at least partially protected from the later disease.

Varicella.

Describe varicella.

Varicella, or chicken pox, is a slight contagious fever, attended with vesicular eruption, which never becomes pustular.

CAUSE.

It is due to a specific poison which is not identical with that of smallpox, and from which it does not protect.

THE INCUBATION PERIOD is less than a week.

THE DURATION is about a week.

SYMPTOMS.

There is a chill, slight fever, malaise, a vesicular eruption, ending in about a week by the formation of scabs, which fall off, sometimes leaving pits.

The eruption comes out during the first twenty-four hours, and is vesicular from the beginning. It is usually most marked on the trunk, and runs a rapid course, scabs forming by the fourth day. It is sometimes seen in the mouth, on the arches of the palate, the uvula and posterior pharyngeal wall, giving rise to sore throat.

COMPLICATIONS are rare. The most important is pleurisy.

PROGNOSIS.

This is favorable. Death is exceedingly rare in this disease, and, when it occurs, it is due to some complication.

TREATMENT.

Isolate the patient, giving him fever treatment and diet, and treat any symptoms which may require it.

If the bowels are constipated make use of laxatives.

If the fever is high give aconite or veratrum viride.

To prevent pitting, when the eruption is on the hands or face make use of the same means as are indicated in smallpox.

Erysipelas.

Describe erysipelas.

Medical erysipelas is an acute eruptive fever, attended with well-marked constitutional symptoms, the eruptions being usually limited to the head and neck.

It is also called St. Anthony's Fire.

CAUSE.

It is due to a special poison, and is slightly contagious. It may, however, be carried in clothes, or on the person of a physician or nurse, to a puerperal woman, and give rise to puerperal septicæmia.

INCUBATION PERIOD is less than a week, and the DURATION is about a week.

SYMPTOMS.

It begins with a chill, fever, eruption, swelling of the skin, gastric disturbance, albuminuria; the glands of the neck are frequently swollen, and cardiac murmurs are often present. Sometimes there is nocturnal delirium. After lasting several days the eruption gradually fades, and desquamation follows.

SPECIAL SYMPTOMS.

The eruption starts as a little red spot on the side of the nose, cheek or ear, and spreads rapidly over one side of the face and head, and sometimes the neck. The color varies from a vivid red to a yellowish-red or dusky hue. Sometimes the entire head and neck is involved.

The swelling is great, the parts are œdematous, and pit on pressure.

The fever is often very high and is preceded by chills of greater or less severity.

VARIETIES.

(1) *Simple erysipelas* is the variety just described.

(2) *Phlegmonous erysipelas* is that variety in which the deeper structures are involved, the swelling being much greater, the parts more dusky in color, and abscesses usually forming in the connective tissues.

(3) *Gangrenous erysipelas* is rare in this country. In this variety the skin and subjacent structures become gangrenous. It may be epidemic, and is especially seen among troops.

(4) *Erysipelas ambulans* or *migrans* is a form of erysipelas in which various parts of the body are successively involved.

COMPLICATIONS.

(1) *Œdema of the glottis.*

(2) *Bronchitis.*

(3) *Pneumonia.*

(4) *Abscesses.*

(5) *Endocarditis* (rare).

(6) *Cerebral Erysipelas.*—In this complication the inflammation extends to the brain, through the communicating branch of the facial to the internal maxillary vein, thence to the pterygoid plexus, and thus to the cavernous sinus.

The symptoms are grave nerve disturbance and very high temperature.

What is the diagnosis and prognosis of this disease?

Erysipelas is recognized by the chills, the fever, the gastric disturbance, the eruption usually limited to the head and neck, which are much swollen, the albumen of the urine, and the swelling of the glands of the neck.

DIFFERENTIAL DIAGNOSIS.

(1) *From Erythema.*—The latter is not confined to the head, as a rule; it runs an irregular course, and there are no constitutional symptoms.

(2) *From Roseola.*—This disease shows no tendency to spread, and runs an irregular course.

PROGNOSIS.

This is usually favorable.

It is *unfavorable*—

(1) In drunkards.

(2) In cases marked by much delirium, stupor, coma or other cerebral symptoms, showing blood poison.

(3) In cases with severe internal complications.

(4) In protracted cases of erysipelas ambulans, in which the patient may die, worn out by the successive attacks.

How should erysipelas be treated?

(1) General Treatment.

Isolation, disinfection and cleanliness. Fever treatment and diet and mild purgatives, with diaphoretics, should always be used.

It is useless to attempt to limit the spread of the eruption by local applications.

(2) Local Treatment, however, gives relief, and should always be resorted to.

(*a*) *Moist applications*, as corrosive sublimate lotion, lead water and laudanum, or poultices may be used. Or—

(*b*) *The surface may be dusted* with powdered starch, bismuth, or oxide of zinc. Or—

(*c*) *Ointments* may be applied, as zinc ointment, cold cream, etc.

(3) Specific Treatment.

When the patient is strong and active, and the disease appears to be spreading rapidly, especially when the temperature is high, hypodermic injections of *pilocarpine* (gr. $\frac{1}{6}$) will often abort the attack.

Tincture of the *chloride of iron* (gtt. xx–xxx) every third hour, combined with quinine (gr. xvj per day) is also of great value.

(4) Treatment for Special Symptoms and Complications.

Should the throat symptoms be severe, cleanliness and disinfecting gargles must be used, and the throat may be painted with Monsel's solution in glycerin.

Should œdema of the glottis be present every means must be taken to arrest the inflammation, and if all fail and the patient is in danger of suffocation, scarification of the glottis or tracheotomy should be performed without delay.

When phlegmonous abscesses form, the pus should be evacuated by an early incision, and the parts treated antiseptically.

Low muttering delirium due to weakness or septicæmia should be treated by quinine and stimulation.

If the inflammation extends to the brain, free purgation, arterial sedatives and bromide of potassium, with opium, should constitute the treatment.

Dengue.

What is this disease?

This disease, which is also known as break-bone fever, dandy fever, etc., occurs in epidemics, principally in the East and West Indies and the southern United States, and is characterized by symptoms resembling both those of scarlatina and those of acute rheumatism combined.

The CAUSE is unknown. It is contagious.

SYMPTOMS.

It begins with stiffness, pains and swelling in some of the smaller joints, with pain in the back and stiffness and aching of various muscles, especially affecting those of the neck. There is fever and headache. These symptoms last for three or four days, and then the fever declines and the other symptoms lessen or pass away. After an interval of two or three days the symptoms return, and in addition there is usually much gastric irritability and a heavily coated tongue. From the fifth to the seventh day of the disease an eruption appears and the other symptoms again subside. The eruption usually resembles that of scarlet fever, but sometimes is in the form of papules and occasionally of wheals or vesicles.

It lasts for two or three days and is accompanied by much irritation of the skin. During convalescence there is much depression and muscular weakness, and arthritic pains and stiffness may persist for some time. The lymphatic glands are sometimes enlarged.

DIAGNOSIS.

The disease is recognized by the muscular and arthritic pains, the fever, the lull, the eruption, the termination, and the knowledge of an epidemic.

PROGNOSIS.

Favorable.

TREATMENT.

The patient must be isolated, as the disease is contagious. He should be placed on fever treatment and diet, and confined to bed until convalescence is established. There is no specific. Quinine may be given in tonic doses throughout the disease and, as convalescence approaches, combined with iron and strychnine.

A moderate amount of stimulation is serviceable after the primary fever subsides.

For the pains, Dover's powder, or even antipyrine or phenacetin may be administered.

The joints are treated locally, as in acute rheumatism.

For the itching and burning of the skin the treatment is similar to that of scarlatina.

Diphtheria.

What is diphtheria?

Diphtheria is an acute general disease, characterized by a contagious sore throat in which membrane forms on the tonsils, uvula, and back of the fauces especially, but may form on the skin, in the bladder, stomach and intestines, nares, or bronchial mucous membrane. Glandular enlargements also occur and there is great general depression, out of proportion to the local symptoms.

SYNONYMS.—It is also called Egyptian sore throat, malignant sore throat, synanche maligna, etc.

This disease was known to the ancient Egyptians, but disappeared for centuries, to reappear again in the early part of this century.

What is the cause of this disease?

It is due to a special poison, connected probably with bad drainage. It clings to walls, is frightfully contagious, may be communicated by the breath or membrane, and is usually epidemic. It is more intense during cold weather, and may affect any age; but is most common from two to seven years of age.

Describe the membrane.

The membrane is diffused on the surface; it consists of fibrin, degenerated epithelium, blood corpuscles, pus, and bacteria. It at first is free on the surface, but soon extends in and through the mucous membrane and into the glands. No peculiar bacteria have been found. As far as has been ascertained, they are not the cause but the consequence of diphtheria. The mucous membrane underlying the false membrane is usually superficially ulcerated, the cervical glands are swollen and full of bacteria. The membrane remains for a week or ten days, disintegrates, is loosened, and comes

off, leaving the underlying surface normal, or only superficially ulcerated. The membrane has a strong tendency to recur, is constantly forming, or spreading to other parts.

What are the symptoms of diphtheria?

It begins usually with a chill, vomiting, slight fever, and a sore throat, with difficulty in swallowing from the first; soon the peculiar membrane is seen in the throat, beginning as a grayish exudation which appears in spots on the fauces, palate, roof of the mouth and tonsils, the spots rapidly coalescing; the glands of the neck become swollen, the voice is muffled and nasal, the breath offensive and there is great general depression. All this lasts for a week or ten days when the membrane begins to disappear by shriveling up, being expectorated or swallowed. Albumen, if not present in the urine before appears about this time; convalescence is tedious, the patient being very weak, pale and anæmic. The patella tendon reflex is abolished.

Relapses may occur, the patient finally dying from exhaustion; or grave symptoms of blood poisoning may supervene and the patient die of septicæmia or pyæmia.

What are the complications of diphtheria?

(1) *Laryngeal diphtheria*, where the membrane extends into the larynx, causing cough, muffled voice, oppressed breathing, and symptoms like those of membranous croup intensified.

(2) *Nasal diphtheria*, where the membrane extends into the nose, causing a thin irritating discharge and usually symptoms of blood poisoning. This is the most fatal form of the disease.

What is the incubation and duration of diphtheria?

The *incubation period* is about a week, the average *duration* two weeks.

What are the sequelæ?

(1) *Paralysis of the muscles of deglutition* causing difficulty in swallowing and regurgitation of fluids into the nose. This may appear two or three months after convalescence.

(2) *Local or general paralysis* of sensation or motion.

(3) *Paralysis of the bladder*, which may appear several months after the disease.

(4) *Paresis or weakness of the heart*, which may give rise to cardiac failure. This may come on during the disease, and last for a long time after convalescence is established.

(5) *Perversion of the special senses*, as defective eyesight, perverted taste or smell.

(6) *Persistent anæmia.*

How is this disease diagnosed?

Diphtheria is recognized by the knowledge of an epidemic, the presence of the membrane in the throat, the great nervous depression, the enlargement of the cervical glands, and the presence of albumen in the urine.

Differential Diagnosis.

(1) *From scarlet fever*, it is known by the tendency of the membrane to spread, by its firmer consistence, by the absence of eruption, and by the difference in the pulse and temperature.

(2) *From follicular sore throat* in children. In these cases the membrane is strictly limited to the follicles of the tonsils, and can be scraped away in masses. It does not tend to return, and there are no sequelæ, and no great nervous depression.

(3) *From stomatitis or thrush* occurring in low fevers. In thrush the deposit occurs on the gum and consists of fatty material interspersed with a parasitic growth. It is soluble in ether and acids, but not in alkalies, as is the diphtheritic membrane.

(4) *Laryngeal diphtheria may be diagnosed from membranous croup*, by the membrane beginning above, in the pharynx, and spreading downward into the larynx in 99 per cent. of cases, by the albumen in the urine, and by the prominence of the constitutional symptoms from the onset.

What is the prognosis and mortality?

The prognosis is always grave, but no case should be despaired of. *Unfavorable symptoms* are, great swelling of the glands, marked albuminuria, and extension of the membrane into the nose, larynx, and gastro-intestinal canal. *The mortality* varies greatly in different epidemics. In a mild epidemic it will average about five per cent., in a grave epidemic about thirty-three per cent.

How should this disease be treated?

(1) PROPHYLACTIC TREATMENT.

Strict *isolation* should be enforced, as in the contagious fevers. Thorough *ventilation* should be practiced, and everything coming from the patient should be scrupulously *disinfected*.

(2) GENERAL TREATMENT.

Fever diet must be given, the patient being sustained every two hours, as in typhoid fever. Free stimulation should be resorted to, giving alcohol, for its effect on the strength, pulse and mind of the patient. There is a great toleration for alcohol in this disease.

(3) MEDICAL TREATMENT.

Very early in the disease, when the membrane is first forming, the application of a strong solution of sulphate of copper, or of Monsel's solution, with equal parts of water freely applied to the throat, is occasionally serviceable.

After the membrane has formed, strong applications are injurious; the throat should then be kept disinfected with potassium permanganate, or a solution of salicylic acid (seven per cent), or thymol (gr. iv, in glycerin and water).

Internally the mercurials give the best result; corrosive sublimate may be given in doses of gr. $\frac{1}{30}$ every two hours, and increased to the point of tolerance, or calomel may be given in doses of gr. xx three times daily, especially when there is tendency to laryngeal complications. Many prefer to give smaller doses of calomel, frequently repeated. Mercurial should always be guarded by a little opium.

Tincture of the chloride of iron, with or without chlorate of potassium, is sometimes used in large doses, with a little quinine, as in erysipelas.

(4) TREATMENT OF COMPLICATIONS.

(*a*) *In laryngeal diphtheria* inhalations of the vapor of slaking lime, followed by emetics if the patient is not too weak, are sometimes of use. The sufferer must be supported by frequent feeding, and stimulation should be pushed to the verge of tolerance.

Saturated solutions of pepsin, trypsin or papaïn may be sprayed into the throat with the view of dissolving the membrane.

Should all else fail, intubation or tracheotomy should be resorted to.

(*b*) *For nasal diphtheria* constitutional treatment must be pushed, and the nostrils kept clean with disinfectant solutions employed with a nasal douche or long syringe.

(5) Treatment of Sequelæ.

In the treatment of *paralyses* time is a great factor, and good hygiene, iron and quinine administered internally, and strychnine hypodermically, with the use of galvanism, and sea-bathing when possible, are useful adjuncts.

For *persistent anæmia*, cod-liver oil, alterative and nutritive treatment and sea-voyages give the best result.

For the *weak heart*, rest, digitalis, strophanthus, strychnine, and a moderate amount of alcohol are to be employed.

Parotitis.

What is parotitis?

Parotitis, or mumps, is an acute, infectious disease due to a special poison occurring in epidemics and characterized by an acute inflammatory process affecting one or both of the parotid glands. It is usually a self-protective disease, and attacks children or young adults.

What is the period of incubation of this disease?

The period of incubation is two or three weeks.

What are its symptoms?

It appears suddenly with a chill, fever, malaise, and headache. The temperature may be as high as 103°, the pulse is quick and irritable. After a day or two there is stiffness of the temporo-maxillary articulation, the parotid gland becomes swollen, the skin is often reddened, œdema of the face takes place, and pain is experienced, which is greatly increased by any movement of the jaws or by taking any acid substance into the mouth.

Salivation is often present. One gland is usually involved at a time, followed, as the swelling subsides (in a week or ten days), by a similar attack in the opposite gland.

Metastatic involvement of the testicle, ovary, or mammary gland sometimes occurs, and is not unfrequently followed by permanent impairment of their functions.

What is the prognosis of mumps?

The prognosis is always favorable. The only danger is that if metastasis occurs, the organs involved may be permanently damaged.

What is the treatment of mumps?

The treatment is entirely symptomatic. Pilocarpus has been used with some success as a specific. The patient should be kept warm, and dry or moist heat applied to the face.

Submaxillitis.

What is this affection?

It is a slightly contagious disease characterized by chilly sensations, fever (the temperature occasionally being as high as 104°), gastric disturbance, a coated tongue, usually constipation, swelling of the submaxillary glands and sometimes of the cervical glands also, difficulty and pain on opening the mouth or in swallowing, but not attended with swelling of the parotid glands, nor is the pain increased by taking acid into the mouth. Both submaxillary glands are usually affected during the course of the disease, although rarely simultaneously or with the same severity. The disease lasts from a few days to two weeks, and terminates by lysis.

The *incubation period* is from twenty-four hours to two weeks.

The *cause* is probably a specific poison. It affects adults as well as children.

The *treatment* consists in fever treatment and diet; reducing the temperature when high by aconite, cold sponging, or even phenacetin; giving minute doses of calomel for the irritable stomach, and using disinfecting mouth-washes. Iron and quinine should be administered as tonics during convalescence.

Pertussis.

What is this disease?

Pertussis, or whooping-cough, is an inflammation of the larynx and bronchial mucous membrane, with characteristic nervous phenomena, and is eminently contagious. It is a disease of childhood, is epidemic, the epidemics usually following those of measles. It is due to a specific poison.

What are the symptoms and complications of whooping-cough?

The disease consists of three stages :—

(1) *The Catarrhal Stage*, in which the symptoms are naso-laryngo-bronchial catarrh, often associated with nocturnal dyspnœa.

(2) *The Nervous Stage.*—During this stage the whoop comes on. The catarrhal trouble continues, but the paroxysms of coughing become more severe, and the expiratory efforts keep up until the eyes fill with tears, the face becomes deep red and livid from interference with the aëration of the blood, and the child is thoroughly exhausted. Then follows an inspiratory whoop, after which the expiratory efforts again continue. After a time a thick, tenacious mucus fills the mouth, the child often vomits, and the paroxysm of cough ends. The paroxysm is often produced by hearing others cough and frequently by the sight of food. In severe cases, bronchial or conjunctival hemorrhages may occur from rupture of small capillaries during the paroxysm; and hernia has resulted from the violence of the cough, in boys with patulous inguinal canals.

(3) *The Stage of Decline.*—The symptoms gradually pass away, the cough becoming less and less frequent.

Duration.

The first stage usually lasts for eight or ten days. The second stage about two weeks; the whole disease about six weeks, or sometimes much longer.

Complications and Sequelæ.

1. *Pneumonia*, a dangerous complication.
2. *The development of latent taints*, as scrofula or tuberculosis.

What is the prognosis, diagnosis and treatment?

THE PROGNOSIS is favorable, the danger lying in the complications or sequelæ.

DIAGNOSIS.

The disease is recognized by the attacks of dyspnœa, the catarrhal trouble, the whoop, and the knowledge of an epidemic. Physical examination of the chest reveals nothing more than in ordinary bronchitis.

TREATMENT.

Warm baths and diaphoretics are always of service. Try to get the patient to resist the tendency to cough. If the catarrhal symptoms continue, squill or ammonium chloride is of advantage. If the secretion is excessive, alum should be employed internally.

For the paroxysms of cough, quinine, chloral and bromide of potassium may be given every few hours, or small doses of prussic acid, or antipyrin, acetanilide or phenacetin may be employed, or cocaine may be applied to the parts. Asafœtida, musk and other antispasmodics are sometimes of use. Quinine in large doses is occasionally very serviceable. It should at any rate be used as a tonic, and should be employed in the form of suppositories.

After the disease is over cod-liver oil should be given, and, if possible, change of scene and air prescribed.

Cholera.

What is cholera?

Cholera is an acute, epidemic, infectious disease, characterized by excessive vomiting, violent serous purging, severe cramps and collapse.

It is a native of India, where it is always present, and from whence it spreads in epidemic waves, usually traversing the entire civilized world before it subsides.

What is the cause of this disease?

It is due to a special poison, probably to the cholera bacillus of Koch, which is always present in the dejecta of those suffering from the disease. This poison is especially transmitted by running water,

in which it multiplies with marvelous rapidity, and to which it finds its way from clothes which may have been washed in a stream, or from cholera stools which have been carelessly thrown away. It may also be transmitted by merchandise. By these means especially does the infection spread.

What is the period of incubation?

The period of incubation is very short, the average being about four days.

What are the symptoms?

Cholera may commence suddenly, or it may be preceded by an attack of diarrhœa, which is more persistent and more severe than an ordinary diarrhœa, and usually accompanied by nausea, vomiting, pain and depression. The disease consists of three stages :—

(1) The incipient stage—that of diarrhœa.

(2) The stage of the fully developed disease, and—

(3) The stage of collapse, or, if the patient is about to recover, the stage of reaction, followed by the condition known as cholera typhoid.

First Stage.—Cholera usually begins with chilliness, a coated tongue, pain in the neighborhood of the umbilicus, thirst, diarrhœa, perhaps vomiting, malaise, and often a morbid dread of the disease. The stools at first are watery, but contain fecal matter; they are voided with considerable force, and rapidly become more serous, losing their fecal characters. After this has lasted for a variable period, usually several days, the disease becomes fully developed.

Second Stage.—The diarrhœa now becomes more marked, profuse and watery, and is so continuous that the patient hardly has time to rise from the commode before he is obliged to return to it again. The force with which they are voided increases until they are fairly shot out from the patient's body. The passages now consist of large quantities of grayish fluid, consisting of serum and epithelium, resembling rice-water in consistence and appearance (hence the name applied to them). The same matters are vomited, or perhaps the vomited matters may contain bile. There is no particular pain attending the passages, but the patient suffers from intense thirst, due to the escape of the fluids from the body and, consequently, from the tissues generally. Cramps soon come on in the calves of the legs

especially, but in all parts of the body as well, which are very severe in character, and the patient becomes too weak to rise from his bed.

Third Stage.—The patient very soon passes into the stage of collapse, or reaction takes place. The latter condition is sometimes seen even when the patient has been in the stage of collapse.

Collapse.—The rice-water discharges continue to be voided both from the mouth and the rectum, and the cramps continue with undiminished severity. The loss of fluids is so great that the patient's body seems to shrink visibly under the very eyes of his attendants. His eyes are sunken, the nose pinched, the cheeks hollow, and the lips and face livid from impeded circulation, the blood having become too thickened to flow freely through capillaries, and otherwise too much damaged to properly perform its oxidizing functions. The surface of the body is cold, the temperature falling sometimes as low as 73°. The breath is icy and seems to have lost its moisture, to have become dry. The vomiting and purging cease for want of expulsive power; the veins become prominent and turgid with thick black blood, the pulse is feeble and very compressible, but may be either slow or rapid. The mind remains clear, although the sufferers usually become apathetic, probably from the accumulation of carbonic anhydride in the blood. The urine is scanty and contains albumen.

This is the condition known as cholera asphyxia, or the algid state of cholera, from which very few recover, although reaction even from this condition sometimes occurs.

Reaction.—When this comes on, the temperature of the body rises, and a secondary, or reactionary fever sets in, the temperature often going up as high as 103° or 104°. Simultaneously, the pulse also increases in volume and in strength, and the vomiting and purging lessen, and finally cease. The urine increases in amount, but still contains albumen, and the patient either recovers in a week or two, or passes, more generally, into a low typhoid state called *cholera typhoid*, which protracts his recovery for four or five weeks.

What is the duration of an attack of cholera?

Sometimes the disease is very rapid, there being no prodromes at all, but simply severe vomiting and purging coming on very suddenly, rapidly followed by collapse, death taking place a few hours after the appearance of the first symptoms. Usually, however, the

disease lasts several days. When recovery takes place, the convalescence is generally very tedious, and is often prolonged by the occurrence of various sequelæ.

What are the complications and sequelæ?

(1) *Uræmia* is of frequent occurrence and may come on in any stage of the disease.

(2) *Bronchitis.*

(3) *Pneumonia.*

(4) *Parotitis*, or,

(5) *Bedsores*, may come on in any stage, but more particularly in the last, and prove to be exceedingly serious complications.

The Sequelæ are—

(1) *Anæmia*, with great debility.

(2) *Irritability of the bowels*, lasting for months.

(3) *A tendency to the formation of boils and abscesses*, and

(4) Other sequelæ such as are common after any acute debilitating disease.

What is found on post-mortem examination?

After death from cholera the *temperature of the body* often rises. *Rigor mortis* comes on very rapidly, and the resulting muscular contractions are so powerful that the limbs are often displaced, or, at all events, the fingers and toes are moved, presenting a very weird spectacle. The *body* is shrunken; the *blood* thick, deprived of its serum, and slightly acid in reaction. The *veins* are distended, while the *arteries* are entirely empty. The *mucous membranes* are pale and anæmic, except here and there in the intestine where there are spots of intense congestion, even devoid of epithelium. The *stomach and intestines* contain large quantities of a rice-water-like fluid, mingled with epithelial debris, and, under the microscope it is seen to contain large quantities of the peculiar *bacillus of cholera.* The *kidneys* are congested and their tubules choked with desquamated epithelium. The veins of the *medulla* are enlarged and distended with blood.

How is this disease diagnosed?

There is only one disease with which it might be confounded, and that (*severe cholera morbus*) is more likely to be mistaken for cholera,

than cholera for it. The distinctive points are, the knowledge of an epidemic of Asiatic cholera, the greater severity of the symptoms from the onset, the early occurrence of the rice-water discharges, which are almost continuous, and ejected with much violence, the cramps which are much more severe in true cholera, and the presence of the comma-bacillus in the stools.

In all cases of doubt cultivation experiments should be made, by inoculating gelatin plates and keeping them at a temperature of from 68°–75° F. for twenty-four hours, when colonies of the cholera bacillus will be formed. The funnel-shaped appearance produced by the liquefaction of the gelatin is of value also in diagnosis, as but few intestinal bacteria grow in nutrient gelatin, and these do not liquefy it.*

What is the prognosis?

The prognosis is very unfavorable. The cases in which the disease comes on suddenly, are nearly all fatal. Those who were previously debilitated, especially if debilitated by drink or by sexual excesses, usually suffer severely. When it attacks the very young or the very old, the prognosis is not favorable.

How is cholera treated?

(1) PROPHYLAXIS.

(*a*) *If cholera is threatening a community*, but is not actually present, everything must be done to insure the best possible hygienic conditions, and to this end old cess-pools must be thoroughly disinfected, and, if possible, entirely destroyed; the utmost attention must be paid to the drainage, and the water supply must be rigidly inspected and guarded. The people must be made to understand that their *safety depends upon the care which they exercise to prevent any of the emanations from cholera patients gaining access to the water which is used for domestic purposes;* that the cholera stools must be thoroughly disinfected, and when thrown away, they must not be cast where they can possibly infect any running water which that community or any other uses for drinking, cooking or washing. Clothes soiled by the discharges of cholera patients had better be destroyed than washed, or when that is impossible, they should first

* Pfeiffer, *Deut. med. Woch.* Sept., 8, 1892.

be thoroughly disinfected, then washed in water remote from any running stream, and this water carefully thrown away. These precautions, if strictly carried out, are much surer than quarantine, as it is usually practiced, which gives a false idea of security, without absolutely excluding the disease.

(*b*) *When the disease is actually present in a community*, the sick must be separated from the well, and the latter pay the most scrupulous attention to their hygienic surroundings, to cleanliness of person, to their food, and to their drink. The water used for cooking or drinking must be previously boiled. The diet of those who are not suffering from the disease should be of the kind to which they are accustomed, avoiding, of course, any indigestible food, unripe fruit, or uncooked vegetables. *Inoculations* with cultures of the "comma-bacillus" have been practiced in Italy and Spain as preventives to cholera. The result is still *sub judice*.

(2) General Treatment.

The patient must be placed on fever treatment and diet from the start of the disease, but should be given no water. Small pieces of ice may be dissolved in the mouth, or a little brandy and ice, or small amounts of iced aërated waters may be given.

It is of the utmost importance to check the diarrhœa as soon as possible, and no case of diarrhœa, no matter how trifling it may seem, should be neglected during the prevalence of an epidemic of cholera. For this purpose the best remedy is sulphuric acid combined with opium or morphine. Acetate of lead (gr. iv) combined with opium (gr. j) repeated in an hour, if the diarrhœa continues, may be used instead of the sulphuric acid.

Thymol has recently been highly recommended as an intestinal antiseptic. It is given by enema, ʒj being dissolved in 7½ pints of water (about 1 to 1000), and retained if possible for five minutes.

If the disease passes into the second stage in spite of the treatment, with vomiting, purging and cramps, continue the use of the acid or of the lead acetate with opium (gr. ss–j) frequently repeated, and give morphine hypodermically as well, to allay the nausea and vomiting and to relieve the cramps. Do not, however, trust entirely to the morphine hypodermically, as opium, either in the solid form, or as

the deodorized tincture given by the mouth, produces the best effect in these cases. Chloral has been used hypodermically for the cramps, and may at all events be beneficially employed as an embrocation dissolved in soap liniment. Stimulating frictions should also be employed, and mustard plasters applied to the surface generally. If this treatment is systematically carried out in the early stages of the disease, and is energetically pushed, the disease may be arrested.

If, in spite of the treatment, the disease passes into the third stage, diffusible stimulants, persistent frictions with hot turpentine, and baths as hot as the patient can bear them, give the best results. Caffeine (gr. ss) and ether have been injected hypodermically with good results, and intravenous injections of saline solutions (sodium carbonate ʒj, sodium chloride ʒiij, with enough water to make the sp. gr. of the solution about 1020), giving about one to two pints of the solution at a time, have been used. Unfortunately, although temporarily of service, its effects are rarely lasting.

When reaction takes place, or when the patient survives the stage of collapse, great attention must be paid to the state of the kidneys. Aërated mineral waters must be given in large quantities, both to supply the water to the system which it has lost, to keep the kidneys acting, and to aid in the elimination from the blood of impurities which would otherwise be retained in the system.

GENERAL DISEASES—(B.) DIATHETIC DISEASES.

Acute Rheumatism.

What is acute rheumatism?

It is an acute constitutional disease, characterized by fever, acid sweats, and a special tendency to inflammation of the larger joints, which, however, are not permanently affected.

SYNONYMS.

Rheumatic fever; acute articular rheumatism.

What is its cause?

It is predisposed to by the so-called rheumatic diathesis, which is generally hereditary. An excess of lactic acid is found in the blood, shortly before the attack, but whether it is the cause of the disease, or its result, is not definitely determined. Exposure to damp and cold is an exciting cause.

What are the duration and symptoms of this disease?

If not treated, the attack will last from three to six weeks, or more. *Under the present plan of treatment* the duration is from ten days to three weeks.

SYMPTOMS.

It begins with a severe chill, followed by fever and stiffness in the joints. Soon, however, the stiffness lessens, and the joint becomes red, swollen, painful on motion or to the touch, and hot. Acid sweats occur which do not lessen the pain, nor reduce the fever.

The fever is usually not very high, ranging from 102° to 104°.

The pulse is full and bounding.

The tongue is coated with thick creamy fur.

The urine is high-colored, scanty, and contains an abundance of urates.

Acid sweats occur often from the beginning to the end of the disease.

The Joints.—One joint, or corresponding joints on both sides, may be involved, and as the disease leaves one joint, others become successively affected; the smaller joints escape.

What are the complications of acute rheumatism?

(1) Complications Affecting Serous Membranes.

Endocarditis.—Vegetations form on the valves of the heart, and permanently impair their functions.

Pericarditis.

(2) Complications Affecting the Mucous Membranes are Rare.

Bronchitis.

(3) General Complications.

Rheumatic Hyperpyrexia.—This is a grave complication, characterized by restlessness, delirium, albuminuria, and a temperature from 108° to 115.

Cerebral Rheumatism.—Very dangerous, but rare.

What is the morbid anatomy of this disease?

There is a local *inflammation of the joint* affected, and effusion takes place into the synovial cavity, but there is no ulceration, and no tendency to the formation of pus; hence, no serious consequences to the joints.

The blood contains lactic acid and an increased amount of fibrin.

Heart lesions may or may not be found.

How is this disease diagnosed?

The joints are hot, swollen, red and painful, but there is no tendency to suppuration. The attack leaves one joint in a few days, and others are apt to be involved, the first being well. The larger joints are affected; there are acid sweats and a tendency to complications, especially of the heart.

Differential Diagnosis.

(1) *From Pyæmia.*—The latter disease is known by the history of some suppuration and the fact that the constitutional depression is much greater than in rheumatism.

(2) *From Gout.*—This disease occurs after middle age, involves the smaller joints, and is followed by merely local sweats and desquamation. A deposit of chalk stones takes place in the joints if the attacks recur, and a similar deposit is also found in the lower part of the cartilage of the ear.

(3) *From gonorrhœal rheumatism* it is differentiated by the cause and history.

(4) *From Rheumatoid Arthritis.*—The latter disease occurs in debilitated subjects, especially women; is not attended by signs of inflammation or constitutional disturbance, and leaves the joints permanently deformed. Heart complications do not occur.

What is the prognosis?

The prognosis is favorable.

If the temperature rises to above 107° (*hyperpyrexia*), it is dangerous.

Cerebral rheumatism is dangerous, but is rarely seen.

Cardiac complications give a grave prognosis as to the future, but there is no immediate danger. They occur in about 50 per cent. of the cases.

What is the treatment of acute rheumatism?

The patient should be confined to bed, should have easily-digested food as in fevers, and the joints should be wrapped in cotton and kept warm. There are *two special plans of treatment.*

(1) *Treatment by Salicylic Acid or the Salicylates.*—This consists in giving the patient sodium salicylate, gr. xv–xx every two or three hours until two drachms have been taken in twenty-four hours, when the dose is reduced. Or in giving salicylic acid, gr. x every hour or two until six doses have been given, when the dose should be decreased or the intervals lengthened. Should disagreeable symptoms arise from the administration of these drugs (as nausea, tinnitus aurium, etc.), the medicine must be stopped. If no effect be observed within the thirty-six hours, the medicine should be abandoned. These drugs must never be given to patients who are weak or anæmic, or where cardiac complications are threatening. The salt is preferred to the acid, as less likely to cause disagreeable effects.

The oil of gaultheria, which contains methyl salicylate, has been successfully used as a substitute for the sodium salicylate. Salol has been also employed for the same purpose.

(2) *The Alkaline Plan of Treatment.*—This consists in giving potassium bicarbonate or acetate, gr. xl every two or three hours until one to two ounces have been administered in twenty-four hours, when the amount should be reduced one-half.

If the latter plan of treatment is adopted, quinine should be given in tonic doses after the first three days, as it appears to lessen the liability to relapse.

If there is much restlessness, ammonium bromide may be conjoined with the other treatment, or small doses of Dover's powder given at night. The latter is also serviceable when there is much pain.

During convalescence tincture of ferric chloride, with quinine, should be administered as tonics.

Locally, hot applications may be applied to the joints, as a hot solution of potassium nitrate (℥j) with laudanum (℥j) in water (Oj). Or, an ointment consisting of cocaine gr. xx in lanolin (℥j) may be applied and the part wrapped in raw cotton.

If endocarditis occurs, the alkaline plan must be substituted for the salicylates, or if the former is being employed, the dose should be doubled.

If pericardial effusion occurs, digitalis should be given, and a small blister applied over the heart.

For hyperpyrexia, a cold bath or the application of iced cloths, and sponging with ice water, may be used externally, or antipyrine administered internally.

For cerebral rheumatism the best treatment is stimulants and the administration of digitalis.

If the joints remain stiff and a permanent lesion is feared, small blisters may be applied over them.

Patients suffering from the rheumatic diathesis should always wear flannel next to their skins.

Muscular Rheumatism.

What is muscular rheumatism?

Muscular rheumatism is pain and stiffness in the muscular and fibrous structures due to exposure.

THE SYNONYMS vary with the situation. Thus, it is called *lumbago* when it affects the muscles of the back; *pleurodynia* when it affects the chest; *torticollis* or *wry neck* when it affects the sterno-mastoid and trapezius muscles, etc.

CAUSATION.

It is due to exposure to drafts, especially when heated. There is usually an underlying rheumatic diathesis.

THE DURATION is usually from three to seven days. But it may become chronic and last for months.

SYMPTOMS.

There is pain, stiffness and sometimes swelling in the affected muscles, without redness, little or no fever, and no cardiac complications. The pain is not continuous, but is produced by motion.

DIAGNOSIS.

The disease is recognized by the history of exposure to drafts, particularly if the patient is of a rheumatic diathesis. The pain remains in one situation, and is aggravated by motion.

DIFFERENTIAL DIAGNOSIS.

(1) *Myalgia*, or straining of a muscle, and is never accompanied by fever, and the history of strain gives a clue to the diagnosis.

(2) *Neuralgia.*—Here the pain is limited to the course of the affected nerve, and it is not influenced by motion. There are painful points on pressure over the affected nerve.

PROGNOSIS.

This is very favorable. When chronic it may be obstinate.

What is the treatment of muscular rheumatism?

In acute cases rest and warmth usually result in a speedy cure. Dry heat may be employed, as by a hot-salt bag, a hot-water bag, or, better, by passing a hot iron over the part, which should be protected by a doubled blanket.

Stimulating ointments or liniments may be used (as a liniment composed of equal parts of chloral and camphor thoroughly triturated together, and the resulting liquid diluted with three parts of soap liniment), and diaphoretics (as broken doses of Dover's powder and potassium nitrate) given internally.

If no improvement results with intwelve hours, phenacetin or salol may be administered or sodium salicylate (gr. xv–xx t. i. d.) given in powder or solution.

If the disease lingers or becomes chronic, iodide of potassium and colchicum, large doses of quinine, or of ammonium chloride (gr. x–xx) may be given.

Externally, belladonna plaster or pitch plaster with cantharides may be used. In very obstinate cases atropine (gr. $\frac{1}{80}$) with morphine (gr. $\frac{1}{8}$) may be used hypodermically, two or three times a week. Deep injections of chloroform or of warm water have also been employed with advantage.

Chronic Rheumatism.

What is chronic rheumatism?

It consists in enlargement of the joints, following repeated attacks of the acute disease, combined with muscular rheumatism, and producing permanent deformity. Heart lesions are also usually present.

How should it be treated?

Everything must be done to improve the nutrition of the patient. He should dress in warm flannel, and should use stimulating liniments containing ammonia, or chloral dissolved in soap liniment. Turkish baths or sulphur baths are of value. Internally, he may take alteratives, as potassium iodide, colchicum, sulphur, or cod-liver oil.

Rheumatoid Arthritis.

What is this disease?

It is a chronic disease consisting in a series of attacks of chronic inflammation of the joints, resulting in enlargement, anchylosis, dislocation, or other permanent deformity.

What are its synonyms?

Rheumatic gout, arthritis deformans.

What are its causes?

It is strongly hereditary. It occurs more frequently in women than in men. Bad hygiene, grief, anxiety, and overwork may favor its development in those who are predisposed to it.

What is the duration of this disease?

It lasts for years, only ending with the patient's life.

What are its symptoms and pathology?

A joint becomes stiff and swollen without fever or suppuration. The attack passes off, and again recurs in the same joint until it is destroyed, distorted, and generally anchylosed. Other joints are affected, and the patient finally becomes a helpless cripple. All this time there is little or no constitutional disturbance, except debility. The large and small joints are both affected.

Morbid Anatomy.

The articular cartilages are ulcerated, and become absorbed, or are destroyed. The synovial membrane is also destroyed, and the bones become eburnated by rubbing against each other. Suppuration never takes place.

On what is the diagnosis of this disease based?

It is recognized by being a chronic disease, affecting the large and small joints alike, without constitutional disturbance, and never being complicated with disease of the heart.

Differential Diagnosis.

(1) *From Gout.*—It is diagnosed from this disease by the history, by the absence of uric acid in the blood, by the absence of redness, by affecting the large and small joints, by the absence of

chalk stones, and the presence of anchylosis or other permanent deformity.

(2) *From Rheumatism.*—It is hardly likely to be confounded with acute rheumatism, and can be diagnosed from the chronic disease by the absorption of the cartilages, the anchylosis and deformity, the absence of involvement of the muscular system, the slow progress and the absence of disease of the heart.

What is its prognosis?

The sooner the treatment is commenced the better is the result obtained. If neglected the prognosis is terrible, the patient becoming a helpless cripple, unable even to feed himself.

How should it be treated?

The patient must have the best possible food, which must be nourishing and even stimulating. His hygienic surroundings should be of the best character, and he should be given alteratives, as cod-liver oil, arsenic, iodides of iron, of potassium, or compound solution of iodine. At the same time the skin should be acted on with Turkish baths. Locally, leeches, iodine and blisters may be applied to the affected joints, and massage and manipulation of the joints should never be neglected. If anchylosis takes place the patient should be etherized and the adhesions broken up.

Acute Gout.

What is gout?

It is an hereditary disease characterized by severe pain and swelling, occurring in one of the smaller joints, an excess of uric acid in the blood, and the deposit of chalk stones (sodium urate) in the joints and cartilages.

SYNONYMS.

When it occurs in the foot (the most frequent seat) it is called *podagra;* in the hand, *chiragra;* in the knee, *gonagra.* It is an old disease, alluded to by the later classics.

CAUSES.

It is strongly hereditary, and affects men more frequently than

women. It occurs particularly in those who lead a sedentary life, and indulge largely in animal food, especially when eaten hurriedly. Wines, particularly sweet wines, and malt liquors in excess, when indulged in habitually, lead to acute attacks. Strong mental emotion, as anger, may excite an attack. In addition to these causes, an excess of uric acid in the blood is a prime factor in the development of the attack.

Acute gout is not common in this country, but is met with especially in England and the beer drinking countries of Europe, as Bavaria.

What are the symptoms of acute gout?

It begins usually at night with intense pain in the ball of the great toe (the metatarso-phalangeal joint). The pain is aggravated by pressure and by vibrations, as those produced by heavy wagons in the street. The patient is restless and feverish; next morning the pain is lessened, but the joint is red, swollen and glossy, and this redness extends to the other joints. The swelling somewhat relieves the pain, which, however, increases toward night. The small joints of the other foot or of the hand may be also affected. This continues for five or ten days, and slowly disappears, followed by slight desquamation. During the paroxysm the urine is deficient in uric acid, there is an extraordinary irritability of temper, nausea and constipated bowels. After the acute attack passes off the patient is much better than he was before it came on.

How is gout diagnosed?

Gout is recognized by the sudden onset with excruciating pains, by affecting the small joints only, by the fever, the deficiency of uric acid in the urine, and the absence of cardiac complications.

What is the prognosis of gout?

The prognosis of the attack is favorable; but it is apt to recur at intervals. Occasionally *retrocessant gout* is seen, which is dangerous. *Gout of the brain* is rare, but is a dangerous complication. Generally the cerebral symptoms are rather due to the alcohol which the patient has habitually imbibed than to the gout. The same may be said of *gout of the stomach*. *Gout of the heart* is characterized by pain and intermittency. It occurs in old persons and gives a bad prognosis.

How should gout be treated?

An acute attack of gout should be treated by rest, warmth, elevation of the parts, and quiet. Internally, colchicum given in small doses, to avoid purgation (wine of the root, gtt. x–xx), every three or four hours, in neutral mixture, affords most relief. This should be given during the acute attacks, and not in the intermission between them. A little opium in the form of Dover's powder may be given at night, to allay pain and cause sleep. Diaphoretics, mild diuretics and an occasional cathartic are also indicated. Locally, warm applications should be applied to the part. Cold externally often gives quick relief, but is very dangerous, as it frequently produces retrogression of the gout.

Chronic Gout.

What is chronic gout?

Chronic gout is a condition occurring from repeated attacks of the acute disease, characterized by enormously enlarged and swollen joints, and swellings occurring under the tendons, from deposit of chalk stones, which may give rise to ulceration and abrasion of the skin, leading to their discharge.

How is it diagnosed?

It is diagnosed by the history of the preceding acute attack, by the deposit of chalk-stones in the smaller joints and cartilage, particularly the cartilage of the ear.

How should it be treated?

The patient should be placed on a vegetable diet, consisting of milk, fish, or oysters, but no meat, no sweet wines, no malt liquors, and no pastry or sweetmeats. If possible, he should avoid all alcoholic beverages, but *if he must* drink, he should take a little claret, or a little whiskey or brandy with water, at his meals. The action of the skin and liver should be maintained by exercise in the open air (walking, horse-back riding, etc.), Turkish baths, etc. Colchicum does no good in chronic gout, but should be kept for the acute attacks. The alkalies, and especially the lithium preparations and the alkaline mineral waters, as Vichy, Buffalo lithia water, Poland water, etc., should be given.

Lithæmia.

What is lithæmia?

Lithæmia is a condition of undeveloped gout characterized by an excess of uric acid in the blood, giving rise to various disturbances of the system, but not attended by an acute attack of gout.

What are its synonyms?

Uric acid diathesis, lithiasis.

What are its causes?

These are the same as those which produce gout.

What are the symptoms of this disease?

The symptoms of lithæmia vary according as one or another organ bears the brunt of the disease. Usually there is great depression of spirits, numbness in the extremities, with pain and slight enlargement of the smaller joints. High colored, scanty urine, with abundant deposit of uric acid, which may contain a small amount of sugar. Impaired digestion, flatulency, irregularity of the bowels, and perhaps symptoms referable to chronic gastric catarrh, chronic hepatic hyperæmia, or even to contracted kidney or interstitial hepatitis. Sometimes there are no joint symptoms; sometimes persistent vertigo or intense headache, or neuralgia, lasting for several days at a time.

What is the prognosis of this disease?

If promptly recognized and properly treated, recovery will be the result. The disease is usually, however, of long duration.

How should it be treated?

The treatment is the same as that of chronic gout.

Diabetes.

What is diabetes?

Diabetes, or *Glycosuria*, is a chronic disease, characterized by the passage of large quantities of urine, which contains glucose as a constant ingredient, and associated with progressive emaciation and loss of strength.

What are the causes of this disease?

It is often hereditary and occurs at all ages, though most frequently in early middle-life. The cause of the excessive formation of sugar in the system is not thoroughly understood. It evidently is not formed in the kidneys, but, being excreted by them, sets up an irritation in its passage through those organs which results in the greatly increased amounts of water which they eliminate. It exists in the blood, and in some instances would appear to be due to excessive formation of glycogen by the liver; in other instances the affection seems connected with disease of the pancreas, while in other instances it appears to arise in connection with some disease or irritation in the neighborhood of the floor of the fourth ventricle of the brain.

What are the symptoms of diabetes?

The amount of urine passed in twenty-four hours is often enormous,—generally about two to three or four quarts,—sometimes very much more, amounting occasionally to several gallons. On the other hand, the quantity of urine is sometimes not increased at all. *Among the earlier symptoms* are, inordinate thirst; great dryness of the throat; a tendency to the formation of boils, and a great itching of the skin. In women there is intense pruritus vulvæ, and in men much pain and itching in the penis is complained of, owing to the development of the penicilium in the few drops of urine which remain about the parts after urination.

Later in the disease, the thirst, which was present as an early symptom, increases and becomes almost unbearable; the urine increases in amount, and the quantity of sugar it contains is usually much increased also; the bowels are constipated, because all the water that the system can spare is being excreted as urine; the skin feels harsh and dry, and cataract develops in one or both eyes; various neuralgiæ occur and give rise to much additional suffering; bone diseases, as periostitis, caries or necrosis may take place, and the patient often suffers, in addition, from attacks of gout. There is much emaciation, and the general health and spirits of the patient suffer greatly.

Special Symptoms.

The urine is clear and pale; it has a sweetish taste and odor, a

high specific gravity (1028–1050, the average being about 1030), which will vary with the amount of sugar passed and its degree of dilution. Sugar can be detected if the proper tests are applied to the urine, the best of which, as rough tests, are Boettger's, Moore's or Trommer's. If, however, it is desired to determine accurately the quantity of sugar which is being passed daily, Pavy's or Fehling's tests must be used, or Roberts' fermentation test resorted to. The quantity of sugar excreted daily varies very much. Usually it is equal to about thirty per cent. of the urine voided, or from one ounce to two pounds or more.

The nervous system also suffers. Not only is the patient irritable, but he suffers from nervous debility and loss of nervous tone. He is often vacillating, where prior to the disease he was firm.

The respiratory system shares in the general malnutrition. There is a cough from bronchial catarrh, and as the disease progresses, fibroid changes, and finally tubercular deposits take place in the lungs.

The circulation is languid, and the first sound of the heart feeble.

The temperature is lower than in health.

What is the duration of this disease?

The duration varies. Under the improvements which have been made in the treatment, the duration is gradually lengthening. Formerly, according to Prout, the average duration was about two years.

How does it terminate?

The majority of the cases terminate fatally, from exhaustion, or from decomposition of the sugar in the blood, which, being converted into acetone, causes either *diabetic coma* (the patient becoming soporose and finally comatose, with dilated pupils and a cold surface) or delirium and convulsions.

What is the prognosis?

Probably the disease never ends in recovery, although much can be done by treatment to retard its progress. Any acute intercurrent affection greatly increases the danger.

How is this disease diagnosed?

The symptoms and the presence of sugar persistently in the urine render the disease unmistakable. If a patient with a chronic disease is suffering with great thirst, has a dry mouth and tongue, and intense

itching of the skin, the urine should always be examined for sugar, particularly when there is a tendency to the formation of successive crops of boils. It should always be remembered that *the presence of glucose in the urine, without it is persistent, does not indicate diabetes.* A temporary glycosuria may occur from many causes, as excessive sexual indulgence, and even from eating heartily of certain articles of food, as asparagus, or following the inhalation of chloroform.

What is the treatment of this disease?

It is of the utmost importance that all starchy matters and sugars should be excluded from the patient's food. *He must scrupulously avoid sugar of all kinds, and such articles as contain starch*, as bread, potatoes, peas, and beans, rice, etc., *if he wishes to live.* Glycerin or saccharin may be used as a substitute for sugar, but it is hard to find a substitute for bread. He must live on an animal diet almost exclusively. He should be warmly clad, use warm baths and occasionally take a Turkish bath, to keep the skin acting, and render it pliable.

Opium seems to exert some effect upon the excretion of sugar, and upon the amount of water passed, but the patient will become a hopeless opium-eater, and the influence of the drug is usually not maintained. Codeine has been highly recommended, and appears to act very favorably upon the disease. Ergot is also useful, and sodium salicylate has been employed with advantage, but *the regulation of the diet is the most important part of the treatment.*

Polyuria.

What is polyuria?

It is a disease characterized by extreme thirst, emaciation, debility and the passage of immense amounts of pale, limpid urine, free from either albumen or sugar.

What are its synonyms?

Chronic diuresis; diabetes insipidus.

What are its causes?

The causes are very obscure. It seems to be produced by some irritation of the nervous system in the region of the floor of the

fourth ventricle, and may follow sunstroke, exposure to cold, malaria, syphilis, etc.

What are its symptoms?

There is great thirst and voracious appetite; the skin is dry and harsh, the bowels constipated, and an enormous quantity of pale urine of low specific gravity (1001–1007) is passed, which does not contain either albumin or sugar. The patient is nervous, irritable, and acquires a curious tendency to inaccuracy in his statements, so that a man who was absolutely truthful before, can hardly be relied upon, after he is afflicted with this disease. There is also inability to concentrate the mind for any period of time upon one object, and severe headache, usually frontal or occipital. Very soon the patient becomes so much weakened, that any acute intercurrent affection is apt to prove fatal.

How is this disease treated?

The amount of water taken by the patient should be limited. This is much easier to order than to have carried out, as the patient will drink whenever he has the chance and deny the fact when questioned about it. Patients suffering from this disease have been known to drink their own urine, the thirst is so intense. Ergot and potassic bromide seem to give most relief, except when the patient is suffering from syphilis (which may, perhaps, be a cause of this, as of so many other affections), in which case potassic iodide and mercury are of more avail. Pilocarpine has also been used successfully. Galvanism has been recommended. Tonic treatment must also be employed, no matter what other remedies are given, and the bowels should be properly regulated.

DISEASES OF THE BLOOD.

Anæmia.

What is anæmia?

Anæmia is a disease of the blood characterized by a deficiency of albumen and red corpuscles.

What is its synonym?

Oligæmia.

What are the causes of anæmia?

(1) Predisposing Causes.

Women are more frequently affected than men. Very young and very old persons are particularly susceptible. Nervous, irritable, and hysterical persons are especially predisposed to it.

(2) Exciting Causes.

Bad hygiene, poor diet, a want of exercise on the one hand, or excessive exercise, great grief or other depressing emotion, drains on the system as from some chronic discharge, poisons circulating in the blood, as the poison of malaria or the metals, disease of the blood-making organs as the spleen or lymphatic glands, and excessively cold weather, all act as exciting causes.

What are its symptoms?

There is great pallor; the tongue, gums, and cartilages of the ears when held against the light, and the conjunctiva are all pallid. There is great muscular weakness, a frequent pulse, with a soft blowing systolic murmur heard over the heart; a venous hum in the jugular veins, frequent respiration, with breathlessness on slight exertion, irritability of temper, dizziness, frequent fainting fits, causeless vomiting, and dropsy (a late symptom). In women menorrhagia is frequently present.

What are the sequelæ of anæmia?

The sequelæ of anæmia are degenerations of various viscera, due to want of nutrition, thus *fatty heart* and *ulcer of the stomach* are frequently seen; *softening of the brain* is among the rarer sequelæ of this disease.

How is this disease diagnosed?

This disease is known by the pallor and pearly hue of the conjunctiva, the rapid pulse, the heart murmurs, and the venous hum in the jugular vein.

What is the prognosis of anæmia?

The majority of cases recover, but suffer from relapses at critical times, as during pregnancy, or at the menopause. Organic changes due to malnutrition render the prognosis less favorable.

What is the treatment of anæmia?

The patient must be placed upon a good diet, and under the best hygienic conditions. She must have plenty of fresh air and sunlight, cheerful surroundings, and properly regulated exercise. Any obvious cause must be removed if possible, and iron given in rapidly increasing doses until the point of tolerance is reached. This is known by the supervention of headache, slight vertigo, coated tongue, nausea and sometimes vomiting, and a slightly elevated temperature. The tincture of the chloride of iron is the best preparation if the digestive organs are in good condition. The potassio-tartrate, lactate or citrate may be used if the digestion is feeble. Cod-liver oil and the extract of malt may be combined with the above treatment if they do not disorder the digestion.

If there is much gastric disturbance, the bitter tonics combined with a mineral acid should be administered, and the preparation of iron deferred until the condition of the patient has somewhat improved.

If there is a tendency toward hemorrhages, a liberal diet, and the use of bitter tonics will give a better result than the treatment by iron.

If there is *profound anæmia due to loss of blood*, transfusion may be resorted to.

Essential Anæmia.

What is essential anæmia?

It is a profound form of anæmia, pursuing a progressive course in spite of treatment and almost invariably terminating fatally.

What are its synonyms and causation?

It is also called idiopathic anæmia and progressive pernicious anæmia.

The cause is not well understood; it may be due to disease of the spleen, lymphatic glands or bone marrow. It is more common in the female than the male, and seems particularly liable to affect pregnant women.

What are the symptoms of this disease?

In the early stages the symptoms are those of anæmia much

intensified, but the patient does not lose much flesh. Later on internal hemorrhages take place, as intestinal hemorrhage, or hemorrhage into the retina; slight albuminuria is also present, and irregular accessions of fever occur. After a time there is almost continual fever, the temperature, however, not rising very high, with slight morning remissions and evening exacerbations. After the fever has continued three or four weeks the difference between the evening and morning temperature becomes more marked. The patient now begins to lose flesh, is extremely weak, dropsy supervenes, and death occurs usually from exhaustion.

What is the pathology of this affection?

The *corpuscles* and *albumen* of the blood are relatively diminished; the blood is therefore more watery than normal, but the relation between the red and white corpuscles is unaltered.

What does a post-mortem examination reveal?

No constant lesion is found; the *heart* may have undergone fatty degeneration from malnutrition, the *spleen* is sometimes enlarged and thickened, the *lymphatic glands* may or may not be normal, the *marrow of the bones* may have undergone degeneration or may be healthy, the *gastric tubules* may be diseased.

What is the prognosis?

This disease almost invariably terminates in death.

How should it be treated?

The patient must be placed on a properly regulated diet, and should have change of scene, and live in the open air. Iron, phosphorus and cod-liver oil may be administered internally, and treatment should be directed to any symptoms which seem to require it. Transfusion does no permanent good. By proper treatment we may prolong the life of the patient, although he cannot be cured.

Leukæmia.

What is leukæmia?

Leukæmia is a disease of the blood, characterized by an enormous increase of white corpuscles.

What is its synonym?

It is also called leucocythæmia.

What is its cause?

It is caused by some disorder of the blood-making organs. Generally there is some disease of the spleen, sometimes of the lymphatic glands, or lymphoid cells, and sometimes of the bone marrow.

What are the symptoms, duration and pathology?

THE SYMPTOMS are those of anæmia, with enlargement of the spleen and often of the lymphatic glands. A hemorrhagic diathesis is developed late in the disease, and bleeding occurring from the stomach or bowels, the anæmic symptoms becoming more profound. Anomalous febrile attacks are now seen, bronchial catarrhs and diarrhœas often take place, with curious alterations of the special senses, great depression, dropsy, and death from exhaustion.

DURATION about two years.

PATHOLOGY.

The *white blood corpuscles* seem to be formed instead of red corpuscles, or perhaps fail to be transformed into the latter. They are enormously increased in number, and may even be as numerous as the red.

POST-MORTEM.

The blood is white in appearance, has a low specific gravity, and forms soft white coagula in the right side of the heart. The *lymphatic glands* all over the body are frequently enlarged. Sometimes new glands seem to be formed (lymphadenomata).

What is the diagnosis, prognosis and treatment of this disease?

DIAGNOSIS.

Under the microscope more than twenty white corpuscles are found in each field. *In the lymphatic form*, the microscope shows in addition numerous free cells and small nuclei. If it is due to *disease of the marrow of the bones* pain will usually be complained of on pressure over the sternum, tibia and other bones.

PROGNOSIS.

The prognosis is usually favorable as regards immediate danger.

The patient may live comfortably for a long time, if the white corpuscles do not exceed one-fifth of the whole number of cells. If they are in excess of 50 per cent., the prognosis is extremely bad.

TREATMENT.

The patient must be well fed, placed under the best hygienic conditions, and given iron and quinine as tonics.

In the lymphatic form, Lugol's solution or potassium iodide in small doses, long continued, sometimes does good. Sea-bathing and sea-voyages are also employed with advantage.

In the splenic variety, if it is seen in the early stages, ergot administered hypodermically is sometimes of value.

In the later stages of the disease the patient's strength should be maintained by all possible means.

Pseudo-Leukæmia.

What is pseudo-leukæmia?

Pseudo-leukæmia is a blood affection, characterized by a high degree of anæmia in connection with disease of the lymphatic and often of the mesentery glands, but without increase of white corpuscles.

What are its synonyms?

It is also called Hodgkin's disease and lympho-sarcoma.

What are its causes?

It develops without known cause. It is not hereditary. It is more frequently seen in males than in females, and in young rather than old people.

What are the symptoms and duration of this disease?

The symptoms are those of profound anæmia,—extreme pallor, breathlessness, altered nutrition, emaciation and dropsy. In addition, lymphatic tumors are found all over the body, which are not painful on pressure. There is a tendency to hemorrhage and diarrhœa; but rarely, if ever, is there an increase of white corpuscles.

The duration is about two years.

What is the diagnosis, prognosis and treatment?

DIAGNOSIS.

The disease is known by the glandular enlargements with anæmia, but without an increase of white corpuscles.

(1) *From Leukæmia.*—The white corpuscles are not increased. In leukæmia the changes in the blood come before the changes in the glands, in pseudo-leukæmia the changes in the glands precede the changes in the blood.

PROGNOSIS.

It is more favorable than leukæmia. The disease is not very amenable to treatment, but can frequently be arrested, if not cured.

TREATMENT.

Good hygiene, good diet, and plenty of fresh air are indispensable. Internally, cod-liver oil, iodine and iron may be given in combination or alternately. *In the latter stages*, chloride of gold (gr. $\frac{1}{12}$ – $\frac{1}{6}$ in glycerin and water), freely diluted, three times a day, may prove of service. *For the enlarged glands*, iodine or the iodides produce the best results.

Addison's Disease.

What is Addison's disease?

Addison's disease is a grave form of anæmia, associated with marked nutritive disturbance, and frequently with degeneration of the suprarenal capsules.

What is its cause?

It appears to be most frequent in those subjected to great physical or mental strain.

What are the symptoms, duration and pathology of this affection?

The SYMPTOMS are those of great anæmia. A peculiar bronzing of the skin frequently occurs, but is not constant. It appears on the face and in the folds of the body, at first in patches, later becoming more general and gradually almost uniform over the surface. There is pain in the back; gastric and intestinal irritation, more marked than in other blood affections; a tendency to dropsy as the

anæmia increases, and, later, symptoms referable to the cerebro-spinal and sympathetic nervous system, with death from anæmia or malnutrition in about eighteen months.

PATHOLOGY.

In addition to the deficieney of red corpuscles and albumin *in the blood*, the *suprarenal capsules* undergo a special strumous degeneration. It is not every disease of the suprarenal capsules that produces this affection.

What is the diagnosis, prognosis and treatment of Addison's disease?

DIAGNOSIS.

The disease is recognized by the anæmia, the bronzing of the surface, and the gastro-intestinal disorders.

PROGNOSIS.

Few persons affected with this disease live longer than eighteen months.

TREATMENT.

Good hygiene and good food are as useful in this as in other diseases of the blood. A moderate amount of stimulus, especially the light red wines, as claret, with meals are of some service. Tincture of ferric chloride, combined with glycerin, is of use, and phosphorus, strychnine, or the chloride of gold and sodium, may also be employed.

Scorbutus.

What is scorbutus?

Scorbutus is a disease characterized by anæmia, malnutrition, ecchymoses, a spongy condition of the gums, which bleed readily on the slightest touch, physical and mental lethargy, and which is produced by improper diet.

What is its synonym?

It is commonly called scurvy.

To what is it due?

It is *caused* by abstinence from fresh vegetables, especially those which contain large quantities of the salts of potash, as the potato

or cabbage. Any depressing influence also acts as an exciting cause in those predisposed to it by improper diet. It has occurred in epidemics, especially on shipboard and among armies. Scurvy was known to the ancients.

What are its symptoms, duration and pathology?

THE SYMPTOMS come on gradually. At first there are general symptoms of anæmia; the patient is easily fatigued, feels weak; is breathless on the slightest exertion; is depressed in spirits; suffers from rheumatic pains in the back, and is less able to resist exposure to cold. These symptoms last from one week to several months. The gums now become dark-bluish, congested, spongy, protrude between the teeth and bleed readily on the slightest touch. The teeth are loosened; the breath fœtid; palpitation occurs on exertion; petechiæ and ecchymoses appear on the surface; hemorrhages occur from the internal organs; old cicatrices and wounds open; old fractures become disunited, and the spleen enlarges; albumin appears in the urine, and the urine itself is diminished in amount and its solids are relatively decreased.

DURATION.

This disease is usually protracted, the duration is largely influenced by hygiene, diet and mental emotion, as depression or hope.

PATHOLOGY.

The red corpuscles and solids of the *blood* are diminished; petechiæ and ecchymoses are found in the skin and internal organs; *serous effusions* are found in the cavities of the body and connective-tissue spaces; *the spleen* is enlarged and softened.

What is the diagnosis, prognosis and treatment of this affection?

THE DIAGNOSIS is made from the history, the spongy gums, the ecchymoses, the lassitude and the diet on which the patient has been living.

(1) *From mercurial poisoning*, it is differentiated by the fact that *salivation never occurs in scurvy*, although the gums are spongy and discolored, and the teeth loosened.

PROGNOSIS.

The prognosis is favorable if the case is properly treated.

TREATMENT.

(1) PROPHYLAXIS.—A diet of fresh vegetables, as potatoes, raw cabbage, sauer kraut, etc., or lemon-juice, will prevent the development of this disease. Lemon or lime-juice should always form part of the rations of sailors who are engaged in long voyages, as in Arctic expeditions.

(2) MEDICINAL TREATMENT.—When the disease is fully developed, the mineral acids, with lemon-juice and tincture of the chloride of iron, must always be administered. Ergot is also employed to arrest the hemorrhages. A change in diet is imperative.

Locally, for the spongy condition of the gums, astringent mouth-washes and gargles should be employed.

Purpura.

What is purpura?

Purpura is an acute disease, characterized by a hemorrhagic tendency, which manifests itself by ecchymotic spots occurring under the skin, and sometimes by internal hemorrhages.

What is its causation?

It occurs in all seasons of the year and under all conditions of life. It is independent of diet, and is probably due to vasomotor paralysis and a deficiency of the fibrogenous materials of the blood,

What are the symptoms, varieties and pathology of this disease?

THE SYMPTOM is hemorrhage; it may take place under the skin, forming petechiæ, ecchymoses, etc.; or it may occur from a mucous surface, as the bladder or stomach. There is slight fever, and malaise, but no debility except when the loss of blood is great.

VARIETIES.

(1) *Purpura simplex*, where petechiæ only occur.

(2) *Purpura hæmorrhagica*, where there are other hemorrhages also.

PATHOLOGY.

The *blood* is altered in structure and in composition, the fibrin-

forming elements being diminished. The *arteries and capillaries* are often diseased, so that the blood more readily leaves them, and, possessing less inherent power of coagulation, a tendency to hemorrhage is developed.

What is the prognosis and treatment of purpura?

THE PROGNOSIS of *purpura simplex* is favorable, but relapses are frequent. *Purpura hæmorrhagica* is unfavorable in proportion to the hemorrhage.

TREATMENT.

Sustain the patient's strength by good diet and stimulants. *For the tendency to hemorrhage*, give ergot internally or hypodermically in small doses, frequently repeated, combined with sulphuric acid or Monsel's solution. The oil of turpentine (gtt. x every one or two hours) is an excellent remedy.

DISEASES OF SPECIAL ORGANS.

(A.) DISEASES OF THE DIGESTIVE ORGANS.

(1) DISEASES OF THE MOUTH, FAUCES, AND PHARYNX.

Stomatitis.

What is stomatitis?

Stomatitis is an acute inflammation of the mucous membrane of the mouth. It may be either *catarrhal*, *follicular* or *ulcerative*.

What are the causes of these affections?

They are caused by the introduction into the mouth of irritating substances; by disorders of the stomach, want of cleanliness, teething; or they may occur as secondary affections, following the acute exanthemata. Ulcerative stomatitis is particularly seen in children where the hygienic surroundings are poor, and is sometimes epidemic, perhaps even contagious.

What is the pathology of these affections?

In catarrhal stomatitis the mucous membrane of the mouth and the tongue is deeply congested; the tongue is usually swollen and

indented with the teeth, and the secretions of the mouth are at first diminished, afterwards much increased, the parts being covered with a tenacious mucus.

In follicular stomatitis, small, white elevations appear on the gums, lips, cheek, tongue or roof of the mouth, surrounded by a distinct zone of inflammation. These may remain separate or coalesce, but after a few days they rupture and leave a superficial ulcer, which heals slowly.

Ulcerative stomatitis consists in the deposit, in patches, of a false membrane on the gums, which are much congested. This substance softens and breaks down, leaving an irregular ulcer, with an unhealthy base, which tends to spread.

What are the symptoms of stomatitis?

There is burning pain in the mouth, increased by taking food or talking; the mouth is hot, and in the two latter varieties salivation occurs; there is some fever, more marked in the severer forms of the disease; the breath is offensive, and diarrhœa, or in the latter variety entero-colitis, is of frequent occurrence. Fever is present, varying with the severity of the case. In ulcerative stomatitis the submaxillary glands are swollen and tender.

How is this disease recognized?

If the mouth is carefully examined, it is impossible not to recognize the affection.

What is the prognosis?

The prognosis is favorable if the case is properly treated, even in the severer varieties.

How should it be treated?

The cause should be sought, and, if possible, removed. Great attention must be paid to the diet, to cleanliness, and to the state of the digestive tract, and potassium chlorate gargles may be employed.

In follicular stomatitis, in addition, the ulcers should be touched with a strong solution of silver nitrate, while in the ulcerative form strong nitric acid may be used locally, and quinine and stimulants given internally.

Muguet.

What is this disease?

Muguet, thrush or sprue is an inflammation of the mouth, probably due to the growth of a vegetable parasite which is always present in these cases, and is called the *oïdium albicans*. It occurs especially in infants, and is favored by unhygienic conditions, as by want of care in cleansing the bottles from which the child nurses.

What are its symptoms?

The symptoms are identical with those of the other forms of stomatitis, viz.: pain increased on eating, increased saliva, fœtid breath and diarrhœa, the stools being greenish and of an acid reaction. The mucous membrane of the mouth shows, on examination, patches of marked congestion which are the seat of whitish, curdy deposits, consisting of epithelium and the fungus, and which resemble curdled milk. These deposits coalesce and spread from the lips and mouth into the pharynx and often into the œsophagus.

What is the treatment of muguet?

Absolute cleanliness, not only of the parts, but of the nursing-bottles and tubes as well; washing the baby's mouth after each feeding with some mild disinfectant, as borax, and paying scrupulous attention to the digestive system and to the secretions, usually effects a speedy cure.

Glossitis.

Describe glossitis.

It is an acute or chronic parenchymatous inflammation of the tongue, characterized by enlargement and pain, occasioning difficulty in articulating, and in chewing or swallowing the food.

CAUSES.

Acute glossitis is due to some chemical, mechanical or thermal irritant; chronic glossitis is usually localized, and is produced by some local irritation, as from a broken tooth.

SYMPTOMS.

In acute glossitis there is fever, rapid pulse, intense congestion and swelling of the tongue, which is often very great, sometimes so

much so that it cannot be contained in the mouth and protrudes as a large inflamed mass beyond the lips; the mouth is hot, there is much pain, and the salivary secretion is increased. If the patient can articulate at all the voice is muffled and indistinct; deglutition is interfered with and causes much suffering; the cervical glands are often enlarged and dyspnœa is usually marked. Sometimes suppuration occurs.

Chronic glossitis is usually confined to the border and edges of the tongue. The symptoms are less marked and there is no fever.

PROGNOSIS.

Acute glossitis usually terminates favorably. Occasionally suffocation occurs, ending fatally. Chronic glossitis is a protracted and often an incurable affection.

TREATMENT.

Remove the cause if possible. In acute glossitis the fever and constitutional symptoms must be treated by a fever mixture containing aconite. The tongue should be incised freely, and if the swelling is great, the incisions should extend deeply into its substance. The application of cold in the early stages, or of hot water when the disease is further advanced, often affords relief. If there are symptoms of impending suffocation, laryngo-tracheotomy must be promptly performed.

Acute Tonsillitis.

What is acute tonsillitis?

It is an inflammation of the tonsils, with a tendency to suppuration.

What is its synonym?

It is also called *quinsy*.

How is this disease produced?

It is sometimes epidemic, especially in the damp cold weather of the Spring and Fall. In those who are predisposed to it, any exposure to inclement weather is apt to produce an attack. One attack predisposes to another in the same individual.

What are the symptoms and duration of this affection?

It generally begins in one tonsil, but soon both are alike involved. There is sore throat, difficulty in swallowing and in breathing, fever, and upon examining the throat, one or both tonsils are seen to be red and swollen, and sometimes so much so as almost to close the passage of the fauces. After lasting five or six days the disease terminates, usually in suppuration, although sometimes resolution takes place and no pus is formed. When suppuration occurs all the symptoms rapidly subside as soon as the pus is evacuated. The bowels are usually constipated throughout the attack.

How is this disease diagnosed?

The diagnosis is easy. The fever, difficulty in swallowing and in breathing, and an examination of the throat, which reveals the tonsils red and swollen, and shows the absence of membrane on the pharynx (which is only slightly, if at all, inflamed), with the absence of sufficient depression and constitutional involvement to denote diphtheria, all render the diagnosis clear and positive.

What is the prognosis?

The prognosis is favorable. In a very few cases death has occurred from rupture of the abscess during sleep, and escape of the pus into the larynx, causing asphyxia.

How should it be treated?

(1) PROPHYLAXIS.

Persons who are subject to attacks of tonsillitis should keep the tonsils constringed with local applications of astringents (as solutions of zinc or tannin, or Monsel's solution diluted with water), and avoid exposure to damp.

(2) ABORTIVE TREATMENT.

If the case is seen very early in the attack, a prompt emetic will sometimes abort it.

(3) GENERAL TREATMENT.

When the disease is fully developed, the mouth and throat should be kept disinfected by means of gargles of boric acid, thymol, or potassium permanganate; no astringents or caustics should be used in this stage. Heat applied externally, or hot gargles of milk and

water, or of potassium chlorate, hasten suppuration and often ease the pain in some degree.

Guaiac is often very efficacious, but sometimes signally fails to afford any relief. It is employed in the form of the ammoniated tincture, in small, frequently repeated doses. When it is well borne by the stomach, it is generally useful. Pilocarpine is also employed in doses sufficiently large to cause slight salivation and diaphoresis. When there is much swelling or great difficulty in swallowing, scarification with a bistoury should be promptly resorted to. Iron and quinine may be given throughout the attack, and should be administered at any rate as the inflammation subsides. The bowels should be moved daily by a saline, as Seidlitz powder.

If the patient complains of much weakness, milk punch may be ordered, giving from ℥ss–iij of whiskey in twenty-four hours.

As soon as pus forms it must be evacuated.

Chronic Tonsillitis.

What is this affection, and how is it treated?

This is the condition which results from repeated attacks of the acute disease, and consists of chronic sore throat and an enlargement of the tonsils from hyperplasia of the connective and glandular structures which compose that organ.

The treatment is rather surgical than medical, and consists in the frequent application of astringents, or, if the enlargement is great or shows a tendency to resist treatment, in the removal of a portion of the tonsil with scissors, a bistoury, or, better, with the tonsillotome.

Angina and Pharyngitis.

What are these affections?

They consist of acute or chronic inflammation of the fauces and upper part of the pharynx, characterized by sore throat, difficulty in swallowing, hawking, some cough, and more or less change in the voice, which generally has a nasal twang.

What are the causes?

They are generally produced by exposure to cold and damp; sometimes by mechanical or thermal irritation; sometimes by disorders of the alimentary canal, especially by disorders of the stomach and upper part of the bowel; and occasionally they are intercurrent in other acute diseases, as the exanthemata.

What are the symptoms?

The acute disease begins suddenly with dryness of the fauces and throat, fever, perhaps preceded by chilliness or an actual chill, pain on attempting to swallow, and, when the pharynx is involved, a constant desire to clear the throat, and a nasal intonation when speaking. There is much thirst. As the inflammation progresses, the secretions, which are at first scanty become reëstablished and profuse, and give rise to a constant desire on the part of the patient to swallow, each attempt being attended with acute pain. Earache is a frequent concomitant of this stage of the disease due either to involvement of the Eustachian tube in the inflammatory action or to plugging of the tube by mucus.

When the throat is examined, the mucous membrane of the soft palate, fauces and posterior pharyngeal wall is seen to be much congested and swollen, the uvula elongated and the parts at first dry, afterwards covered with a thick tenacious mucus. Sometimes little white membranous patches are seen on the surface, which, however, do not tend to spread or to coalesce. Small ulcers are also sometimes seen.

Inflammation of the lower part of the pharynx is a rare affection.

Chronic sore throat results from repeated attacks of the acute disease, and is apt to be converted into the acute trouble at any time, on exposure to cold and damp. There is habitual cough, constant tendency to clear the throat, lengthening of the uvula, thickening of the posterior half-arches, and in some cases, enlargement of the mucous follicles. This latter form is *follicular pharyngitis* and is usually called "*clergymen's sore throat.*" The follicular enlargement may even extend into the larynx.

What is the treatment of these diseases?

In the acute attacks a fever mixture, containing, if there is much elevation of temperature, a few drops of aconite in each dose, is

beneficial. Sometimes the severity of an attack can be materially lessened by pilocarpine administered in the very early stages, or by repeated minute doses of morphine combined with very small doses of tartar emetic.

Locally, in the early stages, demulcents, as a solution of acacia flavored with lemon and sugar, are very grateful to the patient, and small pieces of ice allowed to dissolve in the mouth, tend to allay the inflammation. Spraying the throat with Dobell's solution or applying a weak solution of cocaine is also advantageous. Strong solutions of silver nitrate protect the surface when applied to it locally, and in this way afford much relief, as well as tend to relieve the congestion by their astringent effect upon the tissues and blood vessels, but within the last few years it has been claimed that this treatment does harm, as the salt acts as a superficial caustic, and produces permanent alterations in the tissue to which it is applied.

As the acute stage is subsiding mild astringent gargles, as tannin or alum, are very useful. Gargles with potassium chlorate may be employed in any stage.

Chronic sore throat and the chronic forms of pharyngitis require rest for the voice, and avoidance of such irritation as is produced by smoking, for example. Change of air is often beneficial. Tonic treatment is usually indicated. The sufferer should avoid all exposure which is apt to lead to an acute exacerbation, and should employ strong astringent gargles, as alum, or have the parts painted with zinc sulphate in solution (ʒj–fʒj) or with tannin glycerole, several times a week. If the follicles are enlarged, and this treatment does not succeed, it may be necessary to destroy them by means of the galvano-cautery.

(2) DISEASES OF THE ŒSOPHAGUS.

Œsophagitis.

What is œsophagitis?

Œsophagitis is an acute inflammation of the œsophagus resulting from chemical, mechanical or thermal irritants, characterized by great pain and difficulty in swallowing, hiccough, intense burning in the throat and between the shoulders, fever and great anxiety.

On inspection the mucous membrane is seen to be vividly red and swollen.

How does it terminate?

It may terminate—

(1) In slow but complete recovery, or

(2) In stricture of the œsophagus.

What is the prognosis?

It is usually favorable but tedious. It is unfavorable if there is much tissue destroyed.

What is its treatment?

Very little food should be given by the mouth, and that little should be mild and emulsive in character, as milk or gum-water; solid food should be strictly interdicted. The patient should be principally nourished by nutritive enemata. Minute doses of calomel and sodium carbonate should be placed on the tongue, and bismuth subnitrate may be blown down the throat; small pieces of ice in the mouth and a bladder of ice applied intermittently between the scapulæ afford much relief.

To prevent stricture, an œsophageal bougie should be passed after the acute symptoms subside, at least every two weeks for a year.

Stricture of the Œsophagus.

What are the varieties of stricture of the œsophagus?

(1) *Spasmodic stricture*, due to a spasm of the œsophageal muscles.

(2) *Organic stricture*, an inflammatory narrowing of the tube.

Describe spasmodic stricture.

(1) *Spasmodic stricture* is found most frequently in hysterical women or in hypochondriacal men.

SYMPTOMS.

There is difficulty in swallowing any kind of food, especially liquids. The spasm is frequently inconstant. If swallowing is attempted during the spasm, the face becomes livid and symptoms of

approaching suffocation supervene. Hysterical symptoms are frequently present.

DIAGNOSIS.

An œsophageal bougie is readily passed into the stomach, or is only arrested by the spasm, which gentle perseverance will overcome in a few days at most.

TREATMENT.

It is of the utmost importance to gain the confidence of your patient. Bromides and other nerve sedatives should be given in full doses, and bougies should be used every few days. The hysterical symptoms should be treated by good hygiene and food, and medicinally by zinc and tonics.

What is organic stricture of the œsophagus?

(2) The symptoms of *organic stricture* usually follow an acute inflammation, or œsophageal cancer. They come on gradually with difficulty in swallowing, especially of solid matters, and symptoms of spasm when deglutition is attempted. As the constriction increases the symptoms gradually grow worse, until finally there is difficulty in swallowing even fluids.

DIAGNOSIS.

An ordinary bougie cannot be passed, and a small one may only be passed with difficulty.

DIFFERENTIAL DIAGNOSIS.

(1) *From spasmodic stricture*, by the history, the gradual increase of symptoms, the greater difficulty in swallowing solids, and the passage of the bougie.

(2) *From cancer of the œsophagus;* in this affection there is severe pain, hemorrhages, cachexia, and the stricture grows in spite of treatment until the patient starves to death.

(3) *From aneurism pressing on the œsophagus;* always examine the chest for signs of aneurism before passing the bougie.

(4) *From diseases of the epiglottis;* an examination of the epiglottis will reveal the disease.

PROGNOSIS.

If the stricture follows inflammation, the prognosis is favorable; if, however, it is due to cancer, it will terminate fatally in spite of treatment.

TREATMENT.

When the stricture follows inflammation it should be gradually dilated by bougies, increasing in size, and passed very gently and carefully. If it is due to cancer, bougies do no good, and may do much harm by causing a rupture of the œsophagus.

Should the difficulty arise from an aneurism pressing on the œsophagus, bougies must not be used, as there is danger of rupturing the aneurism. The patient should be treated in these cases by rest, and the cardiac action regulated by aconite or veratrum viride, while iodide of potassium is administered in as large doses as can be borne.

(3) DISEASES OF THE STOMACH.

Acute Gastritis.

What is acute gastritis?

It is a violent acute inflammation of the stomach, generally due to an irritant poison.

What are its causes, symptoms and termination?

It is most frequently *caused* by the ingestion of mineral, animal or vegetable poison; but may arise independent of poisoning, from extension of inflammation from adjacent viscera or from traumatism.

SYMPTOMS.

Severe burning pain in the epigastrium increased by breathing, pressure or taking food; the pain is constant, but is liable to exacerbations.

There is *great thirst*, incessant nausea, *vomiting* often of mucus, fœtor of the breath, at first constipation, later severe diarrhœa, *moderate fever* and a *small, tense pulse.*

TERMINATION.

The inflammation may either cause collapse with clammy sweats, or subside in a few days leaving an irritable stomach.

In case of death, what is found on post-mortem examination?

There is intense congestion and inflammation of the *mucous membrane*, which is softened and coated with a strongly alkaline mucus; localized spots of intense redness are found and ecchymoses are fre-

quent. *The microscope* shows an interstitial infiltration of embryonal cells into the muscular coat and between the tubules, which are undergoing degeneration, and often atrophied from pressure.

What is the diagnosis, prognosis and treatment of this affection?

The disease is recognized by the history, the pain, vomiting, thirst, fever, pulse, and condition of the bowels.

PROGNOSIS.

The prognosis of acute gastritis, independent of the poisoning which may cause it, is usually favorable. It will vary with the intensity of the inflammation and the amount of tissue destroyed.

TREATMENT.

If it is due to poisoning, and the case is seen in the earlier stages, antidotes should be administered, the contents of the stomach evacuated, and, as a rule, the organ thoroughly washed out.

For the pain, the patient should be kept under the influence of morphine hypodermically administered.

The patient should be *nourished by the rectum*, and the stomach kept perfectly at rest; small pieces of ice may be allowed to dissolve in the mouth, or small quantities of iced milk or eggs in a little water administered for their demulcent effect.

For the gastric irritability hydrocyanic acid may be given, or calomel, gr. $\frac{1}{12}$, with sodium bicarbonate, gr. j, dusted on the tongue.

After the acute inflammation has subsided, bismuth, with soda, may be administered internally.

Acute Gastric Catarrh.

What is acute gastric catarrh?

It is a catarrhal inflammation limited to the mucous membrane of the stomach, attended with swelling, and at first a diminished, afterwards increased secretion.

What is its synonym?

It is frequently spoken of as a bilious attack.

What is its cause?

It usually follows errors in diet; it may depend on atmospheric influences, and is sometimes almost epidemic.

What are its symptoms, diagnosis and treatment?

SYMPTOMS.

There is loathing of food, a heavily-coated tongue, offensive breath, vitiated secretions, and a bad taste in the mouth, nausea, bilious vomiting, constipation, the stools being black and offensive, slight epigastric pain, little fever, intense thirst, sometimes vertigo in the early morning when rising, and occasionally headache, disturbed vision and irregular pulse.

DIAGNOSIS.

The disease is recognized by the history, the slight pain and fever, the nausea, vomiting, and coated tongue, and the various nervous disturbances.

PROGNOSIS.

Is favorable. If the cause continues, the attacks are apt to be repeated, and finally to end in permanent gastric catarrh.

TREATMENT.

A brisk purgative, as podophyllin, or calomel, or blue mass, followed by a saline, should be given, and the diet should be regulated.

For the irritability of the stomach, bismuth and soda, or carbolic acid, gtt. j, every hour or two in a little mint-water, may be given.

Chronic Gastritis.

What is chronic gastritis?

It is a chronic catarrhal inflammation of the mucous membrane.

What are its synonym and causes?

SYNONYM.

Chronic gastric catarrh.

CAUSES.

It is usually found after middle life. It frequently occurs from continual moderate drinking, or from repeated attacks of acute gas-

tric catarrh. It is also seen in chronic heart disease following prolonged venous congestion.

What are its symptoms, duration and termination?

SYMPTOMS.

One of the most characteristic symptoms is *early morning vomiting*, the vomited matters consisting principally of glairy mucus. There is delayed digestion, flatulency and acid eructations, due to the fermentation of food; the patient is sleepy after meals, is constipated, has a coated tongue, suffers from great thirst and anorexia, *has very little pain*, but *slight epigastric tenderness*. Various nervous symptoms, as vertigo, headache, restlessness, disturbed sleep and irregular action of the heart, are apt to be present.

DURATION.

It is a very chronic disease, finally becoming incurable.

TERMINATION.

It may end in gastric ulcer, or in pyloric thickening (rare). Generally the patient dies worn out by his bad habits, as drink, or by the disease of the heart, which has caused it.

What is found on post-mortem examination?

There is a persistent swelling of the mucous membrane of the stomach, occurring in patches, which are usually discolored, being grayish or ashen in appearance. The mucous and sub-mucous coats are thickened, the tubules are degenerated and atrophied, the mucous follicles hypertrophied, and the surface of the mucous membrane coated by a thick, offensive mucus.

What is the diagnosis and treatment of this disease?

DIAGNOSIS.

The history of the case, the morning vomiting and the tenderness with the absence of pain, of fever, and of signs of a tumor, should render the diagnosis clear.

TREATMENT.

It is absolutely necessary for the patient to break off all bad habits which act as causative agents. He should have a bland and easily-digested diet, rather scanty in quantity, mostly of animal food. Pepsin should be administered with meals. The salts of silver, with

opium or belladonna or bismuth, are sometimes useful. Mildly purgative mineral waters or salines should be administered, to act on the portal circulation. The vegetable tonics and mineral acids should only be used temporarily when large amounts of alkaline mucus collect in the stomach, as their continued use favors the formation of this secretion.

Washing out the stomach with a stomach-pump affords temporary relief.

Gastric Ulcer.

What are the causes of gastric ulcer?

Gastric ulcer is most common in women about twenty years of age. The most frequent cause is malnutrition of the stomach, due to altered states of the blood, as anæmia. It also occurs from chronic congestion, such as is seen in chronic gastritis, or in diseases of the right side of the heart. It may follow diseases of the left side of the heart, where the branches of the gastric artery become plugged with emboli.

What are its symptoms?

Anorexia and general dyspeptic symptoms may or may not be present. Symptoms due to the disease which causes the ulcer are usually complained of.

The Special Symptoms are *pain*, which is fixed and does not radiate; it is usually under the ensiform cartilage and between the scapulæ, is increased by pressure, is increased immediately on taking food, but often occurs in paroxysms independent of food.

Vomiting occurs soon after the taking of food, due to the irritation produced by the food on the ulcer; the vomited matters often contain blood-streaked mucus.

Hæmatemesis occurs in about half the cases. The blood is generally black, unless the ulcer is very large. The amount of blood varies.

What is the pathology of gastric ulcer?

The ulcer is developed in the same manner as ulcers in other parts of the body. It may be single or multiple, circular or oval; it may perforate the stomach and penetrate into the peritoneum,

or adhesions may form in the peritoneum between the stomach and adjacent organs, and the ulcer open into a neighboring viscus without opening the peritoneal sac. It may perforate the stomach down to the peritoneal coat and then cicatrize, or its progress may be arrested at any stage. It is generally found on the posterior wall or at the pyloric end.

How is ulcer of the stomach recognized?

The DIAGNOSIS is made from the age, the sex, anæmia or other causes, the localized pains, especially on taking food, the vomiting, the hæmatemesis and the absence of signs of tumor.

What is the prognosis?

The prognosis is generally favorable under proper treatment and under good conditions. It will depend to some extent upon the cause.

How should ulcer of the stomach be treated?

The stomach must be given as much rest as possible. Very little food should be given by the mouth, and that little should consist of milk and lime water, or, as cicatrization commences, finely chopped meat with pepsin. The patient should be nourished by nutritive enemata and should be confined absolutely to bed.

Arsenic in small doses or the nitrate or oxide of silver with opium may be administered.

To allay the pain morphine may be used hypodermically.

If anæmia is present, iron may be given in small doses when the stomach will bear it.

If the ulcer tends to perforate, keep the patient under the influence of opium.

Gastric Cancer.

What are the causes of gastric cancer?

It is generally hereditary, and is the most common seat of primary cancer in the male, and, next to the uterus, the most frequent seat in the female. It usually occurs after middle age.

What varieties of cancer are found in the stomach?

Any variety of cancer may be present, but the scirrhus and the encephaloid are the forms most frequently met with.

What part of the stomach is usually affected?

It is most common at the pyloric end, next in frequency the cardiac end is affected. It is sometimes found in the anterior, but is rare on the posterior wall. It begins in the submucous tissue, grows into the cavity of the stomach and ulcerates. Occasionally it is found as an interstitial cancer between the coats of the stomach.

What are its symptoms?

General Dyspeptic Symptoms are frequently, not always, present. There is usually great flatulence, acid eructations and acid vomiting from fermentation of the food; there is much fœtor of the breath and, as the disease progresses, emaciation and cachexia. Hydrochloric acid is said to be absent from the gastric juice. The temperature is always subnormal in cancer.

Special Symptoms.

(1) *Pain.*—Severe in character, independent of eating, often radiating or shooting from the epigastrium, the patient rarely being free from it. It is more severe in the encephaloid and other rapidly growing cancers than in scirrhus.

(2) *Vomiting*, which occurs immediately after eating and is more like a regurgitation of the food, if the cancer affects the cardiac end of the stomach. When the pylorus is affected the vomiting occurs several hours after eating, is acid in character. and often contains sarcina ventriculi.

(3) *Hæmatemesis* occurs in half the cases.

(4) *A tumor* is present in 80 per cent. of cases, is more marked as emaciation proceeds and is found in the epigastrium and right hypochondrium as a hard, resisting mass. As it grows, it may finally cause dropsy from pressure.

(5) Hydrochloric acid is absent from the gastric juice.

What is the duration and termination of cancer?

The duration is from one to five years; it usually *terminates* by death from starvation.

What are the complications?

The *pancreas*, *gall bladder*, or *liver* may become secondarily involved.

How is cancer of the stomach diagnosed?

This disease is recognized by the age of the patient, the gastric

symptoms, the pain, vomiting, hæmatemesis, absence of hydrochloric acid, tumor, emaciation, subnormal temperature, and cachexia.

DIFFERENTIAL DIAGNOSIS.

(1) *From chronic gastritis with hemorrhage due to congestion:* the paroxysms of pain, the emaciation, cachexia, tumor, subnormal temperature, absence of hydrochloric acid, and progress of the disease in spite of treatment, distinguish cancer from the latter disease.

(2) *From gastric ulcer*, by the age, the radiating pain, which is not influenced by food, the tumor, cachexia, subnormal temperature, absence of hydrochloric acid, and absence of localized tenderness.

What is the prognosis?

Death occurs sooner when the cardiac end of the stomach is involved.

What is the treatment of gastric cancer?

The patient's diet must be regulated and should consist of easily digested meat with pepsin, or of milk.

Corrosive sublimate in small doses long continued, or Fowler's solution, are supposed to retard the development of the cancer.

For the fermentation, eructations and *fœtor of the breath*, carbolic acid (gr. $\frac{1}{4}$) may be given in glycerine or mint-water.

For the pain, morphine may be administered hypodermically or by the rectum.

For the excessive acidity, aromatic spirits of ammonia, solution of potassium, or the citrate of lithium do good.

For constipation, enemata of castor oil give the best result. Sulphur, compound liquorice powder, or aloin may be used.

When the eructations are excessive charcoal is sometimes of value.

The stomach should be washed out occasionally to get rid of the mucus which is apt to accumulate.

Hæmatemesis.

What is hæmatemesis?

Hæmatemesis or hemorrhage from the stomach, is not a disease, but a symptom, and may be due to disorders of various organs.

What are its causes?

(1) It is often a symptom of *organic disease of the stomach*, as ulcer or cancer.

(2) Anything which causes *gastric congestion* from obstruction of the portal circulation may produce hemorrhage from the stomach; thus, cirrhosis of the liver, splenic enlargement, tumors pressing on neighboring vessels, or various cardiac diseases may be factors in its production.

(3) *Diseases of the blood*, as anæmia or scurvy, may cause a hemorrhage,

(4) Or it may be *vicarious*, resulting from the arrest of the menstrual discharge or the bleeding from piles.

What are the symptoms, irrespective of cause?

There is a *discharge of blood by the mouth, bowels, or both.* If the vessels give way suddenly, and a large amount of blood is poured out, it will be red in color. Usually, however, the bleeding into the stomach takes place gradually, and when vomited the blood is black, clotted, and like coffee-grounds, being altered by the acid secretions.

Nausea precedes the hæmatemesis, and is one of the earliest symptoms.

Faintness.—There is epigastric weight and oppression; cold, clammy skin, and symptoms due to shock, the duration of which vary and are generally relieved by the vomiting.

There is *vomiting of food mixed with blood.*

Black, chocolate-colored stools are passed.

How is hemorrhage from the stomach diagnosed?

Blood coming from the stomach is recognized by the history of the case, by the fact that the blood is generally black, that it is vomited, that it is usually mixed with food, and by the dark, tarry stools.

Differential Diagnosis.

(1) *From Hæmoptysis.*—In this case the blood is light in color, frothy, and mixed with bronchial mucus. It is usually coughed up.

(2) *In a child blood may be vomited* which has been previously swallowed, having come originally *from the lungs or from the nose.*

What is the prognosis of hæmatemesis?

Very few persons die during the hemorrhage, except when it proceeds from the ulceration of a large vessel or from scurvy. The prognosis is unfavorable in proportion to the likelihood of return.

What is its treatment?

(1) *To check the bleeding*, cold water, pellets of ice, or a little brandy may be administered. If the stomach is not irritable, tannic or gallic acid (gr. xx every half hour) or Monsel's solution (gtt. ij–v) freely diluted, or sulphuric acid in the same doses, are beneficial. Ergot is used, but is not so good.

Absolute rest in a recumbent position is important. To aid in procuring this, as well as to tranquilize the nervous system, morphine may be administered hypodermically.

If the bleeding continues ergot should be administered hypodermically in addition to the medicines above enumerated.

The after-treatment should consist in treating the cause, opening the bowels, and regulating the diet.

Dilated Stomach.

What are the causes of dilatation of the stomach?

It may be due to cancer or other *organic* obstructions to the pylorus, or to *functional* causes, as gastric weakness, atony of the muscular fibres, etc. It is not a rare disease.

What are its symptoms?

The patient is probably a large eater, and is not in very robust health. He suffers from general dyspeptic symptoms. The epigastric region is exceedingly tympanitic except at the lower part, where there will be dullness over the accumulated food. There are occasional fits of vomiting, the vomited matters being enormous in quantity, often containing sarcina ventriculi, and consisting of partly digested food which probably has been accumulating in the stomach for several days or a week. The vomited matters will be more or less digested, according as the gastric tubules are atrophied or degenerated.

What is the prognosis?

If treated early the prognosis is favorable. After a time, however, the gastric tubules become atrophied and the patient will finally die of exhaustion.

What is the treatment?

The patient should live on small quantities of dry solid food with very little liquid. Strychnine should be administered by the mouth or hypodermically, and carbolic acid should be given for the fermentation. The offensive eructations are best treated by the administration of charcoal with soda and bismuth.

Gastralgia.

What is gastralgia?

Gastralgia, or gastric pain, is generally a symptom of organic disease of the stomach. When it recurs in young persons it is usually caused by ulcer, when idiopathic it is of nervous origin, it being either a neuralgia of the stomach or occurring from spasm of the muscular coat.

FUNCTIONAL OR NEURALGIC GASTRALGIA.

Describe functional gastralgia.

It is usually seen in hysterical women or over-worked, overburdened men. It occurs also as a symptom of lithæmia.

What are the symptoms?

(1) *Digestive disturbances* may be present or absent; generally there is a want of digestive tone, a feeling of fullness, and flatulency after meals.

(2) *Intense pain in the epigastrium*, lasting from a half to two hours, relieved by pressure, bearing no relation to meals unless organic disease is present. Eating may ward off a threatening attack.

(3) *The stomach feels hard* to the touch during the attack.

(4) *Sympathetic disturbances* are often present, as feeble, intermittent pulse, cough, vertigo, anxiety, cold extremities, local sweats, etc.

How is this recognized?

It is recognized by the history, the digestive and sympathetic disturbances, the intense intermittent pain, relieved by pressure, and not aggravated by taking food, the absence of hemorrhage and of tumor.

DIFFERENTIAL DIAGNOSIS.

From Intercostal Neuralgia.—Neuralgia is usually limited to the left side, and spots of localized tenderness are found over the nerve.

What is the prognosis?

It is an intractable but not a dangerous disease.

How should it be treated?

The diet should consist of easily digested food, animal food and a little stimulant generally being best.

For the pain, morphine should only be used when absolutely necessary, as from the intractable nature of the disease there is great danger of the formation of the opium habit. Paregoric in teaspoonful doses often gives immediate relief.

Bismuth with minute doses of morphine and aromatic powder is frequently of service.

Nitro-muriatic acid in small doses gradually increased and freely diluted, sometimes does good. Carbolic acid is of use, especially if there is gastric irritability, combined with minute doses of morphine. Fowler's solution in small doses given for a long time produces satisfactory results.

Dyspepsia.

What are the causes of functional dyspepsia?

Too rapid eating; too much water at meals, especially ice water; improper food; want of exercise; too much tea or coffee; and the abuse of tobacco, are the principal causes of this disturbance.

What are the symptoms?

The patient suffers from general dyspeptic symptoms, as listlessness, languor, headache, extreme vertigo, disturbed sleep, jerking of the muscles during sleep, irritable temper and a frequent, irregular heart, with disturbed action of the liver if the disease continues for

any length of time. The urine is high-colored, contains urates and phosphates, the bowels are constipated, the tongue is usually flabby, broad, and indented by the teeth, and not much, if at all, coated. The patient is gloomy and imagines he has some organic trouble.

What are the principal varieties of dyspepsia?

(1) Atonic or Nervous Dyspepsia.

In these cases there is anorexia, and a sensation of weight after meals is usually experienced. During digestion the patient is heavy, stupid and often falls asleep. There is irritability of temper, dread of organic disease, and other nervous disturbance.

(2) Acid Dyspepsia or Heartburn.

The patient suffers from weight, listlessness, pains after meals and extreme acidity. This form is seen in people who "bolt" their food without properly masticating it. The stomach becomes overtaxed, and fermentation occurs from the improper secretion of gastric juice.

(3) Flatulent Dyspepsia.

In these cases there is delayed digestive operations, and perhaps acidity from fermentation of the food.

There is offensive breath, distention of the abdomen after meals, and often eructations of gas with much noise.

(4) Pyrosis or Water-brash.

In these cases vomiting, which is not common in functional dyspepsia, usually occurs. The vomited matters consist of thin, glairy, alkaline fluid from the lower part of the œsophagus or stomach. Flatulency and other dyspeptic symptoms are present.

(5) Hysterical Dyspepsia.

These cases occur in hysterical patients, especially in women, and are characterized by vomiting after meals, enough food, however, remaining in the stomach to nourish the patient, who does not lose weight.

What is the diagnosis of functional dyspepsia?

It is recognized by the delayed digestion, the clean tongue, no tenderness on pressure on the epigastrium; by the pyrosis and other dyspeptic symptoms.

From chronic gastritis it is differentiated by the fact that there is

no diffused pain, no tenderness, but a clean, flabby tongue, not coated behind with red tips and edges ; that there is no similarity in causation, no excessive thirst, not much, if any, vomiting, but great prominence of nervous and heart symptoms, and vasomotor disturbance, with perhaps some fever.

What is the prognosis of dyspepsia?

The prognosis is favorable.

Nervous dyspepsia with oxalates in the urine, or dyspepsias characterized by vomiting are more obstinate than the other forms.

In what does the treatment consist?

(1) *Avoid the cause* if it can be discovered, and prevent any nervous strain, especially at meal times.

(2) *Regulate the diet*, giving the patient whatever agrees with him best. Usually a meat diet is most suitable; potatoes, starches and bread are not good; peas, spinach, celery, onions are better. Fish and oysters suit some. Sweetmeats, pastry, etc., must be interdicted. Milk is not as good in functional as in organic dyspepsia.

(3) *Properly regulated exercise* is always of use.

(4) *Keep the bowels open* by small doses of aloes.

(5) If the patient be a man let him *smoke very little.*

(6) *Give small doses of pepsin* after meals and mineral acids with meals combined with bitters, as nux vomica, gentian, cascarilla or colomba.

(4) DISEASES OF THE INTESTINAL TRACT.

Colic.

What is colic?

It is a twisting, griping pain around the umbilicus, occurring in paroxysms without fever. It is often due to flatulence, sometimes to nervous depression or errors in diet, to constipation or to poisoning by certain metals. It is of short duration, passes gradually away, leaving some soreness behind, but is apt to recur as long as the cause remains.

What are the symptoms of colic?

The patient writhes with pain, which is relieved by pressure, the

extremities are cold; there are often clammy sweats, great anxiety and sometimes vomiting, which, however, is rare.

VARIETIES.

(1) *Colic from Constipation.*—There is usually a history of habitual constipation and the general symptoms of colic.

The disease continues until the cause is removed.

(2) *In colic from mechanical irritation*, such as unripe fruit, the general symptoms of colic are present with irritative fever, and perhaps vomiting.

(3) METALLIC COLIC.

Certain metals, as lead, copper, corrosive sublimate, etc., also give rise to colic.

(*a*) *Copper colic* causes violent recurrent paroxysms of pain, more or less present all the time, persistent nausea, tenderness and vomiting, secondary muco-enteritis with fever. The history of exposure to copper, especially among persons working in copper filings, gives the key to a diagnosis.

(*b*) *Lead colic* is very common; it is particularly seen in painters who do indoor work and who use white paints with much turpentine. Some persons are exceedingly susceptible to poisoning by lead. The use of snuff wrapped in tin-foil, or water contained in lead pipes, may also produce colic.

The symptoms are those of colic generally, constipation, no fever, soreness of the abdomen, legs and arms, and if much lead has entered the system, a blue line will be found on the gums.

It is a chronic, persisting disease, the patient rarely being free from pain until the lead has been removed from the system. It is often associated with other symptoms of poisoning by lead, as violent headache, wrist drop (in which the muscles do not react to electricity), sometimes convulsions, irregular heart, which is apt to be hypertrophied, and sometimes granular, contracted kidney.

What is the prognosis?

The prognosis generally depends upon the cause. It generally passes away quickly when the cause is removed.

The metallic colics are obstinate.

How is colic treated?

For the immediate relief of pain, warmth externally, and inter-

nally by means of carminatives, such as mint, ginger, fennel seed, chlorodyne or hot water, is of service.

If the pain is very severe, an opiate may be administered, and mustard plasters applied to the surface of the abdomen.

To prevent the recurrence of the pain, small doses of nux vomica with regulation of diet, or small doses of alcohol, not continued for too long a time, are of service.

In any case endeavor to ascertain the cause and remove it.

If constipation exists, treat it by castor oil and other laxatives.

If there is mechanical irritation caused by improper foods, laxatives may be administered, and later, astringents, as bismuth and opium, to counteract the resulting diarrhœa.

In metallic colics the same treatment is indicated, and care must be taken to remove the metal from the system.

In colic due to copper the violent muco-enteritis must be treated by bismuth and opium, regulation of the diet and small doses of mercurials.

In colic due to lead the obstinate constipation should be treated by castor oil in large doses, and with warm water injections. Croton oil (gtt. $\frac{1}{8}$, *t. i. d.*) may also be used to aid in the elimination; sulphuric acid and strychnine sulphate, with potassium iodide, is the best treatment.

Belladonna or atropine is said to relieve lead colic very quickly. Persons who are exposed to lead poisoning should be scrupulous in their personal cleanliness, particularly of the fingers and nails, as lead is often conveyed to the mouth with the food. They should also drink diluted sulphuric acid in the form of lemonade, as this acts as an antidote to the lead.

Constipation.

What are the causes of constipation?

It is often a symptom of dyspepsia, is frequently due to atony of the bowels, especially of the colon, is seen in persons who lead a sedentary life, particularly in women, and is sometimes due to defective secretion of mucus.

What are the symptoms?

There is an irregularity of the bowels. Some persons naturally only have one passage in several days. If this has always been their habit, and there is no bad symptoms, and it does not interfere with the health, do not do anything. Besides irregularity in the passages from the bowels there is generally headache, vertigo, gastric disturbance, disordered sleep, mental hebetude and feeling of weight in the abdomen.

How should constipation be treated?

Exercise is of importance. Massage of the abdomen sometimes stimulates peristalsis. The great thing in the treatment of constipation is to *form a habit* of having a passage every day at a certain time.

For temporary purposes enemata may be used. *The diet* of the patient is also of importance. He should take very little milk or meat, live as much as possible on fruit, vegetables, oat meal, and similar substances. *The less medicine they take the better.* Purgative mineral waters may be taken temporarily, but should not be used for any length of time, because they cause a drain of albumen from the blood. Aloes or aloin in small doses is frequently given. It may be combined with rhubarb, or colocynth, or with extract of belladonna and strychnine, when it is given to stimulate the organic muscular fibres of the bowels.

When constipation has existed for some time it may be necessary to give castor oil or small doses of croton oil or compound liquorice powder in order to assist the action of the bowels. None of these are good for persistent use.

Cholera Morbus.

What are the causes and symptoms of cholera morbus?

Cholera morbus is produced by changes in the weather or errors in the diet, particularly by eating unripe fruit.

SYMPTOMS.

It begins with *vomiting;* the vomited matters at first consisting of the contents of the stomach, then mucus and, later, bilious matters; *purging* often severe, the stools at first being fæcal, then bilious, and, later, serous in character; *cramps* in the stomach and extremi-

ties, probably due to the loss of fluid from the blood; cold, clammy surface; feeble pulse; great anxiety and recovery or collapse. If collapse occurs, the symptoms resemble those of Asiatic cholera.

What is the pathology and diagnosis of this affection?

There is over-secretion of the mucous glands, resulting in irritation of the mucous membrane.

DIAGNOSIS.

The disease is recognized by the history, the vomiting, the purging and the cramps.

DIFFERENTIAL DIAGNOSIS.

(1) *From Irritant Poisoning.*—In the latter case there is a history of some irritant having been taken; the sudden onset of the symptoms soon after eating; the great tenderness; the appearance and chemical examination of the vomited matter and the stools, and, perhaps, symptoms referable to the poison which has been swallowed.

(2) *From Asiatic Cholera,*—The latter begins abruptly; there are usually serous discharges almost from the first; the collapse is extreme; the surface and breath are icy; it is epidemic; there is no history of cause, and the cholera bacillus is always present in the discharges.

How should cholera morbus be treated?

If seen early, a brisk emetic and cathartic should be given.

For the relief of pain, morphine (gr. $\frac{1}{6}$) with atropine (gr. $\frac{1}{80}$) may be administered hypodermically, but should not be repeated in less than one hour.

For the vomiting, carbolic acid (gtt. ss–j) perhaps combined with morphine (gr. $\frac{1}{48}$), may be given every fifteen or twenty minutes. Effervescing waters are also grateful, and, locally, the application of mustard is of service.

For the irritability of the stomach which follows, bismuth is an excellent remedy.

If the attack is unyielding, sprinkle on the tongue calomel (gr. $\frac{1}{8}$), with sodium bicarbonate (gr. j), every half-hour until a change occurs in the symptoms, after which the dose should be decreased.

For the cramps, ginger, capsicum or applications of chloral, dissolved in soap liniment, or even chloral given hypodermically, may be employed.

When the stomach becomes less irritable, lime water and small amounts of broth may be given, but the diet should be restricted for several days.

Acute Diarrhœa.

What are the causes of acute diarrhœa?

It may be caused by sudden changes in the weather, by errors in the diet, by various reflex nervous disturbances as teething in children. It is also seen in those who eat arsenic for their complexion and in chronic cases of the opium habit.

What are the symptoms, duration and termination?

The patient experiences griping pains in the abdomen, tenderness with frequent stools, which vary in character and constitution; the tongue is coated, and fever often occurs. It lasts from a few hours to a few days and passes off gradually, leaving the patient weak and often constipated.

What is its pathology?

There is generally increased peristalsis of the muscular layer of the bowel or increased intestinal secretion, sometimes both. An increased secretion of bile or increased epithelium proliferation are sometimes seen with or without the first two conditions.

What is the prognosis?

The prognosis of acute diarrhœa is generally favorable. Sometimes it passes into a chronic condition.

How should it be treated?

If it is due to an irritant and is seen early, a light laxative, as castor oil, should be administered. *If it is not seen in the early stage*, some form of opium combined with other remedies, according to the indications, should be used.

(1) *When the passages are loose, watery and without much color or smell*, the acids, as Hope's Camphor Mixture, hydrochloric, nitric or sulphuric acid, generally give the best result.

(2) *When the stools are loose and offensive and contain much mucus*, bismuth, with opium or Dover's powder, if the stomach is not irritable, is indicated.

(3) *If the stools are more than moderately loose, with mucus, griping and abdominal tenderness (especially in children)*, the antacids, as chalk mixture with opium, should be given.

(4) *When there are bilious discharges which are yellow or greenish, with weight in the abdomen and coated tongue and sometimes vomiting*, the alkalies, as the preparation of sodium or ammonium, or minute doses of calomel frequently repeated, are serviceable.

(5) *When the stools are light in color and frothy, having a fermented appearance*, the diarrhœa is best controlled by an intestinal antiseptic, as salol, combined with bismuth and chalk.

In any case the diet should be plain, consisting of milk, soft boiled eggs, and as little fluid as possible. Corn starch sometimes does good. When there is not much mucus or catarrhal difficulty arrow-root answers well. The patient should not have too much to eat.

Chronic Diarrhœa.

Describe chronic diarrhœa.

Chronic diarrhœa differs from chronic dysentery only in affecting the small not the large intestine at first. After a time the disease extends to the large intestine also, and the affections are so merged as to be identical.

SYMPTOMS.

Those of chronic dysentery, with the exception that the stools contain little or no mucus, no blood, and there is no straining until the large intestine is involved.

THE TREATMENT is the same as in chronic dysentery. (*q. v.*) Alcohol, especially port wine, is of service.

Duodenitis.

What is duodenitis?

It is the catarrhal inflammation of the duodenum following cold, errors in diet, but more especially due to malaria.

What are the symptoms?

There is pain, which is often severe, and comes on a few hours after eating, especially after starchy food is taken, when it lasts for some time, and is accompanied by flatulence. There is marked tenderness in the upper part of the abdomen, great discomfort, often

slight fever, despondency, reflex symptoms pointing to sympathetic disturbance of other organs, as gastralgia or irregularity, often intermittence of the cardiac beat, utter loathing of food, constipation, coated tongue, and frequently jaundice.

What is the duration and prognosis?

The disease lasts several weeks and slowly susbides.

The prognosis is generally favorable, if there is no duodenal ulcer. If an ulcer forms, it is usually due to an embolus. The symptoms denoting it are, an increase of all the symptoms, particularly of the pain and tenderness, which become almost intolerable. Bloody stools are frequently passed.

What is the treatment?

DIETETIC TREATMENT.

Strict attention should be paid to the diet, which should consist principally of skimmed milk, with small amounts of animal foods, or broth combined with pepsin. Occasionally he may be allowed an egg. Game, oysters and fish may be given, but no starchy vegetables, as potatoes, and no bread should be taken. A little celery or spinach will do no harm.

THE MEDICAL TREATMENT.

The alkalies should be given, to deplete the mucous membrane, unload the portal circulation and act as a sedative to the inflamed structures. For this purpose sodium phosphate or bicarbonate, magnesium sulphate, cream of tartar, Rochelle salts, or the alkaline mineral waters are the best.

For the pain belladonna may be used locally and internally. Cannabis indica or hyoscyamus may be given. Opium should be withheld if possible.

If the disease persists bismuth subnitrate should be given in gr. x doses. *If this treatment fails*, Fowler's solution (gtt. j) with deodorized tincture of opium (gtt. ij), every hour or two, will sometimes prove serviceable.

If the bowels become irritable late in the disease, check the passages, as in diarrhœa.

If duodenal ulcers form, or if the disease becomes chronic, silver nitrate or oxide is highly recommended.

Catarrhal Enteritis.

What is catarrhal enteritis?

Catarrhal enteritis, *ilitis* or *muco-enteritis* is a catarrhal inflammation affecting the mucous membrane of the ileum.

What is its cause?

It is sometimes epidemic, especially during the prevalence of influenza; it is also produced by cold and exposure, or by the other causes which favor the development of catarrhal processes in general.

What are its symptoms?

The symptoms are those of acute diarrhœa, which is often nothing else but this disease. There is moderate fever, griping pains around the umbilicus, thirst, loose passages and anorexia. It lasts for a few days and gradually passes away, leaving some abdominal tenderness behind it.

How is it treated?

The patient's diet must be restricted. He should live principally on broths, rice, arrow-root, etc. He should not drink much water, and what he takes had better contain a little lime-water mixed with it. Claret and apollinaris water form an agreeable substitute for ordinary water, and, besides, the claret is somewhat of an astringent. He must be kept as quiet as possible and have a moderate amount of opium in the form of paregoric, deodorized tincture or suppository. If this does not check the trouble, give him bismuth or lead acetate, with opium and small doses of ipecac.

Membranous or Croupous Enteritis.

What is membranous enteritis?

It is a form of enteritis in which a cast or mould of the small intestine is formed, consisting of mucus, epithelium, leucocytes, etc. It cannot be said to be either a rare or a common affection.

What is known of the cause of this affection?

It is found especially among women, particularly those of a hys-

terical temperament, and is often associated with membranous diarrhœa. It is a very protracted disease.

What are its symptoms?

(1) INTESTINAL SYMPTOMS.

(*a*) *Between the attacks*, which recur every few months, the patient is usually pretty well, but suffers from constipation, and has a flabby, coated tongue.

(*b*) *During the attacks* there are violent colicky pains and abdominal tenderness; fever, with a quick, irritable pulse, and constipation. These symptoms last for four or five days, after which the membrane is formed and comes away in shreds or masses, and the symptoms abate.

(2) GENERAL SYMPTOMS.

All kinds of curious hysterical symptoms may be seen during the attack, which usually pass away promptly when the membrane is expelled; thus temporary aphasia is sometimes found, or symptoms simulating tetanus.

How is this disease recognized?

The diagnosis is made by the symptoms of enteritis and the passage of the membrane, which is soft and easily breaks.

The membrane is distinguished from tapeworm by breaking easily, and by a microscopic examination.

What is the prognosis?

The patient always recovers from the attack, but it is a chronic disease, and the attacks are constantly recurring until the patient is worn out by them.

What is the treatment of this disease?

The *diet* of the patient need not be restricted.

Purgatives, as small doses of salines, compound liquorice powder, senna or sulphur, should be administered as necessity demands.

Tar persistently used, in the form of the wine or fluid extract, is occasionally beneficial. Arsenic in small doses, continued for a long time, has sometimes proved of service. Change of climate and scene, as traveling, or visiting the mountains or sea-shore, does some good. A sea-voyage is also useful.

When the membrane is being passed, a laxative is indicated, and

for this purpose castor oil, sweet oil, or magnesia answer better than any others.

For the pain, opium may have to be administered, but should be deferred as long as possible, and cannabis indica, hyoscyamus and other remedies of that class tried first, although, in the long run, opium has usually to be resorted to, and the patient generally becomes a confirmed opium eater. Poultices, or fomentations with turpentine and laudanum or with infusion of poppies, should also be used externally when the pain is great.

If membranous dysmenorrhœa also is present, the womb should be treated, in the hope that if one can be cured, the other affection may pass away at the same time.

Enteritis.

What is enteritis?

Enteritis is an inflammation attacking not only the mucous membrane, but all the other coats of the bowel as well, and characterized by marked fever, griping pains, local tenderness, constipation followed by diarrhœa, and throbbing of the abdominal vessels.

What is its cause?

It is generally caused either by exposure or by mechanical irritation, as from eating indigestible food.

What are its symptoms?

There are violent cramps in the abdomen, with intense pain in the neighborhood of the umbilicus; nausea, vomiting, high fever, a quick, irritable, tense pulse. The bowels are at first constipated, but soon become loose, the stools being watery, but containing very little fæcal matter, and being attended with great pain. There is, in fact, a localized peritonitis. The abdominal vessels throb so markedly that the patient generally complains of it very much, and it can be readily felt and usually seen by the physician.

How is this affection recognized?

It is known by the fever, the marked local tenderness, the griping pains, the throbbing of the abdominal vessels, and the constipation followed by diarrhœa.

What is the prognosis?

The prognosis is not as favorable as that of catarrhal enteritis, but the majority of patients recover under proper treatment.

What is the treatment?

The *diet* should be light and of the mildest possible character.

Internally, *opium* must be given in large doses (gtt. x of the deodorized tincture every hour, the dose being increased if the medicine is well borne). *Mercurials* may be given in small doses, *and later* a laxative, as castor oil, or small doses of salines may be prescribed. Locally. *if seen early*, leeches should be applied to the abdomen. If not seen until later, or if the patient is too weak to leech, the application of hot cloths, sprinkled with laudanum or wrung out of infusion of poppies, answers best. Poultices may be applied if their weight is not objectionable.

Colitis.

What is colitis?

It is a catarrhal inflammation of the large intestine, characterized by *symptoms* similar to those which are seen in muco-enteritis, except that the pain is limited to the locality of the colon.

The treatment is the same as that for the latter affection.

Acute Dysentery.

What is acute dysentery?

It is an inflammation of the descending portion of the large intestine, generally catarrhal in character, but which may become diphtheritic, ulcerative or even gangrenous. It is often epidemic, and is characterized by constipation, tenesmus, bloody, muco-purulent stools and constitutional disturbance.

What are its causes?

Dysentery is due to atmospheric changes ; to exposure to cold and damp ; to errors in diet (not a very common cause), and sometimes to epidemic influences.

Malarial dysentery is often caused by drinking water impregnated with the malarial poison ; or when the disease is epidemic, infection

may occur from the stools. The disease disappears under good drainage and improved hygienic conditions. When epidemics occur in armies, the disease usually is of the diphtheritic or of the gangrenous variety.

What is the pathology of dysentery?

(1) Ordinary Catarrhal Dysentery.

In this variety the mucous membrane is swollen, red and thickened. The proper secretion is at first arrested, afterwards increased and finally becomes muco-purulent. Exudation occurs into the submucous tissue. The engorgement of the small vessels is so great that they often rupture, causing either hemorrhage from, or ecchymosis under, the surface of the mucous membrane. The disease gradually subsides.

(2) Ulcerative, Croupous, or Diphtheritic Dysentery.

Ulcers form on the surface of the mucous membrane. They have a greenish, unhealthy base, and irregular, thickened borders. There is a deposit of a croupous or diphtheritic material (resembling a false membrane) between the coats of the bowel.

(3) Gangrenous Dysentery.

In this form, the above processes continue, until finally large portions of the bowel become gangrenous and are discharged in the stools as sloughs.

What are the symptoms, duration and termination?

(1) In Acute Catarrhal Dysentery there is *tenesmus* with a constant desire to go to stool; *the alvine dejections are purulent and bloody.* Constipation exists in reality, for although the sufferer is going so frequently to stool, yet there is little, if any, fæcal matter in the passages.

If, however, the disease begins above in the small intestine and gradually passes down into the larger bowel, diarrhœa may exist. There is reflex nausea and vomiting and reflex irritability of the bladder.

Marked fever is present while the acute symptoms last, the temperature frequently being as high as 103°, and having a morning remission and an evening exacerbation. The pulse is small, often full and compressible; there is thirst, restless nights, and some pain

and tenderness over the bowel. The disease lasts a week or ten days and gradually subsides, more fæcal matter passing by the stools, and with less straining and tenesmus. Or the disease continues, and finally passes into chronic dysentery.

(2) Ulcerative, Croupous or Diphtheritic Dysentery presents the same symptoms as the foregoing variety, but they are much more severe. There is almost continual tenesmus; large quantities of mucus and pus with shreds of membrane and of tissue are passed with the stools, which are black and offensive and are swarming with bacteria, which are said to be identical with those found in the diphtheritic membrane of the throat. The extremities are cold, the skin clammy, the internal temperature high, delirium is frequent and often marked. The disease usually runs a rapid course and often terminates fatally, from heart failure.

(3) Gangrenous Dysentery is seen particularly as an epidemic among troops. The symptoms are similar to the foregoing variety, except that large shreds of gangrenous tissue are passed from the bowels, and the disease runs a much more rapid course, the prostration being much more severe.

What are the sequelæ of dysentery?

(1) *Abscess of the liver* is frequently seen, in hot climates especially, and is due to emboli which have been washed from clots in the inferior hemorrhoidal vein into the portal vein, and so to the liver.

(2) *Pyæmia* may occur (and in the graver varieties is frequently seen) from absorption of the products of decomposition.

How is this disease diagnosed?

Dysentery is known by the bloody stools, which contain mucus, but no fæcal matter, and by the tenesmus, together with the constitutional symptoms.

(1) Differential Diagnosis.

(1) *From muco-enteritis*, it is differentiated, by the character of the stools, the tenesmus, and the absence of diarrhœa and of griping umbilical pains.

(2) *From enteritis*, by the absence of constipation followed by diarrhœa, and of griping umbilical pains, and by the presence of tenesmus and bloody stools.

(3) *From proctitis* (a rare affection, occuring from piles or some mechanical irritation of the rectum), by the short duration, the very slight constitutional disturbance, and the stools, which are never offensive in the latter disease.

(4) *From typhoid fever*, by the character of the stools, the history, and the absence of the characteristic temperature record and eruption.

What is the prognosis?

An ordinary case recovers in eight or ten days. The more frequent the stools the worse the case.

Bad cases may last three weeks with a remittent fever, the patient getting better and then worse again and finally, if it ends in recovery, the disease gradually subsiding and leaving the sufferer with irritability of the bowels for several months. This is particularly the case in a malarial country.

Very bad cases, such as those in which the disease runs into the gangrenous form, are generally complicated with pyæmia; collapse occurs, and the disease runs its course rapidly, terminating fatally in fifty per cent. of the cases. In these cases the stools are very fœtid and contain shreds of decomposing tissue, membrane and pseudo-membrane. If recovery takes place, the bowel remains in an ulcerated condition, and chronic dysentery results.

What is the treatment?

The diet must be bland, consisting of milk, arrowroot or light broths.

Special Plans of Treatment.

(1) *The ipecac plan.* This consists in giving the patient large doses of ipecac combined with small doses of opium every few hours. In India, ipecac is given in doses of ʒ i–ij every two hours, but in this country, at least, smaller doses (gr. xx) seem to answer better. This treatment is particularly efficacious in puerperal dysenteries.

(2) *The purgative plan* consists in combining small doses of opium with castor-oil, or one of the salines.

If a saline is preferred to the oil, Rochelle salt is probably the best. It should be given in small doses frequently repeated until ℥j has been administered in the first twenty-four hours, after which it is decreased.

(3) *Antiseptic plan* consists in administering such antiseptics as salol, beta-naphthol, etc. by the mouth or in the use of injections of solutions of creolin, thymol, or even of very weak solutions of corrosive sublimate. The results promise well for the future of this plan of treatment.

(4) *The opium treatment* consists in giving gr. ss of the drug every two hours, until an impression is produced on the system; or in administering morphine hypodermically in corresponding doses. The drug acts better than its alkaloid in this disease.

If no effect is produced in forty-eight hours under either of the two former plans of treatment, they should be abandoned, and the opium treatment tried.

If for any reason none of them can be used, or if they fail, bismuth (gr. x–xx), or lead acetate (gr. ij), should be given every two hours, combined, in either case, with opium (gr. ss).

If the dysentery becomes croupous or gangrenous, or if the typhoid state supervenes, brandy must be freely given, and quinine administered as a tonic. Turpentine is serviceable in these cases.

If there is much tenesmus, opium must be administered by suppository or enema.

Locally, washing out the bowel several times a day with ice-water, not only relieves the tenesmus, but gets rid of the mucus, which is adding to the irritation.

Chronic Dysentery.

What are the pathological changes in chronic dysentery?

The mucous membrane is thickened, its surface is ulcerated, the ulcers being irregular in outline, and of an unhealthy appearance. The remains of old ulcers can be seen, the tissue being cicatrized, and often showing actual loss of substance.

Abscesses of the liver are often found, due to emboli washed into the liver from the inferior hemorrhoidal vein.

Sometimes (not often) little polypi are found in the rectum.

What are the symptoms of this disease?

From four to twenty passages take place from the bowels in twenty-four hours, mostly in the daytime. They are persistently

loose, and consist partly of mucus and pus, but contain some fæcal matter also, and sometimes blood. They are not very large, but are extremely offensive. The abdomen is sunken and tender; the patient emaciated, the face of an earthy, ashen hue, and wearing constantly an anxious expression. The spirits are depressed. There is no appetite, and toward evening there is some rise in temperature, amounting to slight fever. There is a good deal of tenesmus from which the patient suffers nearly all the time.

Later, malnutrition is manifested in various ways, as by ulceration of the cornea, anæmia, dropsy, cardiac disturbances, etc.

What is the diagnosis and prognosis?

THE DIAGNOSIS is made from the history of the case, the tenesmus, and the character of the stools.

THE PROGNOSIS is grave. This is a serious disease from which few really recover, although life may be prolonged for years.

What is its treatment?

The diet must be restricted to articles of food which are easily digested, as milk, meat, soft-boiled eggs, rice, corn-starch, or arrow-root. Very little bread should be allowed.

Bismuth (gr. x) combined with opium may be given four or five times a day, and is particularly efficacious in the chronic dysenteries of children. Mineral acids (especially the nitro-hydrochloric acid), or the mineral astringents (as cupric sulphate, gr. $\frac{1}{12}$–$\frac{1}{8}$, if it does not nauseate; zinc sulphate, gr. j–ij, or silver nitrate, gr. $\frac{1}{4}$, or oxide, gr. ss) combined with opium and administered four times daily, are often serviceable. Iron, if it agrees well with the stomach, is an excellent remedy, acting not only as an astringent, but as a hæmatinic as well. It may be given in the form of the sulphate (gr. ij) or as Monsel's solution (ℿij–v) or as the solution of the nitrate (not officinal) (gtt. xx–xxx, *t. i. d.*). Opium may also be employed in suppository (gr. j) at night. Various injections into the bowels have been tried with a view of healing the ulcers. Silver nitrate has been used in this manner without much effect.

If everything else has been tried and has failed, turpentine (gtt. x–xv) may be given in emulsion with a little morphine, and small blisters applied over the spot of greatest tenderness on the abdomen.

Typhlitis and Perityphlitis.

What are these diseases?

Typhlitis is an acute or chronic inflammation of the cæcum, ascending colon and often of the vermiform appendix (the latter sometimes spoken of as *appendicitis*), characterized by the signs and symptoms of localized peritonitis, by fever, and often by the signs of intestinal obstruction. It often results in ulceration, etc., sometimes in perforation of the bowel.

Perityphlitis is an acute inflammation of the connective tissue in the neighborhood of the cæcum, usually resulting in the formation of pus and characterized by the signs and symptoms of an abscess. There is often a localized peritonitis associated with it.

What are the causes of these affections?

They are not uncommon diseases, and are caused by mechanical irritation from the lodgment of foreign substances, as seed or hardened fæces, in the cæcum or appendix vermiformis; by injuries to the abdomen, or by extension of inflammation from contiguous structures. Occasionally they arise from exposure to cold and damp.

What are their symptoms?

In typhlitis there is localized pain associated with tenderness over the region of the right iliac fossa and ascending colon, with more or less swelling; the patient lies on his right side, to relax the abdominal muscles; his bowels are usually constipated, or there may be alternating constipation and diarrhœa, the passages consisting of small watery stools which have found a passage between the hardened masses of fæces occupying the sacculations of the large bowel. There is fever, which in severe cases may be quite high.

Vomiting often occurs, and in case there is complete obstruction of the bowel, it will finally become stercoraceous in character.

If the impaction persists, or if perforation of the intestine occurs from ulceration, general peritonitis will result, with great depression, and usually terminate fatally, sometimes in a few hours.

Perityphlitis usually develops slowly with paroxysms of acute pain in the right iliac fossa, and the presence of a hard mass in the same

situation, which, if suppuration takes place, becomes soft and fluctuates. The symptoms of the suppuration will then be added, as irregular chills and fever attended with profuse sweating.

If it follows typhlitis, all the symptoms of the latter will increase much in severity and be superadded to those of the former.

How are these diseases diagnosed?

The diagnosis is often obscure. Typhlitis is recognized by the fever, the pain and tenderness confined to the right iliac fossa, and constipation, or the constipation alternating with diarrhœa. Perityphlitis, by the paroxysms of pain, and the hard or perhaps fluctuating mass felt over the same situation.

Differential Diagnosis.

(1) *From Malignant Growths in the Iliac Fossa.*—These are harder, grow more slowly at first, more rapidly in their later stages, but are never associated with fever, and finally produce an unmistakable cachexia, with great emaciation.

(2) *From Ovarian Tumors.*—In these cases the history is very different; there is no fever; they are very slow in their growth; there are no symptoms of gastro-intestinal disorder, and the pain and tenderness is not so great. An examination per vaginam or a recto-abdominal examination, will show the connections of the tumor.

(3) *From an Aneurism.*—The symptoms and physical signs of this disease are so distinct from those of perityphlitis, that a mistake is inexcusable.

(4) *Diagnosis between Typhlitis and Perityphlitis.*—In the latter affection the local tenderness and marked signs of local inflammation are more pronounced, and a tumor is present in the right iliac fossa from the beginning of the disease, which will fluctuate if suppuration occurs.

What is the prognosis of these affections?

The prognosis in typhlitis is good, if ulceration does not occur. Perforation is almost always fatal.

In perityphlitis the prognosis depends upon where the abscess opens and upon free drainage.

What is the treatment?

Absolute rest in bed must be insisted on, and a mild diet with a fever mixture prescribed.

In typhlitis, when the case is seen *in the early stages*, leeches must be applied, and they in turn followed by ice-bags, used intermittently. Opium must be given by the mouth, rectum or hypodermically, to allay the pain, and salines or castor oil administered. For this purpose magnesium sulphate, with diluted sulphuric acid and syrup, or Rochelle salts in small but frequently repeated doses, answers best.

If ulceration with perforation and peritonitis should occur, large doses of opium must be given and the patient's strength kept up by stimulants, frequent feeding, and other supporting measures.

Laparotomy and removal of the sloughing or ulcerating portion of the bowel has been resorted to, and occasionally with success. It is a desperate remedy after perforation has taken place. The surgical treatment has, however, been more successful in those cases in which the disease has become chronic, where the patient is suffering from repeated attacks of the acute malady, and in which eventually, perforation is almost certain to result. It is better to operate in the interval between the exacerbations.

If perityphlitis results, the remedies must be pushed, and an occasional blister applied over the iliac fossa. *If it increases*, poultices must be used. If pus forms, quinine should be given, and the abscess evacuated as early as possible, the parts being treated antiseptically. This is better than aspirating the abscess. It must be borne in mind that all pus in the neighborhood of the cæcum has a fæcal odor, whether it connects with the bowel or not, hence the odor of the pus cannot be relied on to determine whether there is a perforation of the intestine.

Proctitis.

What is proctitis?

Proctitis is a catarrhal inflammation of the mucous membrane of the rectum, caused by irritation from hemorrhoids, the habitual use of enemata, or other mechanical irritants, as hardened masses of fæces in persons of a constipated habit. It occasionally arises from cold and exposure, as from sitting on the damp ground. It is not a very frequent disease.

What are its symptoms?

There is an uneasy sensation in the rectum, with a constant desire to have a passage, in bad cases. The stools are preceded and followed by burning in the rectum and tenesmus, which is often very great. They consist of mucus or of muco-pus, which is bloody, or at least streaked with blood. In severe cases there is some febrile reaction, and inflammation of the surrounding cellular tissue may occur (periproctitis), which usually results in suppuration and the formation of anal fistulæ.

What is the prognosis?

The prognosis is favorable in uncomplicated cases. Periproctitis adds much to the patient's suffering, and while not a fatal disease in itself, may lead to pyæmia; or the resulting fistulæ may render him a chronic invalid. Hepatic abscess occasionally results from this disease in the same manner that it does from dysentery.

What is the treatment?

The bowels should be evacuated by salines, aided by injections of oil and opium, administered principally by enemata with starch water. The stools should be rendered soft, so as to produce as little irritation as possible, and a daily movement should be insisted upon. Warm enemata of plain water give much relief, both by softening the fæcal masses in the rectum, and by removing the mucus and muco-pus, which, by collecting there, keeps up the irritation, and produces much of the pain and tenesmus.

If periproctitis ensues, it should be treated in the same manner as abscesses in other situations.

Intestinal Obstruction.

What is intestinal obstruction?

It is a closure of the intestinal canal, produced by fæcal accumulations, peritoneal adhesions, herniæ, twisting of the intestine or invagination, and characterized by severe pain, obstinate constipation, stercoraceous vomiting, and, if speedy relief does not occur, collapse and death.

Describe the symptoms of these affections.

The symptoms may come on gradually or suddenly, and manifest themselves first, as a rule, by invincible constipation, which cannot be overcome either by injections or by purgatives, both of which the patient has probably tried before calling medical aid.

There are intense pains diffused over the abdomen, colicky at first, soon becoming very violent, somewhat paroxysmal, and associated with great tenderness. The intestines are in continual motion, and their peristaltic action becomes so violent that it can be seen through the abdominal wall. There is vomiting, first of the contents of the stomach, later of bilious matters, and finally of a nauseous fluid, containing small masses of fæcal matter and having the characteristic smell of fæces. This is called *stercoraceous vomiting*, and is supposed to be due to reversed peristalsis.

If the patient is not soon relieved, collapse will supervene, with a cold clammy skin, feeble thready pulse, sunken eyes, and death will ensue. If the case terminates in recovery, small amounts of fæcal matter will be passed from the bowel, and the patient slowly recovers.

How are these diseases diagnosed?

The diagnosis is often very difficult. Obstruction is recognized by the obstinate constipation, the pain and tenderness, followed by violent peristaltic action, and stercoraceous vomiting with collapse.

DIFFERENTIAL DIAGNOSIS.

(1) *As to variety:*—

(*a*) *If from fæcal accumulation*, there is generally a history of habitual constipation, and the obstruction comes gradually.

(*b*) *If from peritoneal adhesions*, there is usually a history of preceding peritonitis.

(*c*) *If from hernia, sudden twisting of the intestinal loops* (*volvulus*), *adhesions or invagination*, the obstruction is generally very sudden, and often occurs when the patient is in perfect health.

(*d*) *Invagination* is common in children, and probably often occurs as a temporary condition, a portion of the bowel slipping into another portion, and after a time slipping out again, without causing any symptoms whatever. Should it remain invaginated from any cause for a sufficient length of time, adhesions will form between the peritoneal surfaces of the two portions of intestine, which will

prevent its return. The invaginated portion becomes strangulated, and symptoms due to this condition arise. Occasionally the invaginated portion sloughs off, and if the adhesions holding the two portions together are sufficiently strong, recovery may take place, the slough passing off by the rectum.

The symptoms are those already described; there is also generally an elongated tumor in the abdomen, tender to the touch; the discharges consist of blood and mucus, with some pus, but the local symptoms are evidently higher up in the abdomen than they are in dysentery, and there is stercoraceous vomiting.

(2) *Of the site of the obstruction.*

There is usually no positive way of diagnosing the site of the obstruction. If it is high up in the duodenum or upper part of the ileum, it is said that the amount of urine passed will be small. There is usually great tympanitic distention above the seat of the obstruction.

(3) *Diagnosis from peritonitis.*

In the latter disease there is much more diffused pain and tenderness, and more constitutional disturbance early in the case. There are also some fæcal discharges from the bowel.

What is the prognosis?

The prognosis depends upon the cause. In fæcal impaction it is favorable; in twists or adhesions, unfavorable; in invagination the patient may recover, if the bowel sloughs and inflammatory adhesions glue the two ends together.

What is the treatment?

No matter what the cause, all purgatives must be discontinued as soon as a diagnosis of intestinal obstruction is made. Opium should be given, both for the pain and to quiet the peristalsis. *If the obstruction is from invagination*, the opium must be given in very large doses. Various methods of distending the bowel have been resorted to, with the idea of causing the invaginated portion to slip back, or of aiding the bowel to untwist: thus, copious warm-water enemata have been slowly forced into the bowels by means of a long tube and a funnel or fountain syringe elevated above patient's head, thus using hydrostatic pressure in making the injection.

This sometimes acts very well, as does also large injections of

warm sweet-oil, particularly in those cases caused by impaction of fæces, but if the case is one of invagination, and the bowel is beginning to slough, the adhesions not yet being firm, this practice is not unattended with danger. The latter remark also applies to injecting large amounts of air into the bowel, and of causing the generation of carbonic anhydride by injecting solutions of tartaric acid and sodium bicarbonate.

If the situation of the obstruction can be diagnosed, laparotomy and the removal of the strangulation, or resection of the sloughing mass, should always be attempted. As, however, every inch of intestines which passes through the hands of the operator increases the danger to the patient, the operation does not give very satisfactory results, because the diagnosis of the site of the obstruction is uncertain.

The excessive tympany may be relieved by aspiration, but the gas will rapidly reaccumulate.

(5) DISEASES OF THE PERITONEUM.

Acute Peritonitis.

What is acute peritonitis?

It is an inflammation of the peritoneum, which may be either localized or diffused, and is characterized by intense pain and tenderness, tympanites, fever, a small, tense pulse, vomiting and prostration.

What are its causes?

It may be caused by traumatism (blows and injuries to the abdominal wall); may depend on perforation of the intestines (as in typhoid fever, typhlitis, or ulcer of the stomach); abscesses bursting into the peritoneal cavity also produce peritonitis. Cold and exposure, the extension of inflammation from adjacent structures, or puerperal inflammations also result in peritonitis.

What is the pathology of this disease?

The peritoneum is at first dry and the vessels are irregularly engorged. In from twenty-four to forty-eight hours a plastic exudation (embryonic tissue) occurs upon the surface of the membrane,

which then appears as if smeared with a thin solution of gum. Soft, fibrous bands are seen holding the coils of the intestine together.

If much lymph is effused and but little serum, these bands are converted into granulation tissue, which, in its turn, is transformed into connective, and finally into fibrous tissue, resulting in firm adhesions.

If, however, there is much serum and but little lymph effused, a turbid, bloody fluid, containing flocculi of lymph, will be found occupying the peritoneal cavity. This fluid may be absorbed or may remain permanently. The visceral, as well as the parietal, layer of the peritoneum shares in the inflammation. The bowel is paralyzed and distended with gas. There is no inflammation of the mucous coat of the intestine.

What are the symptoms of peritonitis?

LOCAL SYMPTOMS.

There is extreme, diffused, extensive tenderness, the patient lies with his thighs and knees flexed on the abdomen, and the legs on the thighs, so as to relax the abdominal muscles as much as possible. There is marked tympanites, and constipation, which sometimes alternates with diarrhœa.

GENERAL SYMPTOMS.

There is a chill, marked fever (the temperature ranging from 102° to 105°), with a morning remission and evening exacerbation. The pulse is frequent, tense and small, feeling like a whip-cord or fine wire under the finger. Vomiting is a constant feature of this disease, and is both an early and a late symptom. There is great restlessness, anxiety, and, in bad cases, pinched features and collapse.

What is its duration and prognosis?

If it is due to perforation, it is generally fatal; the patient dying in from two to five days. If it follows traumatism, or arises spontaneously, the prognosis is more favorable, recovery often taking place after two or three weeks. It is always a grave disease. Localized peritonitis is serious in proportion to the extent of peritoneum involved, and its tendency to spread.

How is this affection diagnosticated?

Peritonitis is recognized by the great pain, the tenderness, the fever, the pulse, the vomiting and the constipation.

Differential Diagnosis.

(1) *From enteritis.* In the latter disease the tenderness and other symptoms are local, not diffused.

(2) *From rheumatism of the abdominal walls.* In these cases the temperature is not above 100°; there is no obstinate constipation, no vomiting, but a rheumatic history and, perhaps, some swelling of the joints.

(3) *Hysteria* sometimes simulates peritonitis. In these cases there is no fever as a rule, or if there is, the temperature varies greatly in the course of a very short time. The patient rarely assumes the characteristic position of peritonitis, and although there is generally great tympanites and the patient complains of much pain on the slightest touch, yet if her attention is distracted from the abdomen, the pressure may be gradually increased without any complaint. There are usually other symptoms of hysteria present, or at least there is a history showing that she is of an emotional disposition.

What is the treatment of peritonitis?

Local Treatment.

If seen early, before it has become diffused and before effusion has taken place, leeches applied to the abdomen and followed by the intermittent application of ice, or of cold water (the German method of treatment) is very serviceable. Later in the disease, hot cloths sprinkled with turpentine and laudanum, or with the former alone, are indicated.

After effusion has taken place, an ointment of equal parts of mercurial and belladonna ointments may be used, or the turpentine fomentations continued. Poultices are indicated, but are objectionable in diffused peritonitis, on account of their weight.

General Treatment.

Opium must be given for the relief of the pain. It is given for its effect, independent of the dose required to produce that effect. Formerly the deodorized tincture was given to an adult in doses of gtt. xx every hour, and doubled after the second dose if it was well

borne; or morphine administered until the patient was thoroughly under the influence of the drug, when the deodorized tincture was substituted for it, with the idea of keeping the bowel perfectly at rest; of recent years, however, the so-called *saline treatment* has been growing steadily in favor. This consists in administering small but repeated doses of some saline, as Rochelle salts, continuously throughout the disease, only giving sufficient opium to allay the severe pains. Atropine is sometimes advantageously combined with the opium treatment. The opium may also be administered by the bowel, but answers best when given by the mouth, provided the stomach is not too irritable to tolerate it.

When there is much tympany, turpentine (gtt. x) in emulsion, may be given if the stomach is not too irritable.

If the peritonitis is due to perforation, stimulants must be given to sustain the powers of life.

Peritonitis from puerperal causes must be treated by stimulation, tonics (as quinine) and digitalis, at the same time that the most thorough antiseptic injections are used for the vagina and uterus.

Chronic Peritonitis.

What is chronic peritonitis?

It is a chronic inflammation of the peritoneum, generally following an acute attack, but which, in tubercular subjects particularly, may be chronic from the commencement.

What is its pathology?

Tubercle is nearly always found on the coils of intestine in a case of any duration, whether it occurs in a tubercular subject or follows an ordinary acute attack. The mesenteric glands, also, are cheesy and the seat of tubercular deposit.

What are its symptoms?

The abdomen is enlarged and tender. Dropsical fluid is present in the abdominal cavity, giving rise to fluctuation. There is often a history of tubercle, with hectic fever, and perhaps signs indicating tuberculosis of other organs, as the lungs.

What is the treatment?

Counter-irritation over the abdomen is advantageous, and is best produced by small flying blisters.

Diuretics and diaphoretics should be given to cause absorption of the effused fluid, and iodine and cod-liver oil administered to promote the absorption of the tubercle. The patient should be well and systematically nourished, and should use stimulants in moderation.

(6) DISEASES OF THE LIVER.

Icterus.

What is icterus?

Icterus or jaundice is a symptom of disease of the liver, not a disease itself. It may be due to obstruction of the bile ducts either from catarrhal inflammation, from gall-stones, or from various mechanical causes, such as plugs of mucus in the gall ducts, etc. It may also be due to disorders of the blood, as in pregnancy, yellow fever, malaria, following snake bites, etc., or to disease of the liver itself, as in acute fatty degeneration, or to mental emotions, as violent anger.

What are its symptoms?

A yellow discoloration of the entire skin, mucous membranes and conjunctiva, is seen in strong daylight. All objects appear to the patient to have a yellow hue. The urine when tested on a white porcelain plate by fuming nitric acid, gives a play of colors. The pulse is slow, the temperature is depressed, there is intense itching of the skin and conjunctiva, gastric disturbance, constipation with clay-colored stools, from want of bile, although sometimes they may be black and offensive.

What is the pathology of jaundice?

Where there is obstruction of the ducts, resorption takes place of the bile which has been previously formed. The skin and mucous membrane endeavoring to excrete this bile from the blood become tinged with its coloring matters and the various symptoms are due to the bile in the circulation. Where no obstruction of the ducts exists and the bile is not formed, the hydrocarbons which are usually converted in the liver into urea or uric acid are probably converted into biliary acids in the blood, and produce the same train of symptoms.

How is jaundice recognized?

The yellow conjunctiva and skin, the play of colors in the urine when treated with nitric acid, establish the diagnosis.

What is the prognosis?

The prognosis depends upon the cause. Where it lasts, from any cause whatever, for more than three months, dilatation of the bile ducts and alteration of hepatic cells is apt to result.

How is it treated?

The disease of which it is a symptom should be ascertained and treated. The skin should be kept active by baths and diaphoretics. The action of the kidneys should be maintained by diuretics, and the portal circulation should be unloaded by purgatives, especially salines, as sodium phosphate, Rochelle salt, potassium bitartrate or acetate, or magnesia sulphate. Podophyllin and calomel are sometimes required. For the itching of the skin let the patient soak himself in warm water with sodium bicarbonate (1 ounce to a gallon), or potassium carbonate or borax. Sponging with carbolic-acid water is sometimes efficacious. If these means fail, potassium bromide (gr. xxx) at bedtime will sometimes prove useful.

Acute Hepatic Hyperæmia.

What is this disease?

It is an acute congestion of the liver, not very common in this country, caused by very hot weather, by arrested menstruation or sometimes by errors in diet.

What are its symptoms, duration and treatment?

There is pain and weight in the hepatic region, with increased dullness on percussion. Slight jaundice; anorexia; coated tongue; depressed spirits; constipation, and sometimes vomiting. The disease lasts for from five to six days, and gradually passes away.

TREATMENT.

The diet should consist principally of animal foods, broths, etc., with very little starchy or fatty matters. The patient should be given small doses of calomel (gr. $\frac{1}{4}$) with bicarbonate of sodium, frequently repeated and followed by a saline. If there is much pain over the liver leeches and cups are to be employed.

Chronic Hepatic Hyperæmia.

What is this disease?

It is a chronic congestion of the liver, more common in this climate than the acute disease, and caused by chronic cardiac diseases, especially dilated heart, long continued heat, or hemorrhoids. It is chiefly due to interference with the abdominal circulation.

What are its symptoms?

There is a feeling of weight and tension over the cardiac region, with a bad taste in the mouth; black, offensive stools; moderately increased area of percussion dullness; slight jaundice; depressed spirits; drowsiness; mental hebetude; nausea, often vomiting; anorexia, and usually the patient imagines he is the subject of an incurable affection.

What is the prognosis of chronic congestion of the liver?

The prognosis of chronic, as of acute congestion, is good as far as the attack itself is concerned. If it is caused by a chronic disease it will return, and the ultimate prognosis will depend upon the affection which produces it.

How is it treated?

The diet and general treatment should be the same as in the acute disturbance. The patient should take active exercise.

Alkalies should be administered internally. He should have a Turkish bath twice a week, and friction should be employed over the liver.

Acute Hepatitis.

What is acute hepatitis?

It is an acute inflammation of the liver seen particularly in tropical climates, due to long continued heat and errors in diet, with want of exercise.

What are its symptoms, duration and termination?

The patient complains of great pain and tenderness in the hepatic region. There is high fever with evening exacerbations and morning remissions; constipation, and often clay-colored or black stools;

moderate jaundice, and frequently irregular sweats. The duration is from one to two weeks, and it terminates by gradual recovery, or by the formation of abscesses, which may be recognized by recurring chills, repeated sweats and exhaustion.

How should it be treated?

In the early stages, leeches or cups should be applied over the liver. Purgatives, as calomel and the salines, should be administered. The diet should be restricted, alkaline diaphoretics should be given, and, in the later stages, quinine should be administered.

Chronic Hepatitis.

What is this disease?

It is a chronic inflammation of the liver, occurring especially in India and hot climates, and commonly known as "*liver disease.*" The symptoms are the same as those of chronic congestion, excepting that at times there are irregular accessions of fever and a variable increase in the size of the liver.

The danger in these cases is the formation of abscesses.

The patient will usually recover on going to a cooler climate.

TREATMENT.

The treatment is the same as in chronic congestion. It is of importance to keep up the action of the skin. Quinine should be also administered, and the occasional use of massage over the liver has a good effect.

Acute Catarrh of the Bile Ducts.

What is this affection?

It is an inflammatory disease of the common choledoch duct, spreading to the smaller ducts and finally to the liver itself. It is more commonly known as *catarrhal jaundice*, is due to errors in the diet, to malarial or epidemic influences, or the extension of inflammation from the duodenum.

What are its symptoms and duration?

It usually begins suddenly with gastro-enteric disorder for a day

or two; marked jaundice supervenes with tenderness or swelling over the liver, nausea, vomiting, anorexia, clay-colored stools, slow pulse and intense itching of the surface, and other symptoms common to jaundice, and sometimes, symptoms referable to gout. There is no fever. After two or three weeks it gradually passes away. Sometimes the jaundice persists for several months, and secondary inflammation and hardening of the connective tissue ensues. This is especially seen in old people, particularly those subject to gout.

What is the prognosis?

The disease usually passes away gradually, the patient being convalescent in about six weeks. Sometimes, however, the case grows steadily worse, and nervous symptoms, as stupor and delirium, supervene and are followed by death. In these cases there is usually permanent occlusion of the bile ducts from adhesive inflammation.

What is the diagnosis and treatment of this affection?

DIAGNOSIS.

The disease is recognized by the history and gastric symptoms, succeeded by jaundice and marked hepatic swelling.

TREATMENT.

Unload the portal circulation by means of salines, particularly sodium phosphate, given in drachm doses three or four times a day. Act on the kidneys by alkaline diuretics, such as potassium acetate, and on the skin by alkaline baths. Mercury is not generally serviceable in the early stages. Locally, if seen in the beginning, a few leeches, followed by poultices, in the region of the liver, or if seen later, small fly blisters in the same situation, are of use.

When the disease does not yield readily, or the patient is of gouty diathesis, colchicum or potassium iodide may be used. Should the disease become chronic or the patient be syphilitic, corrosive sublimate or potassium iodide should be employed. If the disease is very obstinate, ammonium chloride (gr. xv–xx *t. i. d.*) will sometimes have a good effect.

For the intense itching of the skin the same treatment should be employed as has already been spoken of under jaundice.

Passage of Gall-stones.

What are the symptoms of the passage of gall-stones?

There is intense pain recurring in paroxysms in the epigastrium, or right hypochondriac region; violent retching and vomiting, and, if the attack lasts, intense jaundice.

All the symptoms rapidly disappear when the stone passes into the bowel.

What is the duration of an attack?

The passage of a gall-stone usually occupies from two hours to two or three days. It may, however, last a long time, even thirteen or fourteen months.

What is the termination of the attack?

Usually the stone passes into the bowel through the duct, and rapid recovery takes place. Gall-stones may, however, cause ulceration, and escape into the peritoneal cavity, or, when adhesion precedes the ulceration, they may pass into the bowel through a fistulous opening. Sometimes they become impacted and give rise to chronic catarrhal jaundice, and multiple abscess of the liver may result.

Of what do these stones consist?

They consist of cholesterin, mucus, bile, etc. They vary in their size and in number.

How should this affection be treated?

During the passage of a stone morphine should be administered hypodermically in doses of gr. $\frac{1}{6}-\frac{1}{4}$, combined with atropine gr. $\frac{1}{100}-\frac{1}{80}$, repeated every two hours, if necessary, to allay the pain. Ether or chloroform may be inhaled for the same purpose. Large amounts of weak alkaline waters, as sodium carbonate, gr. ij to the pint, taken hot, relieves the nausea and vomiting, and perhaps facilitates the passage of the stone.

If it is long in passing, or if secondary catarrh results (shown by the tenderness and jaundice), leeches followed by poultices should be employed, and purgatives, as sodium phosphate, podophyllin, or even mercurials, should be administered.

To dissolve the stones or prevent their formation, Durand's remedy (ether three parts, turpentine one part, gtt. x–xx, three times a day, on an empty stomach), or chloroform (gtt. x–xx, well diluted, three times a day), have been employed. Neither of these remedies, however, are of much avail. The patient should take active exercise in the open air, and should drink alkaline mineral waters, or weak solutions of sodium carbonate, and magnesium sulphate occasionally. Small doses of the mercurials should be given at intervals.

Hepatic Abscess.

What are the causes of abscess of the liver?

It usually follows acute or chronic hepatitis in hot climates, or may occur after dysentery. It is especially frequent in malarial districts.

What are its symptoms?

The symptoms are very latent, sometimes there being no symptoms but depressed spirits and sallowness of the skin. Generally, however, there is pain with a sensation of weight and itching in the hepatic region; nausea and vomiting; clay-colored or black stools; irregular chills, and a remittent fever which is not influenced by quinine; irregular sweats; emaciation and scanty, high-colored urine, depositing urates on standing; tenderness and swelling over the right lobe of the liver; the right rectus muscle is rigid and there is great throbbing of the abdominal aorta. Fluctuation is present, at first obscure, but becoming more and more marked; jaundice is as often absent as present, excepting in cases of multiple abscess.

In multiple abscess the swelling is more uniform, there is much more constitutional disturbance, the symptoms being of a marked asthenic type, and the disease runs a more rapid course, lasting for a few weeks only, while ordinary abscesses last for months.

What is the morbid anatomy?

An hepatic abscess is ordinarily large and single, is usually situated in the right lobe and contains from an ounce to a gallon of pus. Pyæmic abscesses are multiple and are usually preceded by extravasation of blood, and plugging of the vessels. They follow dysentery, etc.

How is abscess of the liver recognized?

Abscess of the liver is diagnosed by the history of liver disease with the physical signs of an abscess in the hepatic region, by the hectic fever, chills and other general symptoms of abscess.

DIFFERENTIAL DIAGNOSIS.

(1) *From Cancer of the Liver.*—By the fever, the fluctuation, the absence of hard nodules, and the localized, not diffused, tenderness.

(2) *From Cancer of the Stomach* (where there is marked gastric symptoms and swelling over the pylorus).—By the fever and jaundice.

(3) *From Abscess of the Abdominal Walls.*—By the absence of the history of traumatism, by the long duration and by the use of accurate percussion of the liver in conjunction with hepatic symptoms. In case of abscess of the abdominal walls, motion is much more difficult and painful than in the abscess of the liver, and the exploring needle introduced into the abscess may bring away shreds of muscle with the pus.

What is the prognosis?

Abscess of the liver is always a serious disease.

Pyæmic abscesses terminate fatally. The prognosis of the ordinary abscess depends upon its size, number, and the destruction of the surrounding tissues, and upon the direction in which it opens. Should it open into the lung, the prognosis is favorable; into the intestine or through the abdominal wall, it is favorable also; into the peritoneal cavity, it is almost invariably fatal. The patient may die worn out by the hectic fever.

How should it be treated?

As soon as pus is present it should be evacuated either by aspiration or by abdominal section. In case of doubt, it is best to aspirate. The patient's strength should be sustained by good food, tonics and stimulants, and quinine should be administered. The sulphites have been employed, but are not of much service.

Interstitial Hepatitis.

What is interstitial hepatitis?

It is a low grade of interstitial inflammation of the liver substance, resulting in the hypertrophy of the connective issue, which finally, by contracting, presses upon the liver cells and the vessels, interfering with their functions.

What are its synonyms?

It is called cirrhosis or sclerosis of the liver, gin-liver, or hob-nail liver.

What are its causes?

The chief cause is alcohol, especially when it affects man. Sometimes it arises without apparent cause, particularly among women. Sometimes it is produced by inherited syphilis, especially in children. Occasionally, it is seen after long-standing disease of the liver, or from congestion due to cardiac (mitral) disease.

What are its symptoms?

There are general dyspeptic symptoms from congestion or chronic catarrh of the stomach, produced by disturbance of the portal circulation; enlarged spleen, congestion or hemorrhage from the intestine, or bleeding piles, produced in the same manner.

Abdominal dropsy (*ascites*) is a common symptom. The symptoms referable to the liver itself are not well marked. Jaundice may be slight or absent. The liver in the first stages is enlarged, afterwards diminished in size; the diminution being difficult to detect, frequently, on account of the dropsy. There is difficulty in the digestion of fatty substances because the bile is altered in character. The liver does not properly defibrinate the blood, the urea remains in the blood and system, and the patient is pale and anæmic. There is a tendency to low grades of inflammation of serous membranes, such as peritonitis, pericarditis or pleurisy.

It is a chronic, progressive disease, lasting for years; the patient finally dying, worn out from exhaustion, dropsy or, with cerebral symptoms, from uræmia.

What is its pathology and morbid anatomy?

A low grade of inflammation of the interstitial connective tissues

around the minute vessels and ducts of the liver takes place, and gradually leads to an increase of the connective tissue (hyperplasia) along the capsule of Glisson, with swelling and enlargement of the liver. Finally, by contracting, it compresses the lobules, and causes an irregular-shaped (hob-nail) liver, diminished in size.

Under the microscope the hepatic cells of the compressed lobules are atrophied, and have undergone granular degeneration; the radicles of the hepatic ducts and vessels are compressed and obliterated or tortuous and enlarged.

When a section is made, bands of connective tissues are found between the lobules, causing hardness and resistance to the knife.

How may this disease be recognized?

The diagnosis is made from the history of steady drink; chronic heart disease, etc. From the gastric, splenic and intestinal disturbance, and the tendency to hemorrhage; from the ascites, the enlarged abdominal veins, and area of hepatic dullness which is at first increased, afterwards diminished.

What is the prognosis?

If treated early, before contraction takes place, the patient may recover; after contraction occurs, life may be prolonged, but the disease can never be cured.

How should it be treated?

In the early stages, before contraction has taken place, leeches may be applied, and the salines, as magnesium sulphate or potassium bitartrate should be administered, conjoined with potassium iodide.

After contraction has commenced, no radical cure can be accomplished. Corrosive sublimate in small, repeated doses, alternating with potassium iodide, is supposed to retard the progress of the disease.

No matter when seen, the portal circulation should be acted on by salines, and the secretion of the kidneys maintained by digitalis or infusion of broom.

For the gastric symptoms regulate the diet, avoid alcoholic stimulants, and treat as occasion may require.

For Dropsy.—When diuretics and diaphoretics fail, paracentesis abdominis must be performed.

Acute Yellow Atrophy.

What is acute yellow atrophy?

It is an acute inflammation of the hepatic cells, resulting in their degeneration, characterized by rapid atrophy of the liver with deep jaundice and grave nervous symptoms (uræmic?), terminating in a week or ten days. It is also called *acute parenchymatous degeneration of the liver.*

CAUSE.

It is especially seen in pregnant women. It may result from the action of phosphorus, arsenic or other metals, or be due to venereal excesses or syphilis.

SYMPTOMS.

It begins with gastro-intestinal catarrh, headache, slight fever and slight jaundice. The jaundice deepens, the pulse becomes slow, and the headache increases. The liver dullness rapidly diminishes in extent. There is sleeplessness, convulsions, coma and death. The liver is found to be reduced in size and weight, and acute fatty infiltration and degeneration has taken place.

PROGNOSIS.

Almost invariably fatal.

TREATMENT.

Free purgation may be tried in the early stages, and the mineral acids may be administered. Phosphorus in small doses has been recommended.

Fatty Liver.

What is fatty degeneration of the liver?

It is a disease in which a gradual fatty degeneration of the liver cells and a fatty infiltration of the liver tissue takes place, caused by over-eating, especially when rich food is habitually indulged in, by want of proper exercise, or by wasting diseases, such as cancer or consumption.

What are its symptoms, prognosis and treatment?

The patient is pale and sallow, with a greasy complexion. Fatty

or starchy foods are not properly digested, and the stools are discolored, usually black, due to altered bile. The area of hepatic dullness is increased, the margins of the liver rounded, the edges not distinct, but smooth, and there is no dropsy.

PROGNOSIS.

It is not immediately dangerous, but lessens the vitality of the patient.

TREATMENT.

The patient should be put on a regulated diet, and should not be over-fed. He should take active exercise, and should make use of occasional salines and diaphoretics.

Albuminoid Liver.

What is albuminoid liver?

Albuminoid, or *waxy amyloid liver* is a disease characterized by an infiltration of the liver tissues with a peculiar waxy material, resulting in their degeneration, and caused by syphilis, long continued suppuration, especially when caused by bone disease, or by malaria.

What are its symptoms?

There is anæmia and albuminuria which may be slight or absent. The patient suffers from marked dyspeptic symptoms. Fatty and starchy matters are not digested; the stools are usually clay-colored or may be dark, due to altered bile, and the liver is enormously enlarged.

What is the pathology and post-mortem appearance?

There is a deposit of albuminoid or waxy material among the liver cells and along the capillaries, giving rise to an enormous increase in the size of the liver, interfering materially with its functions.

The spleen and kidneys are similarly affected, as a rule. On post-mortem examination the liver is pale, smooth, shining, and has a waxy appearance on section. A solution of iodine passed rapidly over its surface produces a mahogany-brown discoloration.

How is this disease diagnosed?

Waxy degeneration of the liver is recognized by the history and cause, by the enormously enlarged, smooth liver, the absence of jaundice and dropsy, the marked anæmia, the albuminuria and probably the enlarged spleen.

DIFFERENTIAL DIAGNOSIS.

(1) *From Cancer of the Liver.*—In the latter disease the liver is irregular in outline and nodulated.

(2) *From Hydatid Cyst of the Liver.*—In this case the history and symptoms are different. The cyst may be discovered on palpation, and fluctuation can generally be detected.

What is the prognosis of this affection?

The patient never entirely gets well but improves very much under treatment, and the liver decreases much in size.

How is it treated?

The cause should, if possible, be ascertained and treated,

The patient's diet should be properly regulated, very little fatty, starchy or saccharine food should be given, and he should live principally on albuminous articles.

He should take active exercise, and the occasional use of salines, as Rochelle salt or the natural mineral waters, are efficacious in relieving portal engorgement.

Ammonium chloride (gr. x, increased to gr. xx, *t.i.d.*), continued for periods of three weeks, or until it irritates the stomach, when it may be alternated with the syrup of iodine of iron (gtt. xx–℥j, *t.i.d.*) is the most efficacious remedy. Ammonium iodide may be used, or cod-liver oil may be given with the ammonium chloride.

Cancer of the Liver.

What are the causes of cancer of the liver?

Cancer of the liver is a very common disease and may be either primary or secondary. The liver is the most common seat of internal cancer after the uterus and stomach.

The disease is more frequent in women than in men. It is rarely

seen in young people, generally coming between forty and sixty years of age.

What are the symptoms, duration and morbid anatomy?

SYMPTOMS.

The jaundice, which is slight, except when the cancer presses on the ducts; the dropsy is also slight, except where the cancer presses on the portal vein or vena cava.

Gastric symptoms are common, from interference with the portal circulation. The urine is high colored and may contain leucin and tyrosin. There is pain and tenderness in localized spots, over the region of the liver. The area of percussion dullness is increased, sometimes upwards, but more generally downwards, and is irregular in outline; on palpation nodosities are detected over the liver. There is no fever. As the disease progresses the patient becomes markedly cachectic. The average duration is about one year.

MORBID ANATOMY.

The diseased process is generally diffused throughout the liver in small cancerous nodules, the intervening structures being healthy; the liver is more enlarged than in any other disease, excepting waxy degeneration. The surface is irregular and nodulated.

How is cancer of the liver recognized?

The diagnosis is made from the localized pain and tenderness; from the enlargement of the liver, the irregular outline, absence of fever and the presence of cachexia.

DIFFERENTIAL DIAGNOSIS.

(1) *From Cirrhosis of the Liver* (in the stage of enlargement). In this disease there is no pain, no tenderness, and no cachexia; the enlargement is not as great; the surface of the liver not so nodulated and more dropsy is present.

(2) *From Hepatic Abscess.*—In the latter disease there is no emaciation, and no numerous spots of localized tenderness, but fluctuation and *fever.*

(3) *From Cancer of the Stomach.*—The greater prominence of gastric symptoms should prevent a mistake.

(4) *From Cancer of the Kidney.*—An examination of the urine renders the diagnosis clear.

What is the treatment?

The treatment is palliative. To retard the growth of the cancer, small doses of arsenic have been used persistently, or corrosive sublimate given continuously for a long time. Conium plasters worn over the hepatic region persistently has been said to retard its growth and to relieve the tenderness.

For pain, morphine or opium must be used.

For dropsy, the treatment should be conducted on general principles.

Hydatid Cyst of the Liver.

What are the symptoms of this disease?

Hydatid cyst is not common in this country, but is frequently seen in Iceland and Australia.

The liver does not increase much in size, but as the cyst grows, it projects forward from under the ribs and upon palpation, gives rise to a jelly-like vibration. There is no pain, but little fever and almost no constitutional disturbance.

What is the morbid anatomy?

It consists of a multiple cyst of the liver, due to the ova of the tapeworm from dogs, which, entering the body probably from the food, travel through the portal vein to the liver and there become encysted. The hooklets of the ova are found in the cyst, which also contains a gelatinous fluid causing the vibration on palpation.

What is the prognosis?

Recovery is frequent. The ova are killed by bile and the cysts may shrivel up, or may be discharged into the intestines, lungs or externally. The duration is from five to thirty years.

What is the treatment?

Glycerin administered internally is said to draw the fluid from the cyst and cause it to disappear. Large quantities of sodium in solution are likewise recommended. These remedies are only partially successful and often fail.

The *surgical treatment* consists in aspiration, and the injection of iodine into the cyst. This is attended with some risk, as hepatitis often results. Electrolysis has also been resorted to.

Functional Disease of the Liver.

What are the causes and symptoms of functional disease of the liver?

They are caused by too rich food, with want of exercise; by exposure to great heat; by malaria, or by hepatic congestion. If functional disease of the liver should continue for a long time it will result in hepatic congestion, even should the latter not have existed before.

SYMPTOMS.

There is a bitter taste in the mouth, gastric catarrh, palpitation, loss of memory, listlessness, and a tendency to bronchial catarrh.

SPECIAL SYMPTOMS.

Pain in the right shoulder is a common symptom. *Constipation* usually exists, due to absence or diminution in the secretion of bile. There is alteration of the glycogenic function of the liver, and as the bile-making function is also interfered with, fatty substances are only emulsified irregularly, resulting in *excessive corpulence, or*, in some cases, *emaciation* of the patient. *The urine* is high-colored, scanty and full of urates. Occasionally sugar is found in the urine in small quantities, which, however, passes away under the use of active purges.

DIAGNOSIS.

From organic hepatic diseases. These should always be sought for and excluded.

What is the treatment?

Active exercise should be taken in the open air and baths should be indulged in daily. The diet should be properly regulated, purgatives, as salines or podophyllin, with the occasional use of a blue pill, should be regularly administered. Diluted nitrohydrochloric acid should be given before meals, and may also be used in baths, either to the surface generally, or locally applied to the region of the liver. The pain in the right shoulder is best treated by purgatives.

(7.) DISEASES OF THE PANCREAS.

Pancreatitis.

What is pancreatitis?

It is an inflammation of the pancreas, occasionally terminating in suppuration, affecting women more frequently than men, and occurring without known cause.

SYMPTOMS.

There is pain in the epigastrium, often shifting to the shoulders or back, nausea and vomiting, flatulency and constipation. There is fever, irregular heart, and frequently collapse, terminating in death.

Sometimes the inflammation gradually declines, giving rise to a chronic inflammation, or ending in abscess.

Chronic pancreatitis resembles in its pathology cirrhosis of the liver or kidneys. It frequently gives rise to cysts by obstruction of the ducts.

The symptoms are emaciation, gastric disturbance, and the appearance of an excess of fat in the dejecta.

TREATMENT.

Entirely symptomatic. Morphine must be administered for the relief of pain, and digestives, as bismuth, pepsin, etc., given.

Chronic interstitial pancreatitis is best treated by minute doses of corrosive sublimate, iodide of potassium, and other remedies of a similar nature.

Cysts of the Pancreas.

Describe cysts of the pancreas.

Cysts of the pancreas are usually due to chronic interstitial pancreatitis. Anything causing obstruction of the duct, as a calculus or the pressure of a tumor, will give rise to cystic degeneration of the whole gland.

Cancer of the Pancreas.

Describe cancer of the pancreas.

Scirrhus is the most common variety of cancer of the pancreas. Medullary and colloid cancers are also met with.

The head of the pancreas is the most frequent seat of the disease. It occurs more frequently among males than females, and is more common under forty years of age.

Pain is the usual *symptom.* It is persistent, radiating, and increases rather than diminishes in intensity. It is somewhat relieved by bending the body forwards. Progressive and rapid emaciation sets in. The patient rapidly loses strength.

A tumor is found in about one-third of the cases. The lymphatic glands are also enlarged. There is an excess of fat in the stools, and cancerous cachexia is rapidly developed.

Jaundice is sometimes present from secondary affection of the liver, or from pressure on the common choledoch duct.

The disease is rapidly fatal; the case rarely lasts over a year.

The treatment is palliative.

(B.) DISEASES OF THE SPLEEN.

Acute Affections of the Spleen.

Describe acute affections of the spleen.

Acute affections of the spleen are usually miscalled *acute splenic tumors.* They are generally secondary to some cachexia; for example, they are found in malaria, typhoid fever and diphtheria; there is some enlargement also in scarlet fever, measles and smallpox; so that in general terms the spleen may be said to be enlarged temporarily in all acute affections. In acute affections of the liver also, from interference with the portal circulation, and particularly in acute yellow atrophy, the spleen enlarges.

What are the physical signs of enlarged spleen?

The enlargement is recognized by percussion; normally the splenic dullness extends downwards in the axillary line from the sixth or seventh rib almost to the border of the last rib. If the dullness extends below this the spleen is enlarged. In all acute affections of the spleen the enlargement extends downwards and forwards and the hilum of the spleen can often be detected on palpation.

What are the symptoms?

When it occurs secondary to general maladies there are no symptoms except enlargement, which, however, may be due to local causes, as congestion, inflammation, or abscess of the spleen.

Splenic Hyperæmia.

This may occur after traumatism, from blows and injuries, or after too violent exercise, particularly exercise following upon a hearty meal.

Symptoms.

Steady pain in the region of the spleen and shortness of breath; sometimes rupture of the vessels and fatal hemorrhage takes place.

Acute Splenitis.

This is sometimes called *Splenic Fever*. There is enlargement of the spleen, pain and tenderness in the splenic region, irritative cough, shortness of breath and fever; sometimes it results in a splenic or in diaphragmatic abscess.

Splenic Abscess.

There is a tumor which fluctuates, preceded by the symptoms of splenitis and attended with hectic fever. There is sometimes delirium, and nausea and vomiting are common symptoms; death usually results from rupture of the abscess. The abscesses are generally metastatic and are often found in connection with emboli resulting from endocarditis.

How should acute affections of the spleen be treated?

In all cases of acute splenic disease, if seen early, leeches should be employed and prompt purgation by means of salines, of podo-

phyllin or of colocynth practiced; later cold may be applied externally and ergot given to contract the vessels.

If an abscess form the treatment is symptomatic. The pus should be evacuated by aspiration, the patient's strength sustained by all possible means, and quinine given for the hectic fever.

Chronic Diseases of the Spleen.

Describe chronic diseases of the spleen.

Large splenic tumors are seen following malaria or in leukæmia, or in amyloid degeneration of the spleen (in which case the liver and kidneys are also affected). Less frequently large splenic tumors are found as the result of cancer (rare) or syphilis.

The spleen is often full of pigment, and after a time there is an increase in the connective and fibrous tissues, causing a permanent enlargement. A large swelling takes place; the tumor projecting sometimes towards the crest of the ileum, and even as far forward as the pubis. In all these affections there is marked anæmia, and dropsy, partly from anæmia and partly from the pressure produced by the tumor. Hemorrhage from the bowels is a frequent symptom of chronic splenic enlargement.

TREATMENT.

Keep up the patient's health and nutrition by all possible means.

Give the different preparations of iron for the anæmia.

Give quinine in small doses continued for long periods of time, with occasional short intermissions.

To reduce the splenic enlargement, ergot may be employed hypodermically two or three times a week, or an ointment of the red iodide of mercury may be thoroughly rubbed in over the tumor before a hot fire; the former gives the best result.

(C.) DISEASES OF THE KIDNEYS.

Acute and Chronic Renal Hyperæmia.

What is acute renal hyperæmia?

It is an acute or active congestion of the kidneys, caused by traumatism, exposure, or renal irritants, such as copaiba, sandalwood, turpentine, cantharides, etc.

SYMPTOMS.

The urine is passed frequently in small amounts, is high-colored, and is not associated with pain, which serves to distinguish it from inflammation of the kidney. It contains a little blood and traces of albumen, due to the presence of blood. There is a dull pain in the back, little or no fever. Sympathetic vomiting, headache and feeling of general discomfort.

What is chronic renal hyperæmia?

It is a chronic congestion of the kidney, usually dependent upon cardiac disease, especially on dilatation of the heart, with disease of the mitral valve. *The symptoms* are similar to those seen in acute congestion, but are more chronic, and associated with disease of the heart. The urine is scanty, high-colored and concentrated, and contains an abundant deposit of urates. There is a feeling of weight in the loins and dragging pain in the testicles.

What is the prognosis of these diseases?

The prognosis of *acute hyperæmia* is always favorable. That of *chronic hyperæmia* is favorable if it is not too long continued, and is not associated with very advanced cardiac disease.

How should these diseases be treated?

Dry cups should be applied over the loins. Hot baths should be employed to stimulate the skin and kidneys. Infusion of digitalis is the best diuretic that can be used. Alkaline waters (as Saratoga

or Vichy water) should be given, or pure water may be used, to dilute the urine. Saline purgatives should be occasionally administered. No stimulating diuretics should be employed.

Acute Parenchymatous Nephritis.

What is acute parenchymatous nephritis?

Acute parenchymatous nephritis, also called *acute tubal nephritis* and *acute Bright's disease*, is an inflammation of the secreting structure of the kidney, attended with scanty, high-colored urine, of high specific gravity, and containing blood and epithelial casts.

What are its causes?

The most frequent cause is scarlet fever; it also follows other exanthemata; is supposed to be due to exposure, or to traumatism, and occasionally follows congestion of the kidneys. Both kidneys are always affected.

What are the symptoms?

The urine is dark, smoky (from admixture with blood), scanty, and of high specific gravity. The microscope shows some free blood corpuscles, casts of epithelium, with blood corpuscles in the casts, and some free epithelial cells, from the tubules of the kidney. Chemical tests show a large amount of albumen. There is moderate fever, some thirst, nausea, vomiting and diarrhœa.

Symptoms due to uræmia are often present, as stupor, headache, and occasionally, not often, convulsions.

After the inflammation has lasted some little time, the blood begins to disappear from the urine, and general dropsy may suddenly supervene.

What is the morbid anatomy of this affection?

The cortical substance of the kidney is swollen and congested, and encroaches on the tubules and pyramids, which become red and congested likewise. Small capillary hemorrhages take place into the pelvis of the kidney.

Under the microscope the Malpighian bodies are found to be turgid with blood; the tubules are full of blood corpuscles, epithelium and embryonic cells. This condition lasts for about ten days.

The redness slowly disappears, leaving the tubules swollen and congested in patches for several weeks. The epithelial casts are due to the distention of the tubules with a fibrinous deposit, and the blood corpuscles, which are washed out by the urine, carrying with it the old epithelial cells.

What is the diagnosis and prognosis?

DIAGNOSIS.

The disease is diagnosed by the history; the cause; by affecting young adults especially; by the appearance of the urine, the presence of blood and blood-casts seen under the microscope.

PROGNOSIS.

These cases usually recover. Sometimes serous effusions take place, as into the pericardium or pleura, or into the ventricles of the brain, which prove rapidly fatal.

What is the treatment?

Absolute rest in bed must be insisted upon. The diet must be limited to milk, no stimulating diuretics should be employed, but the kidneys should be kept acting by means of fluids, especially water. *If the patient is strong*, and it is due to a cold, wet cups should be applied over the loins. *If the patient is weak*, and it follows a debilitating disease, dry cups should be employed, followed by the application of a counter-irritant, as croton oil.

Digitalis, either in the form of the infusion (f℥j–ij every few hours, watching its effect upon the heart), or of the tincture (gtt. iij–x), or of the fluid extract (gtt. j–iij) should be administered. *Cream of tartar* in ℥ss doses, or potassium citrate in gr. xx doses, are excellent adjuvants to the digitalis.

If the secretion of urine is small, the patient dull, the temperature 103° (threatening uræmia), particularly if he has been previously cupped, pilocarpine (gr. $\frac{1}{8}$) should be given hypodermically or the fluid extract of jaborandi (gtt. x–xx) administered internally, provided the heart is sufficiently strong to withstand their depressing effect.

Active purgation with podophyllin, croton oil or elaterium, should also be employed.

Should symptoms of uræmia be actually present, active purgation should be resorted to, and free diaphoresis procured by the hot bath, the wet pack, the hot-air bath, or, if these means fail, by jaborandi or pilocarpine.

If uræmic convulsions occur, the inhalation of chloroform may be employed to stop the convulsions, and chloral and bromide of potassium given to prevent their recurrence.

Uræmic asthma is best treated by the inhalation of a few drops of amyl nitrite, followed by the administration of nitroglycerin. To neutralize the urea in the blood, benzoic acid may be given if the stomach will bear it, or hydrochloric acid or nitro-hydrochloric acid may be substituted if the former produces vomiting.

Chronic Parenchymatous Nephritis.

What is this affection?

This disease, also called *chronic Bright's disease*, and, in its later stages, *large white kidney*, or *fatty kidney*, is a chronic inflammation of the secreting structure of the kidney, characterized by a scanty, high-colored urine of high specific gravity, containing much albumen with granular or fatty casts; dropsy and various disorders of the nervous, circulatory and gastro-intestinal systems are present, due to the circulation of urea in the blood.

What is its cause and morbid anatomy?

CAUSATION.

It occasionally follows the acute disease; sometimes occurs without known cause; is frequently due to continued exposure to cold, and at times to chronic digestive disturbances associated with mental worry.

MORBID ANATOMY.

The kidney is large; is yellow or white on section, the tubules appearing red and congested on the cut surface. The cortex is swollen, thickened, infiltrated, and the capsule easily separated. If this condition alone exists, gradual absorption of the exudate may take place and the diseased kidney recover. Sometimes, however, the disease progresses, the epithelium undergoing fatty degeneration and the case terminating fatally. The tubules are filled with

granular epithelium, which is shriveled and detached. A fibrinous exudation takes place under this epithelium, forming casts of the tubules, which, when washed away, carry the granular epithelium with them. Should fatty degeneration occur these casts will contain epithelial cells full of oil-globules.

What are the symptoms?

The urine is generally scanty and of high specific gravity. Occasionally the amount passed is normal, and the specific gravity low or normal. It does not contain blood, sometimes a few corpuscles may be seen, but granular casts are found containing shrivelled granular epithelium, and as a late symptom fatty casts.

Dropsy is often a prominent symptom ; commencing in the eyelids it invades the face, hands, legs, and later, as a serous effusion, it occurs in the internal cavities, as the peritoneum, pleura, or pericardium.

Progressing anæmia, due to the drain of albumen from the blood, is a frequent symptom, which, together with œdema of the face, gives rise to a characteristic expression.

Gastro-intestinal disturbance, as vomiting and occasionally diarrhœa, is due to the attempts of the mucous membrane to eliminate the urea from the system.

Chronic bronchial catarrhs occur from the same cause.

Nervous symptoms, as headache, impaired memory, irritable temper, drowsiness and defective eyesight, are frequently complained of.

The ophthalmoscope often shows a low grade of retinitis leading to atrophy of the optic nerve and blindness.

Cardiac Symptoms.—There is at first functional disturbance of the heart ; afterwards hypertrophy takes place, associated, at a later stage, with dilatation.

What is the diagnosis of this form of Bright's disease?

The disease is recognized by the dropsy, the large amount of albumen, the scanty urine of high specific gravity, the casts, the secondary effects, and the absence of fever.

What is the prognosis?

The prognosis is hopeful. It is bad if there are marked cardiac

complications, or if the casts contain very granular or very fatty epithelium.

How should it be treated?

DIETETIC TREATMENT.

If the patient can live on a strict diet of skimmed milk, which he usually can if he has no work to do, the result will be most favorable. If this cannot be done, skimmed milk with eggs and easily-digested meat, but no wine or liquor, may be given. If the patient is much run down, or if he be a weak child, a small amount of port wine may be taken with the meals. No food should be given which will throw work upon the kidneys.

MEDICAL TREATMENT.

Tincture of the ferric chloride (gtt. xv–xx, *t. i. d*), particularly in the form of Basham's mixture, is the best remedy. This is especially of service where the urine is scanty, in which case potassium acetate may be substituted for the spirits of mindererus. When the iron does not act, ergot may be given, or the chloride of gold and sodium employed.

For dropsy, purgatives should be administered, as compound jalap powders, Rochelle salts, podophyllin or elaterin. Diaphoretics, as vapor baths or minute doses of Dover's powder, with nitrate of potassium (āā gr. iij, frequently repeated) or pilocarpine, where its depressing effect upon the heart is not feared, are of great service in this condition.

For uræmia active purgation, pilocarpine, vapor baths and small doses of benzoic or nitrohydrochloric acid should be administered, as in acute Bright's disease.

During uræmic convulsions chloroform may be inhaled, and chloral and bromide of potassium given, as in acute Bright's disease.

For pericarditis potassium iodide, purgatives, stimulants and iron should be given. Opium must be administered very cautiously, if at all.

For pleuritic effusions the same treatment is indicated.

Chronic Interstitial Nephritis.

What are the synonyms, causes and duration of this disease?

SYNONYMS.

It is also known as small, granular kidney; gouty kidney; cirrhosis of the kidney, etc.

CAUSATION.

It almost invariably begins as a chronic disease, rarely, if ever, following other affections of the kidney. It is due to the abuse of alcohol, syphilis, cold and exposure, gout, overwork, mental worry, or chronic lead poisoning, and usually occurs after fifty years of age.

DURATION.

It is a very chronic affection, life being prolonged for years if proper care is taken.

What are its symptoms?

The symptoms are very obscure, being referable to almost every other organ of the body except the kidney; thus the patient suffers from dyspepsia, headache, chilliness, defective eyesight, palpitation, which is always seen in cases of long duration, and local palsies. There is no dropsy, except late in the disease, due to dilated heart. The urine is passed in large amounts, has a low specific gravity, and contains a very small amount of albumen, which may be detected by the nitric or picric acid tests, or by potassium ferrocyanide. The presence of albumen is variable, and several specimens may have to be examined before it is discovered. Sooner or later small granular or hyaline casts are found.

What is the pathology?

The kidney is small, contracted, and dense. There is very little cortical substance. The Malpighian bodies are atrophied, from pressure, and there is a proliferation of the connective tissues, at first in the cortical substance and then between the tubules, causing more or less general atrophy of these structures. The changes are very gradual.

What is the prognosis?

It is a disease of long duration. *Degeneration of the vessels* may

take place and the patient die of apoplexy. *Cardiac hypertrophy* results in long standing cases and sometimes causes death.

Uræmic convulsions or coma may come on rapidly at any time and terminate fatally.

Hopeless blindness, from atrophy of the optic nerve, may develop.

How is it treated ?

The diet should be bland and easily digested, consisting of milk, eggs and meat. Milk is not as successful, however, as in the kidney diseases already discussed. Potassium or sodium iodide should be given where there is not much dropsy. Small doses of corrosive sublimate are supposed to retard the development of the connective tissue. Syrup of the ferrous iodide may be alternated with either of the above remedies, and counter-irritants over the kidneys (as applications of croton oil) may be used to advantage.

Nitro-glycerin internally also does good. Iron is not as efficacious as in the other kidney diseases.

Albuminoid Kidney.

Describe the albuminoid kidney.

SYNONYMS.

It is also called waxy, amyloid, or lardaceous kidney.

CAUSATION.

It is caused by wasting diseases, particularly long continued suppurations and bone diseases ; also by syphilis.

SYMPTOMS.

A large amount of urine is passed which has a low specific gravity and contains much albumen and waxy casts, which are large, transparent, and devoid of epithelium. Dropsy is a late symptom. The liver and spleen are usually enlarged, having undergone a similar degeneration, and anæmia is present.

POST-MORTEM EXAMINATION.

The kidney is enlarged, smooth and pale on section, presenting a waxy appearance. A solution of iodine passed rapidly over the surface causes a mahogany-brown discoloration. The disease depends

upon an interstitial deposit of an albuminoid or waxy material. *The spleen and liver* are similarly affected.

DIAGNOSIS.

The disease is recognized by the history, cause, chemical and microscopical examination of the urine, and the marked anæmia, with the enlargement of the liver and spleen.

TREATMENT.

The treatment consists in regulating the diet, giving mostly albuminous food and making the patient indulge in active exercise. An occasional saline purgative is advantageous, and the administration of chloride of ammonium (giving gr. x, increased to gr. xx *t. i. d.* for periods of three weeks, or until it produces gastro-intestinal irritation, when it should be replaced by the syrup of ferrous iodide in doses of gtt. xx–℥j) is the best treatment. The iodide of ammonium may also be used, or cod-liver oil may be conjoined with other treatment. The cause of the affection should be borne in mind and treated.

Cystic Degeneration of the Kidney.

Describe this affection.

CAUSATION.

Cystic kidney, or hydatid cyst of the kidney, is sometimes seen in chronic Bright's disease, especially in contracted kidney. It sometimes occurs from echinococci (similar to hydatid of the liver). Cystic degeneration may be due to impaction of a renal calculus. It often occurs without any apparent cause, in middle or early life. Both kidneys are usually affected.

SYMPTOMS.

The first symptom is usually frequent urination, the urine sometimes containing blood with very little albumen, except when much blood is present. There is no sugar, and only rarely are there a few tube casts. The patient's strength gradually fails, the kidneys reach a large size, bulging out the abdominal wall. They are felt through the integuments as soft, slightly resisting bodies.

PROGNOSIS.

This depends upon the size of the tumor and the amount of kidney involved.

Death may occur from exhaustion or from uræmia. It is a very chronic disease.

TREATMENT.

Treatment does not do much good. Iron has been given and slight amounts of stimulants with good food to keep up the strength of the patient. Any symptoms which may call for treatment should be promptly met.

Passage of Renal Calculi.

Describe the symptoms, diagnosis, and treatment of the passage of renal calculi.

The portions passed are usually but a part of a larger calculus in the kidney.

SYMPTOMS.

The symptoms are intense pains in the loins, shooting down the ureters and finally in the head of the penis. The pain is intense and is followed after half an hour to an hour by a lull, and after a time by a fresh accession of pain.

As a rule the whole attack lasts from four to six hours. There is much soreness and inability to move during the paroxysm, the testicle is retracted and in women there is a soreness over the ovary.

During the paroxysm there is much irritability of the stomach, and often actual vomiting. The action of the heart is rapid, extremities cold, skin clammy, but little urine is passed and that comes away drop by drop, is very high-colored, frequently contains blood and gives rise to scalding pain in the urethra. Sometimes these paroxysms of pain continue for hours followed by a lull which may last several days.

DIAGNOSIS.

(1) *From malarial congestion of the kidney with intense pain.* In this affection there is a malarial history, and the symptoms of malaria, high temperature, a marked periodicity in its recurrence, and it is materially benefited by quinine.

(2) *From intestinal neuralgia* it is diagnosed by the absence in the latter of any signs of urinary irritation or of blood in the urine.

PROGNOSIS.

The prognosis of the passage is favorable.

Usually after the attack has lasted for a longer or a shorter period the calculi is passed into the bladder and the pain ceases. Occasionally it causes ulceration of the ureter followed by peritonitis.

TREATMENT.

Morphine (gr. $\frac{1}{4}$) combined with atropine (gr. $\frac{1}{80}$) should be promptly administered hypodermically, and if in half an hour some relief is not obtained, opium may be given by the rectum.

If the pain is very severe, inhalations of ether may be used between injections.

Large fomentations should be applied over the ureter and kidney, or draughts of hot water or of alkaline waters should be taken. If the calculus is long in passing, the fluid extract of belladonna (gtt. ij omn. hor.) may be employed until its physiological effects are obtained.

Formation of Renal Calculi.

What are the causes favoring the formation of renal calculi?

A little plug of mucus in the pelvis of the kidney often forms a nucleus for a calculus, and the abundance of any of the salts in the urine, as seen in uric acid or phosphatic diathesis, acts as a predisposing cause. Uric acid crystals may form in the tubules of the kidney, and growing in size cause atrophy of the surrounding structure, or by irritation lead to abscess.

What are the principal varieties of renal calculi?

(1) *The uric acid calculi*, the most frequent, are found in persons subject to acute lithiasis and torpidity of the liver.

(2) *Phosphatic calculi* usually consist of alternate layers of phosphates and urates.

(3) *The so-called mulberry calculi*, consisting of oxalate of lime. They are small, jagged, and give rise to much pain.

The number of renal calculi varies. Usually there are several small ones, occasionally one large one.

What are the symptoms?

When the calculus is in the pelvis of the kidney, symptoms of pyelitis will be present; when located in the kidney's structure there will be very little pus in the urine, which will only be a temporary symptom.

There is pain in the loins on motion or jarring reflected to the testicle and glans penis. Nausea and gastric disturbance is frequent. The urine, especially after a fall or jar, will contain blood for several days at a time, disappearing and appearing at irregular intervals. Under the microscope the urine contains a large amount of the crystals of the substance of which the calculus is composed.

If during the passage of the calculus the urine is nearly healthy, it shows the calculi are in one kidney only.

What is the prognosis?

The prognosis is favorable as a rule. Ulceration does not often occur. Sometimes the kidney becomes disorganized, undergoing cystic degeneration.

What is the treatment?

Examine the urine to determine, if possible, the composition of the calculus. *If it is uric acid*, the amount of nitrogenous food should be limited, the patient living principally on milk and vegetables, very little acid of any kind, drinking large amounts of water to keep the kidneys freely washed out. He should also take active exercise, make free use of baths, and take an occasional laxative. Carlsbad water or Carlsbad salts are of service. The alkalies act not only as diuretics, but they render the uric acid more soluble. For this purpose the citrate or carbonate of potassium may be employed, and, best of all, the citrate of lithium used.

If it is a phosphatic calculus the mineral acids, as nitric or nitrohydrochloric in gtt. iij–v doses, freely diluted with water, are of advantage. Benzoic acid or ammonium benzoate will very rapidly render the urine acid.

These calculi are more frequently found in the pelvis than in the kidney proper.

If it is an oxalic calculus, a long course of nitrohydrochloric acid is supposed to give good results.

This variety of calculi gives rise to the most pain and is the hardest of all to prevent.

No matter what the variety of calculus, the kidneys should always be kept acting freely by the use of water, milk and buttermilk, if the urine is not too acid.

If one kidney is hopelessly destroyed, the other being healthy, or if pyelitis is present with symptoms of septicæmia, the kidney may be aspirated, and, if stone is present, it may be cut down upon and removed; a drainage-tube being inserted into the wound and thorough antisepsis practiced.

Pyelitis.

What is pyelitis?

Pyelitis is suppuration connected with the kidney.

It may be a suppurative catarrh of the pelvis or kidney, or an abscess in its tubular structure.

What are its causes?

It may arise from catarrhal inflammation due to cold or exposure from rheumatism; from certain irritant substances, as cantharides or turpentine; from debilitating diseases, as diphtheria or typhoid fever; from renal calculus, when it is called calculus-pyelitis; or from inflammation extending upward from the bladder, as following chronic cystitis, or occasionally gonorrhœa.

What are the symptoms of pyelitis?

There is frequent urination without pain, dull aching over the loins, the urine contains much pus, and columnar epithelial cells from the pelvis of the kidney, and the reaction is generally acid, unless the bladder is also diseased. The pus cells are distinct, having large, well-marked nuclei.

If the disease is limited to the pelvis of the kidney, there will be but slight fever, not much albumen, and no tube casts. If the structure of the kidney is also involved, a large amount of albumen will be present, tube casts will be found in the urine; there will be hectic fever and grave constitutional disturbance.

How is this disease recognized?

Pyelitis is apt to be confounded with inflammation of the bladder,

but in the latter affection the urine is generally alkaline, the epithelial cells are squamous, not columnar, and there is pain in the suprapubic region upon urination.

Calculus-pyelitis is diagnosed by the history of attacks of the passage of calculi, and by bloody urine.

What is the treatment of pyelitis?

The urine should be diluted and the kidneys kept washed out by means of water, particularly light alkaline waters; or, if the urine is acid, by a neutral mixture. The diet should be light, nourishing, but unstimulating, consisting of milk, eggs, and a very little meat.

Numberless remedies have been used in this disease, as tar-water, oil of sandal wood, cubebs, copaiba, etc., which, however, are apt to disagree with the stomach; turpentine occasionally does good. Eucalyptus is more frequently of service, as is carbolic acid (gtt. ss), or salol, which will usually diminish the amount of pus. Tincture of the chloride of iron and cod-liver oil as a nutrient when the stomach will bear it, are useful. Benzoic acid, or the benzoates, are of advantage, particularly if cystitis complicates the disease.

If these means fail, or if the symptoms are urgent, an exploratory incision should be made, the abscess thoroughly drained, or the kidney removed.

Hydronephrosis.

What is hydronephrosis?

It is dilatation of the pelvis and of the calyces of the kidney, accompanied with an atrophy of its substance, resulting from some obstruction, and the consequent accumulation of non-purulent fluids.

What are its causes?

It may be produced by the impaction of a calculus, by the pressure of abdominal or pelvic tumors upon the ureter, by an enlarged prostate, urethral stricture, etc.

Sometimes it results from twisting of the ureter itself or from some congenital malformation of the ureter or kidney, and, occasionally, from tubercle or cancer of the kidney.

When the cause is slow in operating and the occlusion is partial or occurs gradually, the dilatation may be enormous, and the tumor attain so great a size as to be mistaken for ascites.

What are the symptoms of this condition?

When of small size it may not be discovered. When of moderate size, it is readily detected on abdominal examination as a fluctuating tumor occupying the renal region. When the tumor is large, it may easily be mistaken for other fluctuating tumors of the abdomen, and frequently can only be differentiated from them by a careful exploration of the other abdominal and of the pelvic organs, or by the examination of the fluid obtained by aspiration, which will usually be found to contain albumin, with urinary salts and epithelial cells, often similar to those of the pelvis of the kidney.

Sometimes, as when the hydronephrosis is caused by a twist of the ureter, as may occur in "floating kidney," the tumor suddenly disappears, with the discharge of a large quantity of fluid from the bladder. In these cases the fluid may or may not reaccumulate.

What is the prognosis of hydronephrosis?

The prognosis depends largely upon the cause. Sometimes the fluid is evacuated spontaneously through the ureter, and never reaccumulates: at other times the cyst may rupture into the peritoneum. Uræmia sometimes occurs, or suppuration may take place and the disease become pyelitis. When it is congenital death usually ensues rapidly.

How should hydronephrosis be treated?

The treatment is entirely surgical, and consists in aspiration or incision and drainage of the cyst, or even of removal of the kidney.

Hæmaturia.

What is hæmaturia?

This name is applied to the presence of blood in the urine. It is a symptom of many diseases rather than a disease itself.

What are its causes?

The principal cause is the presence of a renal calculus. Other

causes are, acute Bright's disease; scurvy or purpura; parasites in the kidney, which, however, very rarely occurs in this country, and which is seen particularly in the Mauritius; and malignant growths, as cancer, cysts, etc.

What are the symptoms and diagnosis of this condition?

SYMPTOMS.

The only symptom is the presence of blood in the urine; the two being intermixed, giving rise to a dark, smoky appearance.

DIAGNOSIS.

Blood is found in the urine; it may come from the kidney, bladder, or urethra.

When it comes from the kidney it is intimately mixed with the urine, giving rise to a smoky appearance; the urine contains more albumen, and the blood corpuscles are round, full and well developed.

When it comes from the bladder it is not intimately mingled with the urine. Little clots are passed; the urine is passed more frequently, and is accompanied with vesical tenesmus; the blood is sometimes present, sometimes not. The microscope shows small, shrivelled, broken-up cells, and a few drops of pure blood are apt to follow the passage of the water.

When it comes from the urethra it is accompanied by scalding or burning at the beginning of the act of urination, and the passage of the urine is apt to be preceded by a few drops of blood.

What is the prognosis and treatment?

PROGNOSIS.

The prognosis is generally favorable. The patient does not die of the hemorrhage itself, though he may of the disease of which it is a symptom.

TREATMENT.

The treatment depends upon the disease which causes it. The general treatment for hæmaturia consists in the administration of gallic acid, ten grains, increased to twenty if the stomach will tolerate it, every hour or two until some effects are observed. Sulphuric acid may be given, if it is not contra-indicated, alone or alternating

with the gallic acid. The fluid extract of ergot (gtt. xx–f3j), may be administered every hour or two. The patient should have plenty of water to keep the kidneys active and wash out the accumulating blood, and should be placed absolutely at rest.

Tubercular Disease of the Kidney.

Describe this disease.

This affection, which is not very common, is apt to co-exist with pulmonary tuberculosis, and seems to be hereditary.

PATHOLOGY.

A deposition of tubercle takes place in the cortex of the kidney, the deposits run together, and break down. Similar deposits occur in the pelvis and in the ureter, which undergo tubercular ulceration. Gradually the whole mass of the kidney becomes degenerated. Both kidneys are involved, though not equally.

SYMPTOMS.

There is marked pain, more severe than in cancer of the kidney, due to ulceration of the pelvis and ureter, and probably, also, to the small masses of tissues which are softening and being voided. There is no tumor. The water dribbles. Small amounts of bloody urine, sometimes containing broken-down tissue, are passed; there are marked sweats and sometimes other hectic symptoms, the patient either dying worn out from the pain, from uræmia, or from septicæmia.

This disease runs an acute course.

DIAGNOSIS.

It is recognized by the pain, which resembles that caused by the passage of calculus; by the family history; by the hectic; by the character of the urine, which probably contains the bacillus tuberculosis, by the absence of a tumor, and by the production of tuberculosis in rabbits by the inoculation of sediments of the urine.

PROGNOSIS.

Fatal.

TREATMENT.

The treatment is entirely symptomatic. Opium should be given for the pain, and the kidneys should be freely washed out by large amounts of diluents.

Cancer of the Kidney.

Describe cancer of the kidney.

It occurs as a primary or secondary affection, usually in childhood or old age. Medullary carcinoma and scirrhus are the most common forms.

The kidney is usually much enlarged and irregular in shape; cancer elements are found invading the bowels and intertubular structures, and frequently involving the pelvis and ureter.

Cancer elements may even involve the renal vein.

SYMPTOMS.

There is pain of a greater or less degree in the lumbar region; hæmaturia is a common symptom early in the case. The kidney is much enlarged, forming a tumor in the abdomen which is easily recognized.

Albumen is sometimes present in the urine, as is the debris of broken-down tissue. Characteristic cachexia develops later in the disease. One kidney alone is generally involved.

PROGNOSIS.

It is generally fatal. The disease runs a rapid course, especially in children.

DIAGNOSIS.

The diagnosis is made by the cachexia, the tumor, the bloody urine, the pain and the occurrence especially in children.

TREATMENT.

It is merely symptomatic. The endeavor should be made to sustain the patient's strength as long as possible.

Perinephritis.

Describe this affection.

This is an inflammation of the areolar tissue surrounding the kidney.

It is a common disease, especially seen in childhood, particularly among boys. The right side is most frequently affected.

CAUSE.

It is usually due to traumatism, as blows, sprains, or too much exercise, but occasionally follows low states of the system, as typhoid fever or diphtheria.

PATHOLOGY.

This is the same as that of ordinary inflammation, usually terminating in suppuration, the abscess bursting externally in the loin or perhaps into the peritoneal cavity.

SYMPTOMS.

There is pain and tenderness in the loin of one side. Pain is increased by motion; there is a feeling of dull weight, a fullness rather than swelling over the situation of the kidney, with fluctuation, some irritative fever, and although the urine is almost invariably healthy, occasionally it contains a trace of albumen from pressure.

It is a long disease.

PROGNOSIS.

It is generally favorable.

TREATMENT.

Absolute rest, easily digested food in the early stages, frictions with liniments, or counter-irritation over the seat of the inflammation, or if seen early, the application of ice. Later in the disease poultices afford much relief.

Quinine should be given in decided doses throughout the disease, and the pus should be evacuated as soon as it is formed, either by the knife or by aspiration.

Floating Kidney.

What is this affection?

Movable or floating kidney is one which through the loosening of its attachments becomes abnormally movable. It is due to blows, sprains, or other injuries, which loosen its attachments. It occurs usually in weak anæmic patients or in debilitating diseases.

It is more frequent in women than in men, and the right kidney is usually the one affected.

SYMPTOMS.

A tumor is felt under the ribs or forward against the abdominal wall; it is tender to the touch; the outline is found to be kidney-shaped; is movable, and the patient generally enjoys good health.

Sometimes grave symptoms will appear for a few hours, such as chill, nausea, and vomiting, intense anxiety, and interrupted flow of urine.

These symptoms are probably due to the kidney and ureter becoming twisted and stopping the passage of water.

DIAGNOSIS.

The diagnosis is easy. The shape and position of the tumor, which can be pushed back in position, and the absence of the kidney in its normal situation, are sufficient to distinguish the disease.

PROGNOSIS.

No one dies of it. It is only dangerous when adhesions form, fixing the kidney in some abnormal position, and then the danger is from pressure.

TREATMENT.

Replace the kidney and have the patient wear an abdominal bandage. Improve the general condition by tonics. For the symptoms of collapse, should they supervene, rest, stimulants, etc., form the best means of treatment.

(D.) DISEASES OF THE RESPIRATORY ORGANS.

Physical Diagnosis.

What is physical diagnosis?

Physical diagnosis is the art of discovering a disease by means of the sight, touch, hearing, or a combination of these senses.

What are the methods employed to gain this result?

The methods employed are *inspection*, *palpation*, *mensuration*, *percussion* and *auscultation*.

Inspection.

What is inspection?

Inspection is the act of carefully examining a person or thing by means of the sense of sight. As applied to diseases of the chest it enables the examiner to determine the *size*, *form*, *color* and *movements* of the parts. By carefully comparing the movements of the two sides of the chest he can arrive at an idea regarding the *relative action of the two lungs*. He can also see whether the patient is *breathing freely*, *deeply* or *superficially*. He can observe any *bulging* or *obliteration of the intercostal spaces* on the one hand or *any depression* of them on the other. He can also observe the *apex beat of the heart*, see whether it is *diffused* or *localized*, and whether it is *strong* or *feeble*. By means of inspection also he may arrive at a conclusion respecting the performance of the nutritive functions of the patient.

Palpation.

What is palpation?

Palpation is the application of the hands and fingers to the surface of the patient's body, to *correct the impressions* arrived at by inspection, to judge of the *enlargement* or *displacement of various organs*, to locate *spots of tenderness*, to determine the *consistence and attachments of tumors*, and the *frequency of breathing*, or the *strength*, *force*, *diffusion* and *frequency of the apex beat of the heart*.

The existence and character of various vibrations, spoken of as *fremitus*, produced naturally, or by a state of disease, are also determined by palpation.

Vocal fremitus is a vibration produced normally in the chest wall by the act of speaking or of crying.

Rhoncal fremitus is the vibration produced by râles in the chest.

Friction fremitus is produced by two roughened surfaces rubbing together, as in the dry stages of pleurisy. When both fluid and air are present in the cavity a distinct vibration is imparted through the walls by shaking the patient, and this is accompanied by a splashing sound, known as the *succussion sound.*

Vocal fremitus is normally felt over the entire chest wall, but is more distinct toward the right apex. Anything producing a more solid condition of the lung increases this vibration. Anything interposed between the lung and the examiner's hand, as a serous effusion into the cavity of the pleura, diminishes or prevents this vibration from being felt.

Mensuration.

What is mensuration?

Mensuration, as applied to the chest, usually consists in measuring with a tape the circumference of the thorax during the acts of inspiration and expiration. The measurement in the former should exceed that in the latter state by about three inches. Measurements may also be taken from the vertebral column to mid-sternum on either side, and thus an idea of the relative expansion of the lungs may be gained.

Percussion.

What is percussion?

Percussion is the sound produced by tapping lightly over the surface of the body, and as it differs according to the amount of air contained in the body, or, in other words, its solidity, the physician is enabled to judge of the composition of bodies by this means. Percussion may be *immediate* when the surface is struck directly without the use of any intervening medium, or mediate (which is now almost exclusively employed), when the physician strikes on an intervening body, as the fingers of the other hand, or a pleximeter, applied closely to the surface.

In percussing, one or two fingers only should be used to strike with, the movement taking place from the wrist, and being a light, even stroke. The fingers are the best instruments for this purpose. Where much percussion has to be performed, instruments are used to save the fingers; thus various forms of light hammers, usually on a flexible handle, are employed, and a pleximeter consisting of a small plate, made of hard rubber, wood, ivory or other substance, placed on the surface to be struck.

What is the normal sound elicited by percussion over a healthy lung?

When a healthy chest is percussed, a CLEAR SOUND is elicited, which is always of relatively *low pitch*, but which will vary in *intensity*, depending upon the force of the stroke and the thickness of the cutaneous, adipose and muscular coverings of the chest wall. The percussion note is normally clearer anteriorly and above, on the left side. A clear sound denotes a certain amount of air enclosed in elastic tissue. In percussing a chest, the corresponding portions of either side should be alternately percussed, and the sounds elicited carefully compared.

How is the normal percussion note altered by disease?

(1) The percussion note may be HYPER-RESONANT; that is, *the normal pulmonary resonance may be increased.* This note is produced in all cases in which there is a relatively increased proportion of air in the lung, provided there is no alteration in the tension of the chest; thus it is seen where from consolidation of one lung, the healthy lung is forced to do more than its ordinary share of work.

(2) *The normal resonance may be decreased* wherever there is a relatively diminished amount of air in the lungs. When it is slightly diminished, the resonance is said to be IMPAIRED; when much decreased, the note is said to be DULL; and where there is absolutely no resonance it is said to be FLAT. *The pitch of the note is heightened in proportion to the diminution in its resonance.* Any disease that consolidates or condenses the pulmonary tissue gives dullness on percussion. The more absolute the dullness, the more absolute is the exclusion of air.

(3) A TYMPANITIC SOUND is that produced on percussion by large quantities of air enclosed within walls which are yielding and elastic.

The typical tympanitic sound is produced by percussion over the intestines. This sound is not usually perceived over the chest, as the required conditions are absent, but should there be a large cavity in the lung, it may be elicited. If the natural tension of the lung is altered, as sometimes occurs in the upper part of the chest, when there is large pleuritic effusion below, or as also occurs when there is a distention of the air vesicles, a tympanitic sound may be produced. It is more ringing in character and of a *higher pitch* than the normal percussion note.

The tympanitic note is sometimes modified as follows :—

An Amphoric or Metallic sound is a concentrated tympanitic note produced by percussion over a cavity containing a large amount of air, and surrounded by walls which are only moderately tense, as is seen in cases of pulmonary consumption.

The Cracked-pot or Cracked-metal sound is produced when a cavity communicates directly with a bronchial tube and depends upon the rapid forcing out of the air from the former into the latter, caused by a strong, quick blow of the percussing finger, and best heard when the patient's mouth is open. In children it is occasionally heard without these conditions being present.

Respiratory Percussion.

What is respiratory percussion ?

Respiratory percussion consists in percussing the chest not only while the patient is breathing normally, but also during a deep inspiration and a forced expiration. The examiner is enabled to detect slight changes in the percussion note, which might otherwise escape his attention.

Auscultation.

What is auscultation ?

Auscultation is listening to the sounds produced within the chest during respiration, coughing, or speaking. For this purpose the ear may be applied directly to the chest wall (which is spoken of as *immediate auscultation*) or various media may be employed to conduct the sound to the ear (called *mediate auscultation.*) When mediate auscultation is employed, an instrument called the *stethoscope*, made

of wood, rubber, or gun-metal, is used. The ear-piece should be large enough to cover the ear, and may be made of the same or of a different material from the rest of the stethoscope. The stethoscope which is most generally preferred is made of gun-metal with an ear-piece of hard rubber. *Binaural stethoscopes* are also used. They are made upon the principle that the physician can hear better with two ears than he can with one. *Differential stethoscopes* have been invented, the examiner being supposed to hear and compare simultaneously the sounds in both lungs conducted to either ear by means of separate tubes. They are practically useless.

In examining the chest immediate auscultation is the better for diseases of the lungs, mediate for diseases of the heart.

What sounds are heard on auscultating the respiratory apparatus?

When the air is passing through the larynx, trachea or bronchi, it produces a high-pitched tubular sound, heard on both expiration and inspiration. This sound is, however, masked when the ear is applied over the normal lung, by the sound produced in the air vesicles (called the *vesicular murmur*). This is a soft, breezy, inspiratory sound, best heard at the upper part of the lung. It is almost indefinite in duration and is followed by a pause, followed in its turn by a short, scarcely-to-be-heard, expiratory sound. The vesicular murmur is most typical at the left apex anteriorly, the expiratory sound being heard best at the upper part of the chest posteriorly. The vesicular murmur is caused by the expansion of numerous air cells, the walls of which present a certain resistance to be overcome in the act of inspiration, thus producing the sound. As, however, on account of their elasticity, they not only offer resistance, but even aid in expiration, there is no sound produced by this act, or if there is any, it is almost inaudible.

Bronchial respiration, on the other hand, is produced in the bronchial tubes by the air passing through them, and is a blowing sound, similar to that produced by blowing through a tube, hence it is often called *tubal breathing*. Though this bronchial sound exists, it is not heard normally, being overpowered by the sound produced in the air cells. Bronchial breathing may be heard in a person under the influence of fright, because then the breathing is shallow, and the air

vesicles not being expanded, the bronchial sound is not masked. Pneumonia, or any disease which produces consolidation of the air vesicles, will obliterate the vesicular murmur over that portion of the chest, and allow the bronchial respiration to be distinguished.

What changes occur in these sounds as the result of disease?

(A) VESICULAR MURMUR.

This sound may be altered in *intensity*, in *rhythm* or in *character*.

(1) *Alteration in Intensity.*

(*a*) The *intensity may be increased*, and is then called *puerile breathing*, from its resemblance to the loud, strong breathing of a young child. This is generally seen when the lung of one side, or a portion of it, has more than the usual amount of work thrown upon it, by disease or compression of other portions of lung tissue.

(*b*) *Diminished respiration* is a lessening of the intensity of the normal vesicular murmur, and is often spoken of as *feeble breathing*. Anything which will prevent the air from thoroughly distending the air vesicles, will produce this change in the intensity of the vesicular murmur; thus, a plug of mucus in a bronchial tube will produce feeble respiration over that portion of the lung to which the tube and its branches lead. Emphysema, by lessening the elasticity of the lung and allowing the air cells to remain distended at the close of the expiratory act, causes feeble breathing. Intercostal neuralgia or rheumatism, or the pain of pleurisy, by interfering with the expansion of the chest, give rise to feeble breathing. An effusion, by mechanically compressing the air cells, will produce the same result.

(*c*) *The vesicular murmur may be absent or suppressed*, as when the lung is compressed by the presence of fluid or air in the pleural sac or in atelectasis.

(2) *Alteration in Rhythm.*

(*a*) *The rhythm may be interrupted or jerky*, in various spasmodic affections of the air tubes, and in the early stages of pulmonary phthisis, in which it is most marked at the left apex of the lung.

(*b*) *A change may occur in the length of expiration, relatively to inspiration;* thus expiration is prolonged in the early stages of consolidation of the lung, due to the diminution in the elasticity of the air cells caused by the inflammatory action. When expiration is prolonged at the apex of the lung it indicates beginning consoli-

dation from tubercular deposit. When the air cells lose their elasticity, as occurs in emphysema, expiration is also prolonged because a longer time is required to empty it.

(3) *Alteration in Character.*

(*a*) *The respiration may be harsh, or vesiculo-bronchial.*

This indicates that the bronchial sound is becoming manifest while the vesicular sound is disappearing, or *vice versa ;* it therefore shows imperfect consolidation. When heard at the apex of the lung it is usually a sign of commencing phthisis ; when heard over the body of the lung it indicates that the consolidation of pneumonia is either beginning or ending.

(B) Bronchial Breathing.—

This also has its varieties.

(*a*) *Simple bronchial breathing* is produced where there is entire consolidation of the air vesicles, so that the vesicular murmur is absent ; it therefore indicates consolidation, and is heard to perfection in the second stage of pneumonia.

(*b*) *Cavernous respiration* is a modification of bronchial breathing. It is heard over a cavity of moderate size. By cavernous respiration is meant a breath sound similar to bronchial breathing, but more hollow (cavernous) ; of lower pitch, softer, less harsh, and equal both in inspiration and expiration. It is often mingled with *gurgling*, a sound produced by fluids in the cavity. It is only heard in cavities of a certain size, and usually indicates the third stage of consumption, although it may be heard over pulmonary abscesses of any kind, or indeed over dilated portions of a bronchial tube.

(*c*) *Amphoric or metallic respiration* is a sound produced by the air entering a large cavity with firm walls. It is a soft, blowing sound, having a certain metallic ring, and can be imitated to some extent by blowing in the mouth of a jug.

What sounds are sometimes heard in the chest, as a result of disease, that are entirely foreign to those normally produced there ?

Adventitious sounds may be divided into *râles*, or *rhonchi*, and *friction sounds.*

(A) *Râles* are the vibrations produced either in the bronchial tubes

or in the air-vesicles by the air passing through fluids contained in those structures.

(1) *Bronchial râles* may be either dry or moist.

(*a*) *Dry râles* are produced by the air passing through thick fluids which are not broken up by it in its passage.

When these are produced in a large bronchial tube, they are low-pitched and to some extent musical. They are called *sonorous râles.* When however they occur in a small tube, the air whistles as it passes the mucus, and the resulting sound is high-pitched. These are called *sibilant râles.*

(*b*) *Moist râles* are produced in a fluid which is sufficiently thin to allow the air to break it into bubbles in its passage. When the vibration produced is the result of fluid in the larger tubes the bubbles will be large, and the râles are called *large, bubbling or mucous râles.* When they are formed in the smaller tubes fine bubbles are found, and the sound is spoken of as *small bubbling, mucous or sub-crepitant râles.*

(2) *Vesicular Râles.*

The crepitant râle is a sound produced in the air-vesicles and intercellular passages, or about the termination of the finer bronchioles. It is a fine sound, *heard only on inspiration* and somewhat resembling the sound made by throwing salt upon a hot fire, or by rubbing a lock of hair between the finger and thumb in front of the ear. It is a very fine, crackling sound. This is the râle of beginning exudation, and shows that a sticky fluid is present in the air-cells. They are specially heard in the first stage of pneumonia, disappearing when complete consolidation occurs, and reappearing again as the exudate is absorbed.

Crackling râles are modifications of the former. They are fine, dry sounds, especially heard at the apices of the lungs in the incipient stages of phthisis.

Hollow bubbling or gurgling râles, or cavernous râles as they are sometimes called, are heard over large cavities communicating freely with a bronchial tube, and are especially found in the latter stages of phthisis. Their names well describe them.

(B) Friction Sounds.

These sounds are produced when two roughened surfaces, as those of an inflamed pleura, are rubbed against each other. They

are said to sound like the creaking of sole-leather. The rustling of silk or the movements of the body against a chest-protector might be readily mistaken for friction sounds. Certain râles also might be confounded with them, but they are very superficial, uninfluenced by cough, and purely localized, which is not apt to be the case with râles.

What is vocal resonance?

Vocal resonance is the vibration produced upon speaking, when transmitted to the ear of the examiner placed against the patient's chest.

Anything intervening between the lung and the examiner's ear will diminish the vocal resonance or perhaps entirely prevent the transmission of the vibration. In this way when a pleuritic effusion or pneumothorax is present, vocal resonance is entirely absent. On the other hand it is exaggerated by any condition which produces solidification of the lung tissue, as in tuberculosis or pneumonia.

Bronchophony is a vocal resonance, not only of exaggerated intensity, but of elevated pitch, and shows complete consolidation of the lung tissue over which it is heard.

Pectoriloquy is an exaggerated bronchophony, the voice of the patient sounding near to the examining ear and the articulated words being distinct. It is found either where there is a cavity in the lung, or when consolidation is absolute and complete. *Whispering pectoriloquy* possesses the same characters as the foregoing, the slightest whisper of the patient being distinctly heard.

Ægophony is a modified bronchophony, the voice having a certain tremulous or bleating character. It is heard at the edge of a pleuritic effusion, where a thin layer of fluid intervenes between the lung and the examiner's ear. It is therefore a sign of pleurisy or of pleuro-pneumonia. *Amphoric or metallic voice* is heard under the same conditions which produce other amphoric or metallic phenomena. The auscultation of the voice is not of much practical importance, with the exception of its absence in pleuritic effusion and in the presence of bronchophony in consolidation.

These methods of investigating disease, when taken separately, are not of much value. It is by their association in any given case

that they are at all important. The following table, taken from Da Costa's "Medical Diagnosis," shows the manner in which they should be employed in the study of diseases of the respiratory organs, and their importance when properly combined :

Percussion.	Auscultation of Respiration.	Auscultation of Voice	Vocal Fremitus.	Physical Conditions.
Clear.	Vesicular murmur or its modification.	Normal vocal resonance.	Unimpaired.	Lung tissue healthy or nearly so; at any rate no increased density from deposits, etc.
Dull.	Bronchial or harsh respiration.	Bronchophony.	Increased.	Solidification of pulmonary structure.
	Absent respiration.	Absent voice.	Diminished or absent.	Effusion into pleural sac.
Tympanitic.	Cavernous or feeble, according to cause.	Uncertain; cavernous or diminished.	Uncertain; mostly diminished.	Increased quantity of air within the chest, due to cavity or to overdistention of the air-cells.
Amphoric or metallic.	Amphoric or metallic.	Amphoric or metallic.	Mostly diminished.	Large cavity with elastic walls.
Cracked metal sound.	Cavernous respiration.	Cavernous respiration.	Uncertain.	Generally a cavity communicating with a bronchial tube

DISEASES OF THE LARYNX.

Acute Laryngitis.

What is acute laryngitis?

Acute laryngitis is a catarrhal inflammation of the mucous membrane of the larynx, characterized by slight fever, suppressed voice, painful deglutition, some dyspnœa and more or less spasmodic cough.

What are its causes and symptoms?

CAUSES.

It is caused by exposure to cold and damp, inhalation of irritating substances, and straining the voice, as in addressing a meeting in the open air. *The œdematous variety* may be intercurrent with Bright's disease.

SYMPTOMS.

The symptoms are oppression in breathing; a change in the voice varying from slight hoarseness to total extinction; difficulty in swallowing; tickling in the windpipe; slight fever, and dry hoarse cough, more frequent at night than in the day. This lasts for about a week and the cough becomes looser, the expectoration freer and the fever disappears. Sometimes it passes down and results in bronchitis.

In œdema of the glottis the difficulty of breathing increases, with much pain in the throat and paroxysms of dyspnœa which threaten suffocation. The cough and voice become suppressed and there are signs of faulty aëration of the blood. The paroxysms occur more and more frequently, and unless relief is quickly afforded the patient very soon dies.

What is the pathology of the affection?

It is a catarrhal inflammation of the laryngeal mucous membrane, which is inflamed in patches throughout.

There is at first swelling and diminution of secretion, followed by an increased secretion and return to the normal state; or serous infiltration resulting in œdema occurs into the loose connective tissue

about the rima glottidis, which may be entirely occluded, if the swelling is great. The infiltration may be purulent.

What is the diagnosis, prognosis and treatment?

DIAGNOSIS.

The diagnosis of *simple laryngitis* is easy. The active onset, with alteration of the voice, feverishness, difficulty in breathing, and swallowing, some dyspnœa and harsh dry cough can belong to no other disease.

Should there be any doubt, an examination with the laryngoscope will settle the question.

Œdematous laryngitis is recognized by the same history and the sudden occurrence of attacks of suffocation, with an examination of the larynx. By depressing the tongue thoroughly, the epiglottis will usually be seen much swollen and œdematous in these cases.

PROGNOSIS.

The prognosis of *simple laryngitis* is favorable. *Œdematous laryngitis* is less amenable to treatment, particularly if it occurs as an intercurrent affection, or in children.

TREATMENT.

The air should be kept moist and the temperature of the room about 68°. The patient should have a mild diet, principally of liquid food, as that is more readily swallowed without pain. Little pieces of ice held in the mouth aid in relieving irritation. A hot foot-bath may be administered and the bowels opened by a mild aperient.

Locally the inhalation of hot vapor is of service. The inhalation of the steam arising from compound tincture of benzoin, f3j, with spirits of chloroform, gtt. ij–v, poured into a teacupful of boiling water, is often of benefit. The application of bags of ice to the neck is of great service in severe cases.

Internally, in the early stages, mild diaphoretics are useful, as a little Dover's powder with nitrate of potash, or minute doses of tartar emetic and morphine. When the cough becomes loose it may be necessary, if it lingers, to use expectorants, as the ammonium salts or squill.

In the œdematous variety scarification, brisk purgation and profuse diaphoresis (by means of pilocarpine, if not contraindicated)

will generally afford relief. If these means do not succeed, tracheotomy may be resorted to. For the fever, aconite may be administered if necessary.

Chronic Laryngitis.

What is chronic laryngitis?

It is a chronic inflammation of the mucous membrane of the larynx, manifesting itself by irritative cough, change in the voice, difficulty in swallowing, and causeless attacks of oppression.

CAUSATION.

It may follow acute laryngitis, may be due to continued straining of the voice and the inhalation of irritating substances, to syphilis, tubercular ulceration or various neoplasms.

PATHOLOGY.

Examining with a laryngoscope the cords are seen thickened and inflamed. A certain amount of œdema is usually present, and polypi or other neoplasms may be found.

TREATMENT.

The treatment depends upon the cause. The patient should be made to save his voice. He should stop smoking and correct his habits. Any constitutional cause should be treated. His system should be built up with iron, quinine and strychnine, and an occasional laxative should be given. *In catarrhal cases* inhalation of compound tincture of benzoin, or the application of a solution of nitrate of silver (gr. xx–f℥j), or of zinc sulphate two or three times a week, or of glycerole of tannin, is usually sufficient to effect a cure. *If ulceration* of the larynx is present, the application should be stronger. *In tubercular ulceration* the parts may be brushed or sprayed with cocaine, which lessens the tendency to cough.

Phosphates, cod-liver oil, iodide of iron, etc., should be given internally, and tannic acid (gr. xx–f℥j) may be applied by the atomizer. The parts may also be touched with strong solutions of the mineral astringents, but the application should not be made too often. Demulcents, as the infusion of Irish moss, are very grateful to the patient.

In syphilitic ulceration the treatment is the same, iodide of potassium being given internally in large doses.

Should small polypi be found at the junction of the cords, causing incessant irritating cough, but no difficulty in swallowing, or loss of voice, they may be touched with a strong caustic, as chromic acid, and in favorable cases excised.

Croup.

What is croup?

Croup is a catarrhal inflammation of the mucous membrane of the larynx with a spasm of the glottis, recurring in paroxysms. There are *two varieties* of croup, false croup, or spasmodic croup, to which the above definition applies, and true croup, in which there is in addition a pseudo-membrane in the larynx, sometimes extending into the pharynx, or downward into the bronchial tubes.

What are the causes of croup?

It is a disease of childhood, although not confined to any age. Its development is favored by dampness. It would appear to be hereditary.

What are the symptoms of croup?

(1) False Croup.

The attack usually comes on at night; the child starts up with a peculiar, dry, croupy cough, recurring in paroxysms and without expectoration. During the paroxysm there is great difficulty in breathing, sometimes a sensation as of impending suffocation; noisy, stridulous respiration, and a harsh croupy voice and cry.

After a longer or shorter interval the paroxysm passes off, perspiration breaks out, and the child is relieved, although the hoarse voice and harsh cough remain during the next day. There may be slight fever; the pulse is about normal.

Usually the paroxysms return for two or three nights, after which the attack generally passes off, leaving a loose cough for several days.

(2) True Croup.

In true or membranous croup the symptoms at first are those just described, but usually more severe. The breathing remains embarrassed between the paroxysms, and the voice and cough croupy. There

is more fever; the respiration is noisy all the time; the paroxysms occur in the daytime as well as at night, although the former are milder. Each paroxysm is worse than the preceding until the third day, when the disease is at its height. There is then great difficulty in breathing, the circulation is embarrassed, and the vesicular murmur is very feeble, usually absent. Sometimes portions of the membrane are expectorated. In some cases the membrane may be seen in the pharynx. In bad cases the cough and voice become entirely suppressed.

Should the case terminate favorably the paroxysms become less frequent and less severe, and more membrane is expectorated. The dyspnœa, the cough and fever lessen and the voice gradually returns.

Should the case terminate fatally the attacks become more and more frequent and severe, expectoration is absent, and the cough and voice entirely suppressed. The respirations become frequent and shallow, interference with the circulation is more marked and the patient dies of asphyxia.

What is the diagnosis, prognosis and treatment of croup?

DIAGNOSIS.

The diagnosis of *simple croup* is easy. *True croup* may be suspected if symptoms of the first paroxysm continue for two or three days; if there is much fever; and if the respiration is embarrassed, and the vesicular murmur absent between the paroxysms.

If the membrane can be seen in the throat, the diagnosis is established.

DIFFERENTIAL DIAGNOSIS.

(1) *From spasm of the glottis* it is differentiated by the age, the history, the fever and the catarrhal symptoms.

(2) *From a tumor or swelling pressing on the windpipe.* In the latter case the symptoms continue. There is no cessation between the paroxysms.

(3) *From œdema of the glottis*: by the history and the absence of cause, the greater violence of the attacks of suffocation and depression in œdema, and by the laryngoscope.

(4) *From laryngeal diphtheria*, by being a local disease, the result of catarrhal inflammation and non-contagious, followed by no paralysis or sequelæ. There is no albumen in the urine, no enlarge-

ment of the cervical glands, and the membrane extends *from, not into* the larynx. In laryngeal diphtheria the constitutional symptoms precede the local symptoms and preponderate, the reverse being the case in membranous croup.

PROGNOSIS.

The prognosis of *false croup* is favorable. *True croup* is always dangerous. Favorable prognostic signs are the return of the respiratory murmur, the cough losing its harsh sound and the paroxysms returning less frequently.

TREATMENT.

The treatment is the same in both varieties, with the exception that if it be membranous croup the treatment should be more energetic, and surgical interference may be necessary.

(1) *During the paroxysm*, the child must be placed in a hot bath and kept there for some time, and small amounts of hot water administered internally. In addition to this emetics must be given at once; alum and the syrup of ipecac, or ipecac alone, may be given every ten minutes until emesis is produced. Antimony is preferred in England as an emetic, and sulphate of copper in Germany. The hot baths and the emetics should be repeated every time the paroxysm recurs.

(2) *Between the paroxysms*, nauseants should be given in small doses, frequently repeated. For this purpose squill and paregoric, or small doses of antimony (Kermes mineral, gr. $\frac{1}{8}$–$\frac{1}{4}$, with a minute amount of Dover's powder) or turpeth mineral answer best.

During the second night an opiate should be administered (paregoric or Dover's powder) to quiet the nervous system and, if possible, prevent a recurrence.

(3) *If the inflammation continues for thirty-six hours*, and it is feared that a membrane is forming, mercurials should be given in small doses with opium, frequently repeated and pushed to slight constitutional effects. Potassic chlorate is used by many in these cases in place of turpeth mineral.

(4) The patient should have a mild, easily digested diet, but sufficient in amount and liberal.

(5) *Local treatment.* Too many persons should not be allowed in the room. The air should be kept moist; temperature 68° to 70°.

Much relief is afforded by the inhalations of the vapor of slaking lime. To dissolve the false membrane a saturated solution of pepsin may be used by the atomizer, or a solution of trypsin applied in the same manner; the latter is said to give better results. Papaine is also employed for this purpose.

(6) *Surgical treatment.* If the case gets worse and suffocation is imminent, intubation may be resorted to, or, as a final resource, tracheotomy may be performed. The mortality after this operation in croup justifies its performance, while for laryngeal diphtheria it is of very doubtful utility. If the child is strong, if he is over five years of age, and if there is no dullness anywhere over the lungs, tracheotomy may be resorted to to prevent suffocation.

After the paroxysms have been checked and the patient is recovering, stimulating expectorants may be given.

To prevent subsequent attacks cold sponge baths may be employed morning and evening, or an ordinary tepid bath with cold sponging to the neck and chest may be used; but the cold bath is the better. No chest protector should be worn. The bowels should be kept regulated, rich food should be avoided, and great attention should be paid to the condition of the stomach.

DISEASES OF THE BRONCHIAL TUBES.

Bronchitis.

What are the varieties of bronchitis?

Bronchitis may be *acute* or *chronic.*

Acute bronchitis may affect the *large tubes* as a *catarrhal inflammation* (the most common variety), or it may be attended with *plastic exudation* into the tubes themselves. Acute bronchitis may also affect the *small tubes*, when it is called *capillary bronchitis.*

(1) Acute Catarrhal Bronchitis of the Larger Tubes.

What is this disease?

It is acute catarrhal inflammation of the larger bronchial tubes, due to cold and exposure; various diatheses, as the gouty, rheu-

matic or lithæmic; following the exanthemata, especially measles; seen also in typhoid fever, and occasionally due to syphilis.

It is also called *acute bronchial catarrh.*

What is its pathology?

In the first or dry stage secretion is arrested. The bronchial mucous membrane is covered with a thick coating, is swollen and injected. This gradually passes into the *second or moist stage*, in which the secretion is reëstablished and finally becomes profuse, the swelling disappears, and the mucous membrane slowly returns to its normal state.

What are the symptoms?

It begins sometimes with a chill and slight fever. *In the first stage* there is a harsh, dry cough, occurring chiefly in paroxysms; expectoration slight, stringy and streaked with blood; some shortness of breath; anorexia; nausea, sometimes vomiting; aching pain in the limbs and headache.

In the second stage the cough becomes looser, the expectoration freer, and, under the microscope, the sputa is seen to consist of muco-pus and epithelium, with no blood and no shreds of lung tissue. The fever declines, and cough gradually lessens, although it may continue for some time after recovery. *The average duration* is from ten days to two weeks.

What are the physical signs of acute bronchitis?

On Percussion, a clear note is heard.

On Auscultation, the vesicular murmur is almost normal, there is harsh respiration over both lungs in the early stages, and dry, sonorous râles heard throughout the chest, which in the second stage are replaced by large bubbling râles.

Vocal fremitus and vocal resonance are normal.

How is the disease recognized?

It is recognized by the physical signs, the symptoms and the course which it runs.

What is the prognosis?

The prognosis is favorable unless it becomes capillary, or runs

into broncho-pneumonia, which rarely happens except when it is an intercurrent affection.

How should it be treated?

Keep the atmosphere moist and the temperature of the room about 68°.

In the early stages let the patient have a light diet consisting of milk, oysters and other easily digested food, and in the *later stages* keep him well fed.

In the first stage, if the patient is a robust young adult and breathing with much difficulty, the chest should be thoroughly wet cupped.

Dry cups may be used in all other cases when there is difficulty in breathing.

If the disease is mild, counter-irritation may be applied to the chest by means of mustard plasters.

Act on the skin freely in the *very early stages* by pilocarpine, potassium citrate, small doses of tartar emetic, ipecac or small doses of Dover's powders. Let the patient have mucilaginous drinks, which, in some way, appear to allay the irritation.

For the cough let him inhale the vapor arising from the tincture of benzoin (℥j) in boiling water (Oss) every two hours, or give him broken doses of Dover's powders.

If improvement does not take place, or if the disease shows a tendency to move downwards, the fluid extract of veratrum viride (gtt. j) may be given every two or three hours, guided by the effect on the pulse.

In the second stage give stimulating expectorants, as ammonium chloride or carbonate, combined with a little deodorized tincture of opium, and squill or senega.

(2) Plastic Bronchitis.

How does this differ from the disease just described?

In this affection a fibrinous exudation occurs, forming pseudo-membranous casts of the tubes. The symptoms and physical signs are identical with those of acute bronchial catarrh, with the exception that there is more dyspnœa, usually more fever, more depression, and the paroxysms of cough are more severe.

The disease terminates by the expectoration of small, membranous casts of the tubes.

The treatment is similar to that of acute bronchial catarrh, but should be more active. The patient should be kept well nourished, and, in the later stages, even free stimulation may be necessary.

Bronchitis Affecting the Smaller Tubes—Capillary Bronchitis.

What is capillary bronchitis?

Capillary bronchitis, or suffocative catarrh, is a catarrhal inflammation of the smaller bronchial tubes, having the same *pathology* as acute catarrh of the larger tubes. A plug of mucus will often be forced by inspiration into a small tube, completely blocking it up. On expiration this plug will be dislodged sufficiently to allow the passage of air upward from the lungs, but on inspiration it will again block up the tube, and thus, finally, the portion of the lung to which the ramifications of the tube extend, being deprived of its air, will collapse.

The lung structure itself is rarely involved, except when secondary adhesive inflammation occurs in the collapsed portion. The plug of mucus which has caused the collapse will frequently be dislodged by coughing, vomiting, or from some other mechanical cause, thus allowing the air again to enter the collapsed vesicles.

This disease occurs especially in badly-nourished children and in old, debilitated subjects.

What are the symptoms?

The symptoms at first are those of ordinary acute bronchitis, but as the inflammation extends downwards there is more fever, more depression, and symptoms indicating the want of aëration of the blood, as great difficulty in breathing, feeble circulation, blueness of the lips, prominence of the veins, etc. As the disease progresses the patient becomes weaker and weaker, until he has not sufficient strength to expectorate, and the cough itself becomes suppressed.

The patient is dull, stupid, delirious, with cold hands and extremities, profuse sweats, irregular pulse, convulsions, and finally death. The expectoration often consists entirely of pus.

What are the physical signs of this disease?

PERCUSSION elicits a clear note, excepting over the spots of collapsed lung, where the note is dull. The dullness, however, shifts from time to time as the secretion moves and the collapsed lobules fill with air.

ON AUSCULTATION diffused sibilant râles are heard in *the early stages*, and the respiratory murmur is feeble. In *the second stage*, the sibilant râles are replaced by subcrepitant râles, widely diffused over the chest.

As the case improves the râles lessen, harsh respiration is heard, and recovery takes place, as in acute bronchitis of the larger tubes.

If the case does badly, the vesicular murmur is more and more feeble, and the râles disappear as patient loses strength.

What is the diagnosis?

The disease is recognized by the age at which it occurs, the physical signs, and the symptoms of want of aëration of blood.

DIFFERENTIAL DIAGNOSIS.

(1) *From Broncho-pneumonia.*—In the latter disease there is not the same difficulty of respiration or of expectoration. Dullness is persistent, not shifting, and the disease is not so diffused.

What is the prognosis and treatment?

PROGNOSIS.

The disease is always dangerous, especially in old persons.

TREATMENT.

If the case is seen early, the disease is still spreading downward and the patient is strong enough, leeching or wet cupping must be thoroughly employed. In an old person use dry cups.

In either case the use of diaphoretics and of veratrum viride is beneficial.

When the disease has become capillary and there is great difficulty in breathing, if the patient is strong enough, the bold use of emetics, continued as long as the strength of the patient will admit, is of undoubted utility. Emetics act mechanically by relaxing the system and getting rid of the secretions in the tubes. Thus, ipecac, zinc sulphate or apomorphine may be employed, and frequently repeated. In addition to this treatment the chloride or carbonate of ammonium

should be administered; quinine given as a tonic by the mouth or rectum, the patient fed systematically every hour, and stimulants, as champagne, brandy or whiskey, used freely. A jacket-poultice is sometimes of service.

Chronic Bronchitis.

What is chronic bronchitis?

Chronic bronchitis is a chronic catarrhal inflammation of the larger bronchial tubes which may follow an acute attack, but usually is slowly developed, giving rise to a certain amount of winter cough, which disappears in the summer to return the following winter, with very slight impairment of the general health; affecting persons of advanced years, and often associated with certain diatheses, as gout or rheumatism. The expectoration may be slight or profuse.

Pathology.

The pathology is similar to that of the acute disease, but is slower in progress and of longer duration. Finally, a structural alteration occurs in the mucous membrane, which is thickened, as is also the underlying connective tissue. In some cases the bronchial tubes will be dilated as a consequence of long-standing disease.

What are the physical signs?

Percussion of the chest elicits a clear note.

On Auscultation, the vesicular murmur is not interfered with, but is combined with some harshness, almost amounting to bronchial breathing. Râles are frequently heard, especially posteriorly, which will, however, vary with the amount and character of the secretion.

What is the diagnosis, prognosis and treatment of chronic bronchitis?

Diagnosis.

The history and the physical examination render the case clear.

Prognosis.

Favorable as to life. The disease, however, is very obstinate.

Treatment.

The treatment consists in clothing the patient warmly, giving nourishing food, and preventing him, as far as possible, from taking

fresh cold. Cod-liver oil and the iodides, especially the iodide of iron, are very useful. Change of climate is often of service, a mild, dry climate being usually the best.

If the secretion is scanty, the alkalies, particularly the chloride and iodide of ammonium, may be advantageously given. Preparations of tar and of iodine may be inhaled with good effect. Sulphur and arsenic are also used internally.

Should the secretion be profuse the ammonium salts may be administered as alteratives, while astringents should also be employed, as the preparations of zinc or of tannic acid.

Cubebs and copaiba are also given. It is better not to use opium without it is absolutely necessary; in these cases the combination of codeine with diluted sulphuric acid and prunus Virginiana will generally answer the purpose, opium itself being reserved for cases of absolute necessity.

Asthma.

What is asthma?

Pure asthma is probably a spasm of the bronchial mucous membrane.

What are its causes?

It is often inherited. The gouty diathesis, particularly in women, often causes this affection. In some cases it is due to the inhalation of irritant particles. Emphysema is often associated with it, but does not cause it. It is seen in middle age as a rule, rarely in the very young or very old. So-called "cardiac asthma" and "renal asthma" are cases of dyspnœa, not asthma, and are due to a congested condition of the lung. The exciting causes of asthma are bronchitis or an attack of lithæmia.

What are its symptoms?

The attack is apt to be preceded by digestive disturbance; the urine is loaded with urates, and perhaps some slight dyspnœa occurs, lasting for several days.

The attack itself is ushered in by greatly embarrassed respiration, with loud, wheezing râles, usually expiratory.

The patient experiences great difficulty in breathing, with a sense of oppression in the chest, and calls into play the extraordinary

muscles of inspiration, in order to enable him to breathe more freely. After a time there is profuse expectoration, and gradually the paroxysm subsides, to be repeated again on the ensuing evening or evenings, and finally to pass away entirely.

It is apt to lead to emphysema, or to cardiac disease, by impeding the circulation through the lung.

What are the physical signs?

On Percussion the chest is resonant.

On Auscultation, loud, wheezing, expiratory râles are heard, which, later in the attack become large, moist râles.

How is the disease recognized?

The diagnosis is easy. Difficulty in breathing, with the physical signs, render the case plain.

Differential Diagnosis.

(1) *From Cardiac Dyspnœa.*—The latter is more persistent, having nothing paroxysmal about it, and the presence of the cardiac trouble should prevent mistake.

(2) *From Difficulty in Breathing caused by Nasal Polypi.*—Although nasal polypi may sometimes act in a reflex manner and cause asthma, yet, as a rule, the difficulty in breathing is nasal, rather than bronchial. An examination of the nose will reveal the difficulty.

(3) *Reflex Asthma from Uterine or Hepatic Disease.*—In these cases there will be symptoms calling attention to the organ involved.

(4) *Laryngeal irritation or laryngeal polypi causes noisy respiration* which is sometimes paroxysmal. In cases of doubt the laryngoscope will decide the question.

What is the prognosis and treatment of asthma?

Prognosis.

The prognosis, as regards life, is favorable, though the underlying causes to which the disease is due, or the affections to which it leads, may cause death. It is a chronic malady.

Treatment.

(1) *Of the Paroxysm.*—The patient should be kept in a moist atmosphere and counter-irritation, especially by dry cups, applied over the chest.

Nauseants, as lobelia, may be given frequently in doses not large enough to produce actual emesis.

Inhalation of the fumes arising from burning stramonium leaves is of use. Coffee, caffeine or cocaine are of service, especially in uncomplicated cases where the nervous element is pronounced. Grindelia is sometimes of service.

Where the case lingers and is very severe, chloroform may be administered by inhalation.

Ammonium salts may be given to promote expectoration. Hypodermic injections of pilocarpine sometimes materially modify the affection, particularly if the urine is scanty. In these cases dry cups may be applied over the kidneys. Diffusible stimulants, as Hoffman's anodyne, and alcoholic stimulants are of value.

(2) *To prevent the recurrence of the attacks*, potassium or ammonium iodide may be administered in decided doses for a prolonged period. Arsenic or belladonna may also be used for this purpose. Change of climate exercises a decidedly beneficial effect. The high lands of Colorado suit the majority of patients. Some cases are benefited, however, by low altitudes, and in some a very moist climate gives the best result. Some cases are benefited by inspiring compressed air, and others by inspiring rarefied air.

DISEASES OF THE LUNG-TISSUE.

Emphysema.

What is emphysema?

Emphysema is usually a dilatation of the air-vesicles of the lungs, or, occasionally, the rupture of these air-vesicles, the air traveling along the connective tissue of the lobules of the lung.

What is its pathology?

The air-vesicles are distended or dilated and lose their elasticity, in some places running together, especially along the anterior edges of the lung. The tubes which terminate in these air cells are inflamed, giving rise to bronchial catarrh. The lung is pale and

anæmic, because the vessels are pressed upon by the dilated vesicles. It is rarely a local disease, both lungs being more or less affected, though not equally. The vesicles rupture into one another, or into the neighboring bronchial tubes; they rarely rupture into the pleura. The pulmonary circulation is impeded, and dilatation of the right heart results. This is the great danger in emphysema. Under the microscope the walls of the vesicles are seen to have undergone degeneration. The lung, of course, is lighter than normal, and contains more air.

What are its causes?

It is often hereditary. It may be acquired by prolonged expiratory efforts, as when it occurs in performers upon wind instruments; chronic bronchitis is also a cause. Degenerative tissue changes in the walls of the air-cells, without other cause, may produce it.

What are the symptoms?

There is shortness of breath, increased by the slightest exertion. The patient frequently suffers from attacks of bronchial catarrh. In cases of long standing, symptoms due to dilated heart, as dropsy, weak heart, palpitation, and interference with the function of the liver and kidneys from backing up of the blood in their veins, with more or less enlargement of these organs, supervene. The patient also suffers from dyspepsia, caused by interference with the venous circulation of the stomach. His face is melancholy, and he is depressed and gloomy.

What are the physical signs?

On Inspection, the thorax is barrel-shaped, the intercostal spaces being obliterated or bulging. There is a want of proper expansion on inspiration relative to the size of the chest.

On Percussion the vesiculo-tympanitic note is elicited. *Respiratory percussion* shows no change in the note, a point of importance.

On Auscultation the vesicular murmur is feeble and expiration is prolonged. Bronchial râles are often present, from temporary catarrh, and may mask the auscultatory signs. Later in the disease the signs of dilated heart are present.

How is this disease recognized?

It is recognized by the shortness of breath and the physical signs which are present on both sides of the chest.

What is the prognosis?

Though few patients recover, the disease is not dangerous to life, but may produce cardiac disease, which will cause death.

What is the treatment?

The patient must, if possible, live in a dry climate, but the altitude should not be great, on account of the tendency to cardiac disease.

To prevent or control the bronchial catarrh he should be protected as much as possible from exposure. *Should an acute exacerbation occur* he should be put to bed, thoroughly cupped, and diaphoretics and diuretics administered.

This complication should be very actively treated. Iodide of potassium is of service by relieving the chronic bronchitis and lessening the tendency to the attacks of asthma.

With the above treatment should be combined frequently repeated small blisters applied to the chest.

Inspiring compressed air and expiring into rarefied air is reported to have done good.

For the tendency to cardiac dilatation digitalis is preëminently of value. The secretion of the kidneys should also be kept up, and the portal circulation acted on by blue pill, by the salines, etc.

If rupture of the air-vesicles takes place, as may occur in whooping-cough or during the violent expulsive pains of labor, there will be, in addition to the difficulty in breathing, cough, and frequently the presence of air in the areolar tissue of the neck. *The treatment* should consist in keeping the patient perfectly quiet and giving opium to prevent the recurrence of the coughing, and if there is a crepitating swelling externally, puncture it and let the air out.

Pulmonary Hyperæmia.

What is pulmonary hyperæmia?

Pulmonary hyperæmia is an active or passive congestion of the lung, which is exceedingly rare as a primary affection, and when

present is usually secondary to, or intercurrent with, some other disease. It is characterized by shortness of breathing, slight cough, and the physical signs indicating some impairment in the respiratory powers of the lungs.

What are its causes?

Active congestion may occur after violent efforts in singing or talking; or after violent exercise, as in ascending high mountains. An over-acting, powerful heart, by forcing more than the normal blood into the lungs, may also produce congestion in them, as in other parts of the body.

Passive congestion is seen when the blood slowly accumulates in the lungs, as sometimes occurs in low forms of fever (*e. g.*, typhoid fever); or in Bright's disease; in certain cardiac diseases, as mitral insufficiency, or in anæmic states if the patient lies on his back continuously for too long a time.

What are the symptoms?

The symptoms are the same in either variety, and are, marked shortness of breath; cough, without much expectoration, but what there is will be streaked with blood; quickened circulation, with little or no fever, and restless nights.

What are the physical signs?

On Percussion the resonance is slightly impaired, but only to a trifling extent.

On Auscultation the vesicular murmur is diminished in intensity; a few fine bubbling râles (subcrepitant) and occasionally, perhaps, a vesicular râle (the crepitant râle) will be heard.

What is the prognosis of pulmonary hyperæmia?

In active hyperæmia the prognosis is favorable; in the *passive form* it is not so good, and will depend upon the cause of the congestion.

How are pulmonary congestions treated?

In active congestion, if very severe, wet cups may be employed, or even venesection resorted to. The action of the heart must also be regulated, if very strong and forcible, by aconite.

In passive congestion digitalis should be administered for its effect on the heart.

In either variety dry cups are very useful, and purgatives must be used freely, as must, also, diaphoretics. In passive congestions occurring in Bright's disease, the kidneys must be thoroughly cupped, and diuretics administered.

Œdema of the Lung.

Describe œdema of the lung.

This may follow either acute or chronic pulmonary hyperæmia, as a result of which it is much to be dreaded. It sometimes follows alcoholic excesses, and consists of an effusion of serum in the pulmonary tissues and into the air-vesicles.

The SYMPTOMS are those of congestion of the lung, with great difficulty in breathing, much anxiety, constant, short cough, and a frothy expectoration, often streaked with blood.

The PHYSICAL SIGNS consist of slightly impaired percussion resonance, feeble or distant vesicular murmur, and fine vesicular râles diffused over both sides of the chest, with loud râles, from a serous effusion into the bronchi.

The PROGNOSIS is very grave. It is worse if the disease follows passive congestion.

TREATMENT.

The treatment is identical with that of congestion of the lungs, in addition to which, as the fluid accumulates in the vesicular structure and the dyspnœa increases, stimulating expectorants, particularly ammonium carbonate, should be freely used. If cardiac failure threatens, digitalis should be administered hypodermically.

Hæmoptysis.

What is hæmoptysis?

Hæmoptysis, or hemorrhage from the lungs, is a symptom rather than a disease.

(1) It often results from congestion, especially of the acute variety, and occurs from this cause in those who are ascending high mountains, or who strain their voices to the utmost, as in public speaking, particularly in the open air.

(2) It is very frequently due to the structural alterations of the lungs, and especially to tubercle (this should always be suspected if there has been no excitement or other cause leading to acute congestion); also to abscesses, gangrene or cancer.

(3) It sometimes occurs as a vicarious hemorrhage, taking the place of, or accompanying, menstruation or the bleeding of hemorrhoids.

(4) It sometimes occurs in individuals with peculiar idiosyncrasies, whenever they indulge in certain articles of food, as honey.

(5) Occasionally it is seen in women who do not belong to the class of hæmatophilia, and who are apparently in perfect health. In these cases it may occur daily, and continue for years without apparent cause, and without the slightest injury.

Pulmonary hemorrhages may be due either to rupture of a vessel or to transudation through its walls.

What are the symptoms of pulmonary hemorrhage?

It often comes on suddenly, the patient having a slight cough and spitting up blood, a mouthful at a time. There is a feeling of utter demoralization, the breathing is quickened, and the patient pale and alarmed.

The blood is red and frothy, and at first is uncoagulated, but as the case proceeds, coagulated blood which has remained in the bronchial tubes will finally be expectorated.

These hemorrhages are very apt to occur in series, the bleeding taking place frequently for some time, and then ceasing, an interval of several months elapsing before the hemorrhages recur.

Pulmonary apoplexy is the name given to this affection when the patient bleeds internally, but very little blood escaping externally. In this condition the blood flows into the vesicular structure of the lung, and while in an ordinary hemorrhage the lung clears up when the trouble is over, in this condition it remains in the lung, sets up a secondary inflammation, and results in pneumonia. It is usually seen with cardiac disease, and is due to thrombosis or to embolism. It is not common.

What are the physical signs of hæmoptysis?

There are no physical signs of any importance in this affection. Some râles may be heard during the hemorrhage, but the blood is

expectorated, and the lung clears up entirely, or at least returns to the condition in which it was previously, as soon as the hemorrhage ceases.

In pulmonary apoplexy the blood remains in the lungs, and the signs of enfeeblement of the respiratory powers followed shortly by those of pneumonia result.

What is the diagnosis of hæmoptysis?

(I.) Diagnosis of the Origin of the Blood.

(1) *From Hæmatemesis.*—In this affection the blood is black and clotted; there is usually preceding nausea and vomiting; and the blood is usually mixed with the contents of the stomach.

Sometimes, however, in hemorrhage from the stomach, the blood will be red. In these cases a large vessel has generally been pierced by an ulcer, and the blood poured out in such large quantities that it has irritated the stomach, and caused vomiting, before it had time to become altered by the secretions.

Here, however, the stools will be black, which is not the case in hæmoptysis, except in children who are too young to expectorate the blood, and consequently swallow it.

(2) *From Epistaxis.*—The blood may trickle down the throat from the posterior nares, and be subsequently vomited.

In these cases an examination of the throat will reveal the blood trickling down; or a laryngoscopic examination of the posterior nares will make the diagnosis plain.

(3) *Hemorrhage from the gums* may be diagnosed from hæmoptysis by examining the gums.

(II.) Diagnosis of Cause.

The patient must be questioned closely in order to elicit the true cause of the hemorrhage.

Pulmonary apoplexy is recognized by the slight external hemorrhage and the great amount of oppression; by the spots of localized dullness, and the underlying cardiac disease.

What is the prognosis?

The prognosis is favorable as regards the result of the hemorrhage itself. People never die of pulmonary hemorrhage, except when it occurs as the result of the rupture of an aneurism, when death is usually almost instantaneous.

Vicarious hemorrhages are not dangerous.

Pulmonary apoplexy is apt to eventuate in consolidation and to terminate in pneumonic phthisis.

The hemorrhage is of little moment; the cause of the hemorrhage is the matter to be determined from a prognostic point.

How is hæmoptysis treated?

Absolute rest must be insisted on. The sufferer must not be allowed to speak a word, and must be kept perfectly quiet. He should be given easily digested food in small quantities, with small pieces of ice in his mouth, but not much to drink. Externally, mustard plasters or turpentine or dry cups may be employed when they can be used without disturbing the patient too much. The circulation should be attended to, and if the pulse is strong, aconite must be administered, giving gtt. j of the tincture every hour until it is reduced. In active hemorrhages ice applied to the chest is of use.

The best hæmostatics in pulmonary hemorrhage are ergot in f℥ss doses, administered every hour or two. It may be used hypodermically. It is often alternated with gallic acid. This drug is given in doses of gr. xx repeated every fifteen or twenty minutes until the blood turns black, when the intervals between the doses should be increased. If it induces nausea, add a few drops of a mineral acid to each dose.

Lead acetate may be given in doses of gr. ij. with opium gr. $\frac{1}{4}$, every two hours; diluted sulphuric acid gtt. x, or turpentine ♏x, combined with opium and given every two hours sometimes succeeds when other remedies have failed; small and repeated doses of cupric sulphate (gr. $\frac{1}{12}$–$\frac{1}{8}$) combined with opium, or the tincture of matico (℥j every two hours) are also useful.

Whatever remedy is used, opium should be combined with it, to calm any irritative cough, or nervous perturbation which may be present.

Acute Lobar Pneumonia.

What is acute lobar pneumonia?

It is an acute, croupous inflammation of the lung tissue, attended with exudation into the air-vesicles.

What are its synonyms?

It is called croupous pneumonia, pneumonitis and pneumonia.

What is its pathology?

The disease consists of three stages.

In the first stage (that of congestion) the blood vessels are engorged, proliferation of epithelium takes place in the air-vesicles, partly occluding them, but sufficient air remains to make the lungs float in water.

In the second stage (the stage of exudation, consolidation, or red hepatization), the lungs are solid and firm. The small tubes and air-vesicles are filled with embryonic tissue and retained secretions. On section the lung is of the color of a piece of liver, hence called *red hepatization*, and a portion of lung-tissue placed in water sinks.

Third stage. The disease either ends in resolution or in gray hepatization. If it ends in *resolution*, the exudate undergoes fatty degeneration, and assumes a yellow appearance, due to fatty degeneration of the embryonic tissue, which is either absorbed or expectorated. This is sometimes spoken of as "*yellow hepatization.*"

If it ends in *gray hepatization* the exudate and air-cells soften, undergo caseous degeneration, break down, and form a sort of purulent infiltration, spoken of as "gray hepatization." The whole lung softens, but abscess rarely forms, there being no circumscribed cavity containing pus, but purulent infiltration of the lung tissue.

Sometimes the pleura is slightly involved.

The bronchial tubes are also slightly inflamed.

There is a tendency to cerebral or meningeal congestion.

Endocarditis is not uncommon, perhaps leading to heart-clots, which sometimes prove fatal.

Congestion of the kidneys with albuminuria is also frequently found.

What is the seat of this disease?

In two-thirds of the cases, the lower lobe of the lung on the right side is affected. If the upper lobe is affected, the whole lung usually is consolidated. Double pneumonias are very rare.

What is the cause of this disease?

It may occur at any age, but is most common between the ages of twenty to forty years. Pneumonias of children and of old persons

are usually catarrhal. Sometimes it appears to be epidemic, and is believed to be due to a specific germ (the *pneumococcus*). It is usually, however, due to exposure to cold and damp. It may follow bronchitis, is produced by malaria, and may be intercurrent in rheumatism, Bright's disease, or any affection characterized by blood-poisoning.

What are its symptoms?

There is a chill, more or less severe; flushed face; headache; fever, the temperature rarely being above 105°, and falling rapidly to or below the normal during convalescence. There is some oppression in breathing, the respirations being hurried, from twenty-four to eighty per minute. The pulse does not rise in proportion to the rapidity of the breathing, and during convalescence falls below the normal. Some cough is present, paroxysmal in character, but not very marked. The expectoration is striking, it is of a rusty color, sticky, tenacious, and adhesive. The urine is deficient in chlorides during the height of the disease, but they rapidly reäppear as recovery takes place. Albuminuria occurs in bad cases. Delirium is sometimes seen, and is a bad sign. In typhoid pneumonias or pneumonias of the upper lobe in children, it is usually present. When it occurs in an adult, always examine the urine for kidney complications. There is pain in the neighborhood of the right nipple, shooting downwards to the region of the ileo-cæcal valve. Jaundice is also occasionally seen.

In the third stage, when absorption takes place, perspirations more or less profuse occur; the urates and chlorides return to the urine; the cough becomes looser, the sputa is freer and loses its rusty appearance, and the fever rapidly declines.

If gray hepatization occurs, there is great prostration. The respiration becomes faster, shallower, and more labored; there is flapping of the alæ nasi; the hectic flush disappears from the face, and signs of impeded circulation are present. The sputa is dark, offensive, and purulent, loses its rusty color, and becomes light brown (called *prune-juice* expectoration).

What is the duration?

The average duration is from ten days to two weeks. *The first*

stage usually lasts from one to three days; *the second stage* for five days or more, and *if resolution takes place*, it occurs from the seventh to the eleventh day of the disease. *Gray hepatization may occur* from the fifth to the seventh day, terminating in death after two or three days.

What are the physical signs of this disease?

IN THE FIRST STAGE *the percussion resonance* is somewhat impaired. *On inspection*, the respiratory movement is diminished on the affected side. *On auscultation*, there is a feeble inspiration, prolonged expiration and crepitant râles (pathognomonic of pneumonia), heard only on inspiration.

DURING THE SECOND STAGE there is marked dullness on *percussion*, marked deficiency of respiratory movement on the affected side, the movements of the sound lung being increased; marked bronchial breathing *on auscultation*, the vesicular murmur being absent, and marked exaggeration, as a rule, of both *vocal fremitus* and *vocal resonance.*

DURING THE THIRD STAGE, *if the exudation is being absorbed and expectorated*, the dullness on percussion lessens, the movement of the chest increases, some slight vesicular breathing is heard, the bronchial breathing is less distinct, and the crepitant râles return.

When, on the other hand, gray hepatization and softening of the lung is taking place, the signs of consolidation remain as in the second stage, without change.

How is this disease diagnosed?

The diagnostic points are the hurried respiration, the rusty-colored sputum, the pain, the physical signs, and the short duration.

(A) DIFFERENTIAL DIAGNOSIS.

(1) *From Acute Bronchitis.*—By the dull percussion note and other physical signs indicated in consolidation.

(2) The first stage might be mistaken for that form of *congestion leading to œdema*, in which there are crepitant râles, hurried breathing, slightly impaired resonance and moderate fever; but the latter occurs in fevers, or intercurrent with Bright's disease, or in cardiac diseases, and the signs are diffused over both lungs. In case of

doubt, a few days will give dullness and the sounds of consolidation in pneumonia, which readily distinguish it from congestion.

(3) *The first stage of pneumonia differs from the first stage of pleurisy*, in that there is less pain in the former, more cough, rusty-colored sputum and crepitant râles, but no friction sound, such as is heard in pleurisy.

(4) *The stage of consolidation might be mistaken for pleurisy, with effusion;* but in pneumonia there is a severe chill and the fever is higher, there is rusty-colored expectoration, and the physical signs of consolidation of the lung (bronchophony, increased vocal fremitus and marked bronchial breathing) are all absent in pleurisy.

(B) Diagnosis of Complications.

(1) *The Cardiac Complications, from Acute Endocarditis.*—A cardiac murmur may occur in pneumonia without the supervention of acute endocarditis; the former is due to blood changes and alteration in pressure, and is an inconstant murmur, while the latter persists.

(2) *The formation of heart-clots* in pneumonia is diagnosed by the increasing difficulty in breathing, cold hands and feet, and signs of impeded circulation usually occurring during the second stage. Death may take place suddenly, in these cases, from cardiac paralysis.

(C) Diagnosis of the Different Forms of Pneumonia.

(1) *Epidemic pneumonia*, *embolic pneumonia*, and *pneumonia due to cold and exposure* cannot be differentiated absolutely except by the history. If the temperature is very high it is probably a general (epidemic) pneumonia. If the bloody sputa continues for a long time, especially if cardiac disease is present, it is probably an embolic variety.

What is the prognosis?

Eight to twenty per cent. die of this disease. The mortality steadily increases with the age of the patient, and is lower before the twentieth year. Unfavorable symptoms are—temperature above 105°; very rapid pulse; early delirium; repeated attacks; relapses; and double pneumonias. Intercurrent pneumonias are exceedingly dangerous. Acute pneumonias, or pneumonias occurring in drunkards, give a very grave prognosis. Pneumonia occurring during pregnancy is also very serious.

What is the treatment?

The patient must be confined to bed and kept quiet. He should

be fed moderately, stimulants only being required in typhoid pneumonias or in the third stage with purulent infiltration.

Venesection generally does harm. If the disease is caused by cold and exposure, and occurs in a strong, active adult, with a flushed face and much oppression in breathing, with a tense and frequent pulse, and if he is seen in the first stage before consolidation has occurred, f℥viij–xij of blood taken by wet cups from the chest will relieve the oppression, but has no influence on the course of the disease.

In the first stage, before consolidation is present, if the patient is young and the circulation very active, a few drops of veratrum viride, or of aconite, may be given every few hours, watching the effects closely, and discontinuing the medicine as soon as consolidation occurs.

When consolidation is present quinine should be administered, giving from gr. xij–xviij or xx in twenty-four hours, in divided doses. It lowers the temperature and limits the amount of exudation, and is especially useful in intercurrent, typhoid or malarial pneumonias, and in those of old persons or of drunkards. Digitalis should be combined with quinine in this stage, the dose being proportioned to the effects produced. It acts by reducing the pulse, equalizing the circulation, strengthening the heart, and keeping up the secretion.

Alkalies, especially ammonium carbonate, are given throughout the case, not as expectorants, but to liquify the exudation. They may be administered alternately with quinine and digitalis. If the ammonium salts are not well borne by the stomach, potassium acetate or citrate may be substituted for them.

Locally, if there is much pain or oppression, dry cups are of use.

If the case is complicated with pleurisy, poultices are of service. In all other cases, if there is much pain, mustard plasters or turpentine stupes should be resorted to. When resolution is taking place, stimulating expectorants are useful, as squill or senega, in addition to the ammonium preparations already given. If consolidation lingers, the ammonium salts, the iodides, or corrosive sublimate should be administered. Repeated blisters are also of value.

If grey hepatization takes place, or if the pneumonia becomes typhoid in character, sustain the vital powers by stimulants, promote

free expectoration, give iron and quinine, and administer brandy judiciously.

During convalescence keep up the secretions by laxatives and diuretics, and give quinine and other tonics.

Stimulants have done as much harm as the lancet in pneumonia. If, however, the patient is weak, or the heart-sounds feeble or irregular, or if the stage of gray hepatization is present, stimulants must be administered, as in any other disease. In pneumonias of drunkards, stimulants are required because of the weakness and the feeble heart.

Broncho-Pneumonia.

What is broncho-pneumonia?

It is a catarrhal inflammation of the vesicular structure of the lungs, resulting in patches of consolidation diffused over both sides, affecting especially young children or old persons, and usually commencing in an attack of acute bronchitis.

What are its synonyms?

It is also called catarrhal or lobular pneumonia.

What is the pathology of this affection?

It is a catarrhal process, beginning as a bronchitis and extending down into the air-vesicles. The pathology is similar to that of lobar pneumonia, excepting that in this disease the lobules, not the lobes, are involved, and that it usually affects both lungs symmetrically.

The process of resolution is similar to that of lobar pneumonia, but the spots of consolidation are apt to linger, may undergo caseous degeneration, and result in pneumonic phthisis.

What are the symptoms?

When the inflammation is about to invade the lung structure, the temperature rises considerably and may be very high; the cough increases and often gives rise to much pain; the sputum is purulent, tenacious and streaked with blood, but is not rusty-colored, or, at any rate, only for a short time; there is violent headache, often delirium, and sometimes early in the disease convulsions occur.

The breathing is not nearly so frequent as in ordinary lobar pneu-

monia, and there is nothing resembling a crisis in the remotest degree, on the contrary, the fever continues for several weeks—much longer than in ordinary pneumonia, and gradually subsides. Sometimes spots of consolidation remain, leading to chronic pneumonia, and often eventuating in pneumonic phthisis.

This disease does not so frequently attack adults as does lobar pneumonia, but is more frequent among children than is the latter. When it attacks adults the nervous phenomena are not so marked as they are when the disease occurs in children.

What are the physical signs?

The physical signs are those of acute bronchitis, with here and there spots which are dull on percussion, and over which are heard fine vesicular râles and an approach to, or perfect bronchial breathing with other signs of consolidation as in lobar pneumonia. These spots are usually symmetrically distributed over the lungs, and much more commonly affect the apex, than does the other variety of pneumonia. Signs of pleurisy are often present.

How is this disease recognized?

It is diagnosed by the ages at which it occurs; by following an attack of bronchitis; by the symptoms, and by the signs of patches of consolidation, symmetrically distributed over both lungs.

Differential Diagnosis.

(1) *From Acute Lobar Pneumonia*, it is differentiated by the preceding bronchitis; the higher temperature and more continuous fever; by the absence of rusty-colored sputum, and by the physical signs of consolidation diffused here and there in spots over both lungs.

(2) *From Capillary Bronchitis with collapse of various lobules.* In this disease there is no dulness unless the lobules are collapsed or inflamed (when it passes into broncho-pneumonia). Where the lobules are collapsed the spots of dulness will shift, as the plug of mucus is dislodged by the cough and the lobules again expand. In pneumonia the spots of dulness are fixed and diffused; the signs of deficient aëration of the blood are not so pronounced as in capillary bronchitis; there is less difficulty in breathing and the disease is not so grave, nor attended with so much depression of the vital powers.

What is the prognosis?

The prognosis does not differ materially from that of acute croupous pneumonia. It is more liable, however, to leave behind spots of consolidation.

How should it be treated?

The patient should be confined to bed and must remain there for a long time. He must be kept perfectly quiet and not allowed to talk too much. If he is active, strong and young, a few leeches may be advantageously applied to the chest.

Counter-irritation by means of turpentine or mustard plasters, followed by the application of poultices, often give great relief.

In other respects the treatment is the same as that for other forms of pneumonia, except that expectorants must be used more freely. The ammonium salts, with quinine and digitalis, are all indicated, the two latter being useful not only as tonics and alterants, but as tending also to reduce the temperature.

When the morbid process is extending, or when it lingers and chronic pneumonia is feared, potassium iodide must be given in decided doses and pushed, with the hope of causing absorption, at the same time that counter-irritation, by blisters, may be employed over the spots of consolidated tissue.

During convalescence cod-liver oil and the iodide of iron must be administered, and a change of scene should be ordered if the patient can afford it.

Chronic Pneumonia.

What is chronic pneumonia?

Chronic pneumonia may follow acute pneumonia, a spot of consolidation remaining after either variety of the affection, or it may develop slowly, being chronic from the start. It is usually so closely associated with tuberculosis that it will be discussed in connection with the latter affection, under the head of "pneumonic phthisis."

Pulmonary Gangrene.

What is pulmonary gangrene?

Gangrene of the lung is a rapid breaking down of lung-tissue following pneumonia, traumatism, pyæmia or embolism, and characterized by cough, rapid wasting, profuse sweating, a horrible, sickening, gangrenous odor, especially after coughing, and the physical signs of a cavity.

What are its symptoms?

The symptoms are, rapid wasting, with great prostration and progressive loss of strength; dyspnœa; hemorrhage, often profuse; a rapid irritable pulse, and a cough accompanied by a disgusting, sickening odor, and a profuse greenish-brown expectoration.

What are the physical signs of gangrene?

The signs at first are those of consolidation, followed shortly by those of a cavity, usually situated at the lower part of the lung and generally confined to one lung only.

How is it recognized?

It is recognized by the symptoms, particularly by the persistent odor, and by the signs of a cavity resulting from the breaking down of the lung-tissue.

What is the prognosis?

The vast majority of cases die of exhaustion, or of hemorrhage. It is not, however, entirely hopeless, as occasionally the gangrenous portion of the lung sloughs and is expectorated, and the patient recovers, the cavity having cicatrized.

How is gangrene of the lung treated?

The patient must be well fed on a liberal diet, freely stimulated, and everything done which will sustain his strength. There should be free ventilation of his room, and the air and sputa must be thoroughly disinfected. Tonics, as iron and quinine, should be administered.

Turpentine administered by inhalation and given internally at the same time (♏x-xv four or five times a day in emulsion) appears to produce the best effect. Terebene has also been used, as has carbolic acid, internally and by inhalation in the form of spray.

Tuberculosis.

What is tuberculosis?

Tuberculosis is an acute or chronic disease, usually the latter, caused by the deposition, softening and breaking down of tubercle in the lung, and the ultimate formation of cavities in those organs, and characterized by fever, accelerated pulse, cough, emaciation, and other symptoms of hectic fever, attended with signs denoting consolidation of lung structure, and afterwards of the formation of cavities.

What is the pathology of tuberculosis?

Tuberculosis is a specific disease. A tubercle is one of the infective granulomata, and causes, is not caused by, inflammation. It is preëminently a disease of the lung, although it may be found in any organ of the body, usually, however, associated with infection of the lung also.

Like all other low-grade tissues of new formation, it has a tendency to undergo caseous degeneration, to soften and break down. The neighboring tissues become inflamed, and, softening in their turn, break down also, and thus cavities are formed.

Tubercle is due to the presence of the bacillus tuberculosis, which is found in the tubercular masses, in the breath and in the sputum of those suffering from this disease.

This is a rod-shaped body, blunt at both ends, and absolutely motionless, which varies from $\frac{1}{1200}$ to $\frac{1}{3500}$ of an inch in length, and produces spores. It cannot be seen, even by the microscope, without having been previously stained.

Under the microscope a tubercle is seen to consist of a giant cell, which contains the bacillus, and which is surrounded by a reticulum consisting of delicate filaments interlacing with each other and containing within their meshes numerous embryonal cells, fatty globules and broken-down connective tissue.

It seems positively settled that without the presence of the bacillus tuberculosis there can be no tubercle, and yet if this be the cause of the disease, it is difficult to understand how heredity can play a part in its production, as it undoubtedly does. It certainly acts as a

carrier of infection, and its presence, when recognized under the microscope, is an invaluable aid to diagnosis.

What are the causes of tuberculosis?

The ever-present cause appears to be the bacillus tuberculosis. It is strongly hereditary; is slightly infectious, in the sense that a person, as a nurse, constantly breathing the atmosphere of a sick room impregnated with the bacillus, may finally contract the disease. Anything which lowers the vitality aids in developing the disease: thus it occurs in those who lead a sedentary life; in persons overworked in mind and body, and often develops at the close of debilitating diseases, especially the acute fevers. Perhaps in those who inherit a predisposition to this disease, and in those whose vitality is otherwise depressed, the bacillus or its spores find a suitable nidus for their development, while in a healthy individual the tissues possess sufficient vitality to resist the disease, or the bacillus, not finding suitable pabulum, is cast out of the system even after it has entered the body.

Acute Tuberculosis.

What is acute tuberculosis?

It is an acute disease, characterized by fever, sweats, rapid pulse, great emaciation, cough and dyspnœa, running a rapid course, and due to the deposition from the blood of miliary tubercle, diffused throughout the lungs and other organs of the body.

It is usually called *galloping consumption.*

Although not so common as chronic tuberculosis, it is by no means unfrequently seen.

What is the pathology of acute tuberculosis?

The pathology is the same as that already discussed when speaking of tubercle. The bacillus tuberculosis is deposited from the blood in lungs, bowels, brain, etc., and usually produces diffused miliary tuberculosis.

It is generally a disease of childhood or of young adults.

What are its symptoms?

There is some cough, which is usually slight, and not at all violent.

Emaciation takes place rapidly, and is out of all proportion to the

violence of the cough. Fever is always present, the temperature being high and irregular, varying much in the course of twenty-four hours, a point of diagnostic importance.

Profuse colliquative sweats are seen, which are out of all proportion also to the violence of the cough. The pulse is irritable and very frequent, and there is great dyspnœa.

Delirium, photophobia and other symptoms of cerebral disorder are present, even in those cases where there is no deposit of tubercle in the brain. These symptoms are then probably the result of sympathetic disturbances, possibly induced by the rapid circulation, the fever and the exhaustion.

Hemorrhage is not of frequent occurrence, although the disease sometimes begins with hæmoptysis. Diarrhœa is sometimes present, and may be severe. There is often some pain in the chest. The disease runs a rapid course, and usually terminates fatally from exhaustion. Occasionally the acute process is arrested, and the disease proceeds as chronic tuberculosis.

What are the physical signs?

The physical signs are generally very obscure at first, being simply those of diffused bronchitis. Later in the disease, signs of consolidation are usually present at the apices of the lungs, followed by those of softening and the formation of cavities, as in chronic tuberculosis.

How is this affection recognized?

This disease is diagnosed by the fever, pulse, emaciation, sweats and dyspnœa, all of which are decidedly out of proportion to the cough; and later by the physical signs, and the presence of the bacillus in the sputum.

Differential Diagnosis.

From Acute Bronchitis.—In the latter disease there is not the rapid wasting, nor the high temperature, rapid pulse, profuse sweats and great dyspnœa which are found in the former, nor do the physical signs ever point to consolidation, nor to the formation of cavities.

What is the prognosis?

Recovery sometimes takes place, calcareous degeneration occurring in the softened tubercle, the caseous process thus being arrested.

Or the acute symptoms may subside and the disease continue as an ordinary case of chronic phthisis. The disease usually, however, terminates in the death of the patient within a very short period, one case being reported in which death occurred eleven days from the onset of the disease.

What is the treatment?

The indications for treatment are to reduce the temperature and hectic, to nourish the patient and to treat those symptoms which may require it. The patient should, therefore, have a light, easily-digested, nourishing diet, but he should not be overfed, as this tends to increase tissue waste. Quinine and digitalis are preëminently the remedies needed in these cases, to which opium may be joined, as in Niemeyer's pill, in case there is much irritating cough.

Chronic Tuberculosis.

What are the synonyms of this disease?

It is called tubercular phthisis, tubercular disease of the lung, and consumption.

What are the causes and pathology of this disease?

It is strongly hereditary, and is predisposed to by a sedentary life. In some rare instances it is unquestionably due to breathing the emanations of a patient suffering from the disease, as in the case of a husband nursing a wife. The pathological cause is tubercle, which has already been discussed.

What are its symptoms?

The disease consists of three stages, *the stage of deposit*, *the stage of consolidation*, and *the stage of the formation of the cavity.*

First Stage.—The disease may start as a neglected cold, or as a slight, hacking cough, with loss of flesh and health, and a certain amount of digestive disturbance coming on gradually; or as a slight cough followed by hemorrhage; or simply as loss of flesh. No matter how the disease begins, the circulation is always quickened.

Second Stage.—The cough increases, expectoration is more profuse and is purulent or nummular, and emaciation progresses more

rapidly. There is a tendency to diarrhœa, and irritative fever in the latter part of the day. The heart is irritable, and there is a quick, irritable pulse, the frequency of which continues even when the patient is free from fever.

Night-sweats now occur, and the patient is much exhausted in the morning; the appetite is poorer and poorer; the diarrhœa is apt to increase; hemorrhage may occur at any time, and the respiration is accelerated.

THIRD STAGE.—The cough, weakness and dyspnœa increase, and the emaciation is more marked; the irritative fever and night-sweats are more pronounced; the patient is hopeful; and the mind is clear. Hemorrhages are rare in this stage of the disease. In cleanly persons, a red line is seen on the gums, which appear to be retracted from the teeth, making the latter look longer than natural. This is called the *gingival line*, and as a diagnostic point is of some value. The *nails become clubbed* at the ends, and bluish in color from defective aëration of the blood. Swelling of the feet occurs as a late symptom. Occasionally acute cerebral symptoms appear with pain in the head, delirium, irregular pulse, and the patient (usually a child) dies of tubercular meningitis; or acute pleurisy may occur; or pneumonia be intercurrent; or pneumothorax supervene and cause death.

What are the complications?

(1) *Laryngeal phthisis.* Always a grave complication. It usually begins as a simple catarrhal laryngitis, with thickening and swelling of the mucous membrane, tubercular infiltration afterward taking place. The infiltrate spreads downward to the cartilages and ulceration occurs. The symptoms of these cases are, great difficulty in swallowing from involvement of the epiglottis; frequent, harsh, paroxysmal cough, and change in the character of the voice.

(2) *Diarrhœa.* This is sometimes only a catarrhal trouble. At other times it is caused by tubercular infiltration of the walls of the bowel, of the peritoneum, or of the mesentery glands. An ulcerative process sets in and the patient's life is much shortened. In all cases of tuberculosis where there is persistent diarrhœa resisting ordinary treatment, tubercular ulceration should be suspected.

LESS FREQUENT COMPLICATIONS are

(3) *Tubercular meningitis*, especially seen in young people.

(4) *Localized pneumonia or pleurisy* occurring after exposure.

(5) *Pneumothorax.*

What are the physical signs?

IN THE FIRST STAGE, OR THAT OF EXUDATION OR EARLY INFILTRATION, the signs are limited chiefly to the apex of the lung, usually to that of the left lung.

On inspection there is a want of expansion of the upper part of the lung, which is flatter than that of the opposite side.

On percussion there is a slight impairment of the normal resonance in the same situation (the left apex is normally somewhat clearer than the right).

Respiratory percussion renders the sound relatively more dull than before.

On auscultation a few fine subcrepitant or crepitant râles are heard, limited to the left apex. There is prolonged expiration and feeble or harsh respiration. There is slight increase of the *vocal resonance.*

On palpation a want of expansion is detected at the left apex, and the *vocal fremitus* is increased in this situation.

SECOND STAGE, OR THAT OF COMPLETE INFILTRATION. When this stage is reached in the left lung, infiltration has usually commenced at the other apex also, hence the signs of the first stage will now be detected in the second lung.

Percussion will show decided dulness at the left apex.

On auscultation vesiculo-bronchial breathing is heard, and bronchophony is marked; crackling râles are also heard, not as much limited to the left apex as they were in the earlier stage.

On palpation there is increased vocal fremitus.

THIRD STAGE, STAGE OF SOFTENING AND FORMATION OF CAVITIES. There is absolute dulness *on percussion.*

On auscultation large, moist râles and vesiculo-bronchial breathing are heard, but are not so limited in extent as in the second stage.

Over a cavity, percussion produces a vesiculo-tympanitic resonance. On auscultation there is cavernous breathing, cavernous voice and gurgling.

If the cavity is large, percussion will elicit an amphoric note, and on auscultation there will be an amphoric respiration and voice.

How is chronic tuberculosis diagnosed?

In the early stages the disease may be recognized by the slightly elevated temperature; the loss of flesh; the family history; the quickened pulse; the impaired resonance on percussion, limited to the left apex; the prolonged expiration, and the few fine râles heard in the same situation, and the microscopical examination of the sputum in which the bacillus tuberculosis can be seen.

If there are any physical signs limited to the apex of the lung, with loss of flesh, tubercle probably exists.

Later in the disease the marked physical signs at one or both apices, the fever, sweating and other characteristic symptoms, render the diagnosis clear.

Differential Diagnosis.

(1) *From chronic bronchitis*, tuberculosis is differentiated by the dulness and other signs limited to the apex, and by the deterioration of health.

(2) *Lingering consolidation after pneumonia* usually affects the lower part of the lung, is preceded by the acute disease; the health is not as much impaired, and there are no bacilli in the sputum.

(3) *Bronchial dilatation.* In these cases the general health is good, there is no fever, no hurried respiration, no bacilli in the sputa, and the cavities due to the dilated bronchi are in the posterior and at the lower part of the lung. There are diffused râles over both sides of the chest. On percussion the chest is resonant, the note being but little changed from that of health. It is not dull here and tympanitic there, as in the case of the tubercular cavities. There is, therefore, a disproportion between the physical signs and the symptoms, which is not found in phthisis.

(4) *From chronic malaria.* In these cases the absence of the physical signs would exclude phthisis. Phthisis should, however, always be sought for where there are recurring chills, fever and sweats conjoined with loss of flesh, before a diagnosis of chronic malaria is made.

(5) *From emphysema.* The hemorrhages, emaciation and other

symptoms, and the great difference in physical signs make the diagnosis clear.

What is the prognosis?

A certain proportion of cases absolutely recover. If these cases die from other diseases, the post-mortem examination shows little calcareous masses, usually highly pigmented, occupying the site of the former tubercle. If there is no hereditary predisposition, and the disease follows a cold (*pneumonic phthisis*), the prognosis is all the more favorable.

If the disease starts with a high pulse, soon followed by fever, it is generally a rapid case. Hemorrhages do not add to the danger without they are repeated and severe. If a case is marked by persistent evening temperature of 102°, the prognosis is bad, unless the temperature soon becomes normal again, which will indicate the arrest of the tubercular process.

Persistent diarrhœa, profuse night-sweats, swelling of the feet, etc., indicate a bad case.

When cavities are present the disease is generally very serious, although in rare cases they may cicatrize.

What is the treatment of phthisis?

The treatment of phthisis may be divided into the radical and symptomatic treatment.

(A.) Radical Treatment.

This is of the utmost importance.

(1) *The patient must lead an out-of-door life*, no matter where he lives.

(2) *The climate* should be such as will allow him to live in the open air. An equable climate, especially if dry, is best suited for nearly all cases.

Colorado is a good climate if there are no laryngeal complications and no excessive action of the heart. If the heart is irritable, the altitude of Colorado is too great. It is especially good in the early or the second stage of the disease.

New Mexico is also good for the same class of cases.

Southern California is better for those cases with laryngeal complications, or where there is an irritable heart, or where they do not bear our winters well.

South Carolina is dry and not very high.

Florida is a useful climate where there is much bronchial irritation and when it is desirable to increase the expectoration.

The Nile or Algiers are good climates when there is some bronchial irritation, and where altitude is not to be desired.

In summer, mountainous regions usually suit all classes of cases best, provided the altitude is not too great. The Adirondacks is suitable for many cases during the hot weather.

(3) *The patient should be warmly clad*, but not sufficiently so to produce perspiration. He should wash with *cold water* in the morning, bathing his neck and chest, and undergoing what is called a hardening process.

(4) *Food.*—He must have a plain but nourishing diet, and abundance of meat if it is well digested. If, however, the stomach becomes disordered, he must be placed upon a milk diet, gradually increasing the amount until he is able to take it in large quantities. The extract of malt, beer, etc., are foods, not medicines, in this disease.

(5) *Drink.*—The average consumptive should take small amounts of alcohol in the form of wine or whiskey, especially when he is leading a life in the open air. If he takes whiskey, let him have about half an ounce three times a day with his meals. If there is much irritability of the heart or a quick pulse and an irritable, nervous system, alcohol does more harm than good.

(6) *The patient may smoke in moderation*, if the heart is not irritable.

(7) Should the physician be consulted regarding the *marriage of a consumptive*, it is his duty to lay the question of heredity before both the contracting parties. Marriage itself does not affect the prognosis of the case. During pregnancy the tubercular process is retarded. After childbirth, however, it is apt to be hastened in its development. *No consumptive mother ought ever, under any circumstances, to nurse her child*, both for her own sake and that of the child as well.

(8) *Drugs.*—The best remedy is *cod-liver oil*, which should be given in doses of from f℥j–iv three times a day, stopping when the weather becomes warm, as it is then more apt to disagree with the stomach. It may occasionally be omitted with benefit, even in cold weather. It is better given pure, if the patient can take it, and

may be prescribed in malt, soda water, with whiskey, or with a few minims of ether, which for a time appears to aid its digestion, but cannot be taken for a long period.

Arsenic.—Fowler's solution may be given in small doses, ♏j–iij, or sodium arsenite, gr. $\frac{1}{40}$, three times a day. These may be given early in the disease and continued for a long time. They are especially useful where there is not much bronchial irritation.

Iodine.—In the form of Lugol's solution, or, if the patient is anæmic, as ferrous iodide, is very useful. If there is much bronchial catarrh, potassium iodide answers better.

The hypophosphites only act as an ordinarily good tonic, but have no specific effect.

The inhalation of compressed air is moderately good in the early part of the disease, but has no curative effect.

The same may be said of *oxygen inhalations.*

Inhalations of iodine and carbolic acid do some good in bronchial complications, or for the relief of fœtor where there are cavities, but never cure.

To kill the bacillus, disinfect the sputum with corrosive sublimate solution or chlorinated lime, and thus prevent the spread of the disease. As yet, no means has been invented to destroy the bacillus in the lung.

SYMPTOMATIC TREATMENT.

As this is a disease of nutrition, the more attention that is paid to the general health, and the less the symptoms are treated, the better for the patient.

Cough. If the cough is not excessive, if it does not prevent the patient from sleeping, the less it is treated the better. Should it be necessary to give some remedy to allay it, opium in some form or other has the best effect. Codeine allays the cough, and does not constipate as much as opium. It should be given in about double the dose of morphine. Diluted hydrocyanic acid (gtt. j) or the fluid extract of Prunus Virginiana are also useful. The diluted acids are also good as adjuvants to opium.

For the irritative fever, small doses of aconite or a combination of digitalis with quinine and opium should be given every three or four hours. Where the high temperature persists, antipyrine has been employed in doses of gr. v, repeated every hour, until twenty grains

are taken, or quinine may be given in doses of gr. x, repeated three times at intervals of an hour.

Night sweats. Do not keep the patient too warmly covered at night. A hot sponge-bath with alum or borax is sometimes of value. Zinc sulphate, gr. j every three or four hours, or ergot, or ergotine, gr. ij two or three times a day, or aromatic sulphuric acid, gtt. xv–xx, freely diluted three times a day, or most potent but most disagreeable in its after-effects, atropine, gr. $\frac{1}{60}$ at night, may be employed.

For the laryngeal complications. Iodoform has been successfully used in these cases, but the taste and smell constitute strong objections to its employment.

Cocaine in solution (four to eight per cent.), applied by a brush to the larynx or even to the epiglottis, affords great relief to the patient, and allows him to swallow without pain.

For loss of appetite and debility, the best remedies are quinine and ignatia.

For diarrhœa, if it is possible let it alone, and treat the disease. Should it continue and prove obstinate, bismuth, silver nitrate, cupric sulphate, or the mineral acids (as Hope's Camphor Mixture) may be combined with opium.

Pneumonic Phthisis.

What is this disease?

It begins acutely like pneumonia, but the consolidation lingers, and after a time tubercles form in the consolidated lung. After the deposit of tubercle, the *symptoms* and the *physical signs* are identical with those of tuberculosis, the signs being, however, in the lower part of the lung instead of at the apex. *The prognosis* is far better than that of tuberculosis, as the results of treatment, and especially the change of climate, often produce wonderful effects.

The treatment is the same as that of chronic tuberculosis.

Fibroid Phthisis.

What is the cause of this disease?

It occurs in stone-cutters, knife-grinders, miners, weavers, etc., from the inhalation of small particles of foreign substances floating in the atmosphere, due to their occupation. These produce a low grade of interstitial pneumonia which results in hypertrophy and hyperplasia of the connective tissue of the lung, which contracting compresses the air-vesicles. After a time the lung becomes the seat of secondary tubercular infection. Pleuritic adhesions may also form, causing contractions of that side of the chest; dilatation of the heart is apt to result from interference with the circulation of the lungs, and cardiac dropsy occurs. It is a late symptom. In some instances the disease is arrested before the lung becomes the seat of tubercular infection.

The symptoms are like those of tuberculosis, but the disease is much more gradual in its development.

Physical Signs.

There is sinking in of the chest wall on one side, dulness on percussion with areas of tympanitic resonance over dilated bronchial tubes. There is bronchial breathing over the contracted lung or signs of a cavity over the dilated bronchi. Later in the disease the signs of tubercle are present.

Prognosis.

This is a disease of long duration. It may develop into tuberculosis, or it may recover without tubercular infection.

Treatment.

What has already been said of the general treatment of tuberculosis applies to these cases as well. Potassium or ammonium iodide is also of use. Counter-irritation by repeated blisters over the chest are much more useful in this than in other forms of phthisis.

If there is much offensive expectoration, disinfectant inhalations, as iodine, creasote, carbolic acid, etc., should be employed, and ammonium carbonate should be administered internally.

DISEASES OF THE PLEURA.

Acute Pleurisy.

What is acute pleurisy?

It is a plastic inflammation of the pleura, characterized by a sharp pain in the side, dry cough, fever, and difficulty in breathing on account of the pain.

What is its synonym?

Acute pleuritis.

What is the cause?

Acute pleurisy may follow cold and exposure or traumatisms. It is often secondary, occurring during an attack of pneumonia or other lung disease, pericarditis, smallpox or other exanthemata, Bright's disease, rheumatism or other diathetic disease, or following blood affections, as pyæmia, snake bites, etc.

What is its pathology?

The inflammation may be localized or diffused. It is usually limited to one side. The pathology is similar to that of inflammation affecting the other serous membranes. The pleura is at first red and congested in spots, and the epithelial cells covering its surface undergo rapid proliferation, causing it to become opaque. This exudation of lymph may undergo changes common to other plastic exudations terminating in strong, fibrous adhesions which bind the opposing surfaces of the pleura together. The lung immediately under the inflamed area is generally slightly congested.

Instead of this exudation of lymph a serous exudation may take place, very little lymph being formed. This fluid may become gradually absorbed, and adhesions form at a later stage. In this way the dry stage (where there is little serum and much lymph) may end in adhesion, or may be followed by a moist stage (where much serum is exudated), which in its turn may be followed by adhesion. Or the fluid may remain as a chronic condition or may become purulent.

What are the symptoms of acute pleurisy?

The symptoms are obscure and occasionally may be entirely absent.

Pain.—There is usually sharp, lancinating pain, increased by coughing and by breathing, which is generally referred to the seat of the inflammatory action, but may be reflected to other parts and sometimes to the opposite side. When the fluid is poured out the pain lessens or disappears.

There is *shortness of breath*, due in the early stages to the pain, and later on in proportion to the amount of fluid effused, and caused by the resulting compression of the lung.

There is *dry cough*, or perhaps it is accompanied by a little frothy expectoration.

There is a certain amount of *fever*, usually preceded by a chill; temperature rarely being over 102°, the local temperature being higher on the side affected. The pulse is frequent and compressible, varying from 90 to 110 per minute.

The urine is suppressed or greatly diminished in quantity, especially when the effusion is taking place. The case lasts from ten days to two weeks and the effusion is rapidly absorbed, the patient convalescing or perhaps having permanent adhesions remaining. In other instances the effusion remains and the case becomes chronic.

What are the physical signs of acute pleurisy?

(1) The Dry Stage (*when the pleura is roughened by lymph*).

Inspection and palpation show that the chest does not fully expand on the inflamed side during inspiration.

On percussion the note is not materially modified.

On auscultation the vesicular murmur is weaker because the chest does not expand on that side.

The *vocal resonance* is not much, if at all, impaired.

A friction sound is heard both on expiration and inspiration. It is rough, superficial and creaking, is not influenced by cough, and is usually better heard in the lower part of the chest. It may cease when the side of the chest is strongly compressed below.

It ceases when liquid effusion takes place.

(2) In the Stage of Effusion, there is dulness on the lower part of the chest *on percussion.*

On auscultation there is great enfeeblement of the vesicular mur-

mur in the lower part of the chest, or it may be entirely absent, while in the upper part it is frequently exaggerated.

The *vocal resonance and fremitus* is also greatly diminished or absent.

Ægophony is heard at the edges of the effusion, while higher still the friction sound may be present.

These physical signs become more marked as the effusion becomes greater. The chest wall frequently bulges, and the viscera are often displaced; thus, if the right side is affected the liver is displaced downward; if the left side is affected the heart is forced toward the right side.

(3) In the Stage of Absorption. As the effusion is being absorbed the dulness lessens, the respiratory sound becomes stronger and stronger, the vocal resonance and fremitus gradually increase, the friction sound returns, and the viscera resume their normal position.

(4) *If permanent adhesions form*, the chest remains contracted on that side and feeble breathing is present for life at this point, while a chronic friction sound may, but generally does not remain.

How is acute pleurisy diagnosed?

In the dry stage it is recognized by the slight fever, the pain, the cough, but especially by the friction sound and other physical signs.

In the moist stage by the dyspnœa, the scanty urine, but especially by the feeble or absent respiratory sounds, the diminished or absent vocal fremitus or resonance, the absolute dulness over the seat of the effusion and the other physical signs.

Differential Diagnosis.

(1) *From pleurodynia.* It is differentiated by the absence of physical signs in the latter.

(2) *From intercostal neuralgia.* By the absence of fever and physical signs in the latter, and the fact that the pain is strictly limited to one or two of the intercostal nerves with a tenderness on pressure over their corresponding nerve points.

(3) *From acute pneumonia.* In the early stages of the latter disease the crepitant râle is heard, while in the dry stage of pleurisy the friction sound is present. In the second stage of both diseases there is dulness on percussion; but in acute pneumonia ausculta-

tion reveals bronchial breathing, bronchophony, and exaggerated vocal fremitus, while in acute pleurisy there is feeble or absent respiration with diminished or absent vocal resonance and fremitus.

What is the prognosis?

Prognosis is usually favorable if it is a primary affection. When it is secondary, the prognosis will depend upon the disease which it follows or complicates.

How should it be treated?

Morphine should be administered hypodermically to relieve the pain as soon as the patient is seen. *If it is the result of cold*, dry cups should be freely applied to the chest, or if the patient is strong and robust leeches or wet cups may be used; large jacket poultices afford much relief.

Internally, if the patient is strong and the pulse is full and bounding, tincture of aconite, gtt. j, may be given every ten or fifteen minutes, in neutral mixture or spirit of mindererus until it is reduced.

After effusion has taken place the aconite must not be given. The amount of liquid the patient takes should be much restricted. Active purgation should be resorted to, and diuretics, as digitalis or the acetate of potash, used. Small doses of concentrated solutions of the saline purgatives frequently repeated, aid materially in the absorption of the fluid.

When the effusion is being absorbed, or if it lingers, iodide of potassium or a combination of calomel with squill and digitalis are advantageous.

If the effusion is large or is increasing, or if there is an effusion in both pleural cavities, as sometimes occurs when it is secondary to the exanthemata or blood affections, the pleura should be aspirated and the fluid removed.

Chronic Pleurisy.

What are the causes of chronic pleurisy?

It may follow an acute attack, or, more commonly, a subacute attack, the symptoms of which have been latent, or it may result from a gradual accumulation of fluid in the pleural cavity, as is sometimes seen in chronic pneumonia.

What are its symptoms?

There are no symptoms of any moment, shortness of breath being the only positive one. The disease lasts for months, or it may be years. If it lasts for more than six months, the effusion is probably purulent (*empyema*). If the patient is not relieved, he will usually die in a year or eighteen months.

What is the prognosis?

About 17 per cent. of cases get well spontaneously, the pus being evacuated through a fistulous opening in the chest wall. As a rule, the cases of *empyema*, when untreated, die of exhaustion, worn out by the hectic fever, or from tuberculosis developing in the pleura or lung.

What is the diagnosis?

The diagnosis of chronic pleurisy is similar to that of pleuritic effusion, except in point of time. *Empyema* is diagnosed by the symptoms and physical signs of chronic pleurisy, with the irregular chills, fever, night-sweats, and rapid pulse of hectic fever. If in doubt, introduce the needle of a hypodermic syringe and draw off some of the fluid.

Differential Diagnosis.

From hydrothorax. This disease is a pleuritic effusion, double-sided, and being part of a general dropsy due to the cardiac or renal disease. The diagnosis is therefore easy.

How are chronic pleurisy and empyema treated?

If the effusion is serous, an effort should be made to cause its absorption. Very little liquid should be given to the patient, but diuretics, as Basham's mixture, and diaphoretics, with laxatives and occasionally active purgatives, should be freely administered. Blisters applied to the chest favor the absorption of the effusion.

If there is no sign of absorption within six or eight weeks the fluid may be removed by aspiration, the needle being passed into the cavity of the chest near the lower angle of the scapula, and nearer the upper than the lower border of the rib, so as to avoid injuring the intercostal arteries.

If the symptoms lead to a diagnosis of empyema, the pus should be evacuated, and quinine, iron and salines administered. It is bet-

ter to evacuate the pus by a free incision than by aspiration. The cavity of the pleura should be thoroughly washed out with boiled water, creolin, boric acid or other antiseptic. A drainage-tube should be inserted and the parts dressed antiseptically, the disinfectant washings being repeated two or three times a week until the formation of pus is arrested, when the drainage-tube is withdrawn and the wound allowed to heal. Adhesions usually take place and adhesive pleurisy results.

Pneumothorax.

What is pneumothorax?

It is an accumulation of air in the pleural sac, which, by causing irritation, leads to inflammation and serous exudation, and is characterized by sharp pain, dyspnœa and peculiar physical signs.

What is its ætiology?

The air may find an entrance through the chest wall by a wound or it may pass through from the lung itself, due to the broken rib, to the rupture of the air-vesicles, as in emphysema, or in gangrene; but the most frequent cause is the softening and breaking down of tubercle on the surface of the lungs.

What is its pathology?

The air which has gained entrance into the pleural cavity frequently causes a pleuritic effusion, and is then sometimes called *hydro-pneumothorax*. The air gradually becomes absorbed if the opening through which it enters closes, and a case of chronic pleurisy results.

What are the symptoms?

This disease is very latent. There is usually dyspnœa, suddenly developed, generally intense, associated with sudden and sharp pain and irritating paroxysmal cough; but dyspnœa may be the only symptom.

What are the physical signs?

On inspection the chest is distended, the intercostal spaces are

effaced, the thorax upon the affected side bulges like the side of a barrel. The respiratory movement of that side is absent, and the apex beat is often displaced if the left side be affected.

On percussion over the air, a tympanitic or amphoric note is produced. Over the fluid it is dull.

On *auscultation*, if the opening in the lung is closed, absolutely no sound is heard. If, however, it is open, we have amphoric or metallic breathing, amphoric or metallic voice and frequently metallic tinkling is heard from drops of fluid or the bursting of small bubbles. If a silver dollar is pressed firmly against the chest over that portion of the pleura which contains air, and is struck lightly by another piece of silver while the examiner's ear is applied to the chest above the line of the fluid, a clear metallic note like a silver bell will be distinctly heard, if the opening has not closed. If he shifts his ear placing it over the fluid and the dollar is again struck it does not resound, but gives a dull sound, such as would be produced by striking two pieces of lead together (Bruen).

A succussion sound is heard upon shaking the patient. This is very significant, as it requires for its production the presence of air as well as of fluid in a large cavity. The viscera are, of course, displaced, the liver being displaced downwards if the disease affects the right side, or the heart being pushed toward the right, if the left is the seat of disease. After the air is absorbed the physical signs of a pleuritic effusion remain.

What is the prognosis?

When the result of injury the patient usually recovers, and even when it is caused by tubercle, recovery from the acute disease may occur.

What is the treatment?

If there is much pain, morphine should be given hypodermically. For the relief of the dyspnœa, morphine, cannabis indica, the inhalation of ethyl iodide or the administration of aromatic spirits of ammonia may be used. If the dyspnœa is extreme, as a last resort the fluid and air may be removed by aspiration. After the air has been absorbed, the case should be treated as one of chronic pleurisy, with diuretics, as Basham's mixture, diaphoretics, laxatives and plenty of good food and stimulants.

(E.) DISEASES OF THE CIRCULATORY SYSTEM.

What are the physical signs elicited on the examination of a normal heart?

On inspection and palpation the impulse, caused by the apex of the heart striking the chest wall, is seen and felt between the fifth and sixth ribs, occupying only one intercostal space, and about an inch to the inner side of the nipple line.

On percussion the portion of the heart uncovered by lung tissue can be ascertained by the dulness elicited. It extends from about the fourth intercostal space below the middle of the clavicle, downwards for nearly two interspaces (sometimes normally even a little more), and is from 2 to 2½ or even 3 inches in a transverse direction. This area varies with inspiration and expiration.

On auscultation *two sounds* are heard with intervening pauses. *The first sound* is loud, dull, heavy and booming; it is caused by the contraction of muscular fibres, the apex-beat against the chest-wall, and the closure of the auriculo-ventricular valves; the timbre of the first sound is owing to the muscular contraction, and will vary as the latter is more or less powerful. After a very short pause the *second sound* is heard. It is a short, sharp click, not so loud or so prolonged as the first sound, and is due to the sudden closure of the aortic and pulmonary semilunar valves. It is heard in greatest perfection at the base of the heart, while the first sound is better heard at the apex.

How are these signs altered by disease?

(I) Inspection.

The cardiac impulse may be displaced if the heart is pressed or drawn to one or the other side by pleuritic effusions or adhesions. *It may be more forcible* and occupy more intercostal spaces than usual in hypertrophy, or from strong emotion. *It may be feeble and wavy* in dilatation, weakness, debility or pericardial effusions.

(II) Palpation.

Palpation of the impulse shows similar changes to those described under the head of inspection.

(III) Percussion.

The area of percussion dulness may be increased in hypertrophy, when the enlargement usually occurs to the left, or in dilatation when the enlargement occurs to the right.

(IV) Auscultation.

The cardiac sounds may be variously modified by disease. *The first sound is increased in volume and strength* in hypertrophy or in a heart which is over-acting from excitement or other temporary stimulus. *It is diminished in force* in dilatation, weakness, debility or pericardial effusion. *The second sound may be accentuated* in all states of high arterial tension.

FEEBLE HEART SOUNDS.

First sound, enfeebled.	Advanced fatty degeneration; dilatation. Neurosal influences. Changes in, or diminished vasomotor tension.
Second sound in pulmonary artery or aorta, enfeebled.	The above conditions. Also reduced vasomotor tonus in aorta or pulmonary artery, whether from feeble cardiac systole, or owing to degenerative or neurosal causes.

Pericardial effusion is a prominent cause of the enfeeblement of the heart sounds.

ACCENTUATED HEART SOUNDS.

First sound, accentuated.	Mitral obstruction, possibly aortic obstruction and regurgitation. Degenerations or dilatations of the ventricle.
Aortic second sound, accentuated.	Chronic forms of Bright's diseases. Palpitation when the heart muscle is normal. Hypertrophy of the heart. Atheroma; increased vasomotor tonus from neurosal causes.
Pulmonary second sound accentuated.	Circulatory obstruction in the lungs. Pleurisy with effusion. Emphysema. Mitral valvular disease, especially mitral obstruction.

(Bruen's "Physical Diagnosis.")

Sometimes two first or two second sounds may be heard, which merely shows perverted nerve action.

What adventitious sounds may be heard when the heart is auscultated in disease?

The adventitious sounds are classified either as pericardial or endocardial.

(A) Endocardial Murmurs.

These are chiefly of a blowing character. They vary in kind,

some being rough and some soft; some musical and some not, and others having no distinguishing mark except their abnormality. The chief point of importance in connection with them is the time at which they occur, in other words, what sound of the heart they supersede, and their point of greatest intensity. Should they be masked by the respiratory sound, they can be better heard if the patient is made to hold his breath. The sound is loudest at the point where it is produced, and travels in the direction of the blood current, hence is sometimes much more distinct at a little distance from its point of production in the direction in which it is transmitted.

Endocardial murmurs may be divided into three classes.

(1) *Organic Murmurs.*

These murmurs are produced when there is an altered relation between the blood and the openings of the heart. The great characteristic of these murmurs is their persistency and harshness or roughness.

(2) *Temporary Murmurs.*

These are of a functional character and originate from a temporary improper action of the valves. This class of murmurs is usually most marked over the left ventricle, and they are soft in character, usually systolic, and better heard at the base than elsewhere. They may be produced by excitement, and, often, by excessive exercise. If there is any doubt whether a murmur is functional or organic, it is better to let the patient rest in a recumbent position for some time and examine him again, when, if it is functional, it will very often have disappeared.

(3) *Blood Murmurs.*

In profound states of anæmia a murmur is frequently heard over the base of the heart, systolic in point of time, soft and blowing in character and transmitted into the arteries of the neck. It depends upon the altered condition of the blood. A venous hum is frequently heard in these cases in the large veins of the neck.

(B) Pericardial Murmurs.

In health no sound is produced in the pericardium. If it is roughened by inflammatory exudation, a *friction-sound* will be produced as the two surfaces rub against each other during the movement of the heart. Sometimes these murmurs have a whistling sound and simulate those of endocardial origin, but the pericardial friction sound

differs in being strictly localized, never being transmitted in the direction of the blood current. They follow the movements of the heart, coming after, but never replacing the cardiac valve sounds, and they frequently change their seat between the visits of the examiner. These murmurs are distinguished from pleuritic friction sounds by making the patient hold his breath, when, if it is of pleuritic origin, the murmur will cease, but if of pericardial origin, it will continue.

DISEASES OF THE HEART.

Cardiac Hypertrophy.

What is this disease?

It is an increase in the muscular tissue of the heart, characterized by a forcible impulse; a loud, booming first sound; a strong, full pulse, distention of the arteries, and a tendency to active congestion of various organs.

What are its causes?

It may be produced by a long continued obstruction to the flow of blood through the arteries in any part of the body whatever; thus a narrowing of the valves of the heart itself will eventuate in hypertrophy. A dilatation of the orifice of the valve allowing regurgitation will also often result in the same affection. Any disease impeding the flow of blood through the lungs, as emphysema, will have the same effect. States of high arterial tension, such as are seen in chronic Bright's disease, will also produce it.

It may result eventually from excessive functional disturbances where the heart is continually excited and in a state of irritation, as is seen in those who indulge to excess in the use of tea, coffee or tobacco; persons who dance to excess; in athletes, soldiers on forced marches, porters, women with chronic uterine diseases, or as a result of venereal excesses or of masturbation.

What are the varieties of hypertrophy?

(1) *Simple hypertrophy* is a simple increase in the thickness of the walls of the heart, the size of its cavity remaining normal.

(2) *Concentric hypertrophy* is an increase in the thickness of the muscular wall at the expense of the size of the cavity. This is a very rare form of cardiac hypertrophy.

(3) *Eccentric hypertrophy.* This is an increase in the thickness of the cardiac walls with an enlargement of the cavity of the heart. It is also called *dilated hypertrophy*, or *hypertrophy with dilatation*, and is usually seen in the latter stages of all valvular diseases of the heart.

What are the symptoms of cardiac hypertrophy?

The symptoms are indicative of a too active circulation. The pulse is fuller and firmer than in health. The temporal arteries throb, as do also the carotids; the face is florid and epistaxis is common. Vertigo, dull headache, ringing in the ears, disturbed sleep, jactitation during sleep, sometimes symptoms like those of congestive apoplexy may be seen. The conjunctiva are injected; the patient sees objects floating before his eyes; there is often a dry cough and dyspnœa on exertion. These symptoms are usually aggravated during digestion.

Cardiac Symptoms.—The cardiac symptoms are not prominent. Attacks of palpitation are not common, though incessant exertion will sometimes occasion them. Pain is usually absent, and when present, is dull and not very prominent. There is often a tumefaction of the thyroid gland and sometimes prominence of the eye. In these cases, however, the hypertrophy probably forms a part of the disease, and is not a cause primarily of the symptoms.

What is the pathology?

The pathology consists in a true hypertrophy of the muscular and fibrous tissues of the heart. The hypertrophy is usually limited to the left side of the heart, the left ventricle especially suffering.

What are the physical signs?

On Inspection the præcordial region has often an appearance of fullness or of prominence. The impulse is distinct, diffused and powerful.

On Palpation the impulse is felt diffused in several intercostal spaces, is usually lower and more to the left, is forcible and powerful.

On Percussion there is an increase in the area of cardiac dul-

ness, especially upon the left of the sternum and to the outer side of the nipple line.

On Auscultation there is no murmur, the first sound is duller, longer, heavier and more powerful than normal. The second sound is unchanged, or, it may be, accentuated.

What is the diagnosis of this disease?

The disease is recognized by the evidences of a very strong, active circulation; by the strong, forcible and extended impulse; by the increased area of percussion dulness; by the accentuation of the cardiac sounds, and by the absence of a cardiac murmur.

Differential Diagnosis.

From Functional Disease.—The increased area of percussion dulness in the former serves to differentiate these affections.

What is the prognosis?

If the patient is young and the hypertrophy not very great he may recover entirely.

If the disease comes on at puberty, following functional disorder, and is not very great, nor associated with dilatation, an increase of the disorder may be prevented.

The unfavorable cases are those which are caused by organic disease, as chronic Bright's disease or uterine affections. Hypertrophy, when occurring as compensatory to valvular disease of the heart, is of advantage to the patient, provided it be not too excessive.

How is it treated?

It is better to reduce the amount of food which the patient takes, as in this way excessive nutrition of the heart is prevented. The patient should not be starved, however, as this appears to increase the hypertrophy. The diet must be unstimulating, consisting of milk, vegetables and fish. He must eat very little, if any, meat; live moderately and avoid stimulation, tea, coffee and tobacco. He may drink cocoa. Moderate exercise may be taken, but all active exertion, as running, jumping, boat-racing and athletic games must be sedulously avoided. He should rest in the recumbent position for several hours each day.

An occasional laxative must be given, particularly a saline, and the action of the skin and kidneys maintained.

SPECIAL AGENTS.

Small doses of aconite, (tincture, gtt. j–ij) should be given four times a day, and continued until it produces some cardiac impression, when the dose may be reduced. This remedy should be administered for months.

Veratrum viride (tincture, gtt. v) may be given instead of the aconite. If it nauseates, a small amount of tincture of ginger, or of brandy given with it, will frequently overcome this effect.

Ice applied intermittingly over the heart also is of use. These are the best remedies for combating this disease.

Cardiac Dilatation.

What is cardiac dilatation?

It is a disease in which one or more cavities of the heart are increased in size, out of proportion to the development of its muscular wall, and is characterized by a feeble action of the heart, accumulation of blood in the veins, œdema or general dropsy and exhaustion.

What is the pathology of this affection?

Occasionally a partial dilatation is seen, often called *aneurism of the heart.* Usually, however, there is a general dilatation in which all the cavities share, being greater and more serious, however, on the right side of the heart, and affecting the ventricles more than the auricles. The muscular tissue is anæmic, and frequently undergoes degeneration. The orifices of the cavity often share in the general dilatation, and in consequence the blood will be allowed to regurgitate.

What are the causes of this affection?

Acute dilatation often occurs as a temporary condition, accompanying fevers, and passes off as the fever subsides.

Chronic dilatation is more common. It affects persons in feeble health, especially where there is some strain on the heart, as occurs when the circulation is obstructed in the lungs, liver or uterus, which, in a strong, robust person, would lead to hypertrophy.

In children of feeble parents, dilatation sometimes occurs at

puberty. In persons of a gouty diathesis it may occur late in life. Organic valvular diseases usually terminate in dilatation.

What are the symptoms?

The symptoms all indicate a feeble circulation. The veins are turgescent; the skin is pale or cyanosed. There is puffiness of the ankles; a languid, feeble pulse; dull headache; vertigo, despondency, and oppression in breathing, sometimes amounting to severe dyspnœa, occurring in paroxysms, and usually at night. Some cough is often present from passive congestion of the lungs or larynx. The liver is slightly enlarged, congested, and somewhat tender; the conjunctiva is yellow and the tongue coated. The kidneys are congested, the urine is scanty and sometimes albuminous. Palpitation is frequent, and a certain amount of uneasiness is often complained of in the cardiac region as the disease advances. Not only is there œdema, but anasarca, and dropsical effusions in the internal cavities come on late in the disease. The patient becomes weaker and weaker; disorders of the digestion occur from interference with venous circulation, and he finally dies of exhaustion or of cardiac paralysis.

What are the physical signs?

On inspection, the veins of the surface, especially those of the right side of the chest, and frequently those of the abdomen, are dilated. The cardiac impulse is indistinct, wavy and diffused.

On palpation, the pulse is fluttering, feeble, and extended, but uncertain.

Percussion shows an increased area of cardiac dulness, especially in a transverse direction, and usually extending to the right of the sternum.

On auscultation the first sound is feeble, short and indistinct; the second sound is sharp and valvular. No organic murmurs are present without there is valvular disease in addition to the dilatation.

What is the diagnosis of this disease?

Dilatation is recognized by the feeble health, the symptoms and signs of feeble circulation, the increased area of cardiac dulness, and the dropsy.

Differential Diagnosis.

(1) *From hypertrophy.* In dilatation there is dropsy, feeble heart

and pulse, pale face, distended veins, cold extremities, fluttering impulse, and a weak first sound.

(2) *From hypertrophy with dilatation.* Simple dilatation can be differentiated from that occurring in hypertrophy by the weakness of the cardiac impulse, and the dropsy, which is never present as long as the hypertrophy compensates for the dilatation.

What is the prognosis?

The patient may be very much benefited by treatment, but cannot be cured.

How should it be treated?

Persons suffering with feeble heart should avoid sudden excitement, as this, by putting too much strain upon a weakened heart muscle, might result in cardiac paralysis.

The diet should be nourishing, but easily digested. A meat diet is of advantage. A moderate amount of wine taken with meals and a little whiskey taken at bedtime, are of much use. He should be warmly clothed; have some regular systematic exercise, not too severe, and should avoid all strain of mind or body.

Special Agents.

Digitalis is of preëminent value in cardiac dilatation. It strengthens the muscular fibre, at the same time acting as a diuretic. It is best given in infusion (f℥j *t. i. d.*), or the powder, fluid extract or tincture may be employed. It must be discontinued temporarily every now and then, so as not to reduce the frequency of the cardiac beat too much, for the pulse will continue to sink for some hours or days after the medicine has been withdrawn. It should therefore be intermitted for a week or two at a time, during which period strychnine (gr. $\frac{1}{30}$) may be substituted for it.

If digitalis causes nausea or acts almost entirely upon the kidneys, or if it is too slow in manifesting any effect, *strophantus*, or its active principle, *strophantin* (gr. $\frac{1}{100}$–$\frac{1}{60}$), may be used in its place, and is an admirable heart tonic. It possesses very little, if any, diuretic properties.

Sparteine (gr. ¼–ij of the sulphate) has a similar action, and may be used as a substitute for either of these drugs.

Adonidin (gr. $\frac{1}{10}$ in pill) acts on the heart like digitalis, but is not nearly as serviceable as strophantin. It frequently causes headache.

Atropine is a valuable cardiac stimulant for temporary purposes,

but cannot be used for any length of time, on account of its other effects.

Convallaria may be employed (fluid extract, ♏v–x), but cannot be used as a substitute for digitalis, possessing nothing like its power over the heart, and having no diuretic action whatever.

In addition to the above special remedies, an occasional purgative should be used, and dry cups applied over the lung, particularly if cardiac dyspnœa is present, when the carbonate of ammonium or the aromatic spirits may be given at night.

For Dropsy.—Dry cups should be applied over the kidneys frequently, and mild diuretics taken, as cream of tartar, small doses of Rochelle salts, etc. Free purgation should also be used if the patient is not too weak, and at any rate there should be a daily movement from the bowels.

Caffeine citrate, in doses of gr. ij, is an excellent diuretic, and at the same time a cardiac stimulant. Cocaine, in doses of gr. ss, may be employed for the same purpose.

Fatty Degeneration of the Heart.

Define fatty degeneration of the heart.

It is a very common disease, characterized by feeble action of the heart, shortness of breath, and a tendency to venous stasis, with attacks of vertigo and other signs of a weak heart, and consisting of a fatty infiltration and transformation of the muscular fibres of the heart.

What is its cause?

It is found in debilitated persons; often in drunkards; coincident with fatty liver; in prolonged anæmia; in poisoning by phosphorus; in diseases of the coronary arteries, and in elderly subjects.

What is its pathological anatomy?

There is not only fatty infiltration, but granular and fatty degeneration of the cardiac muscle, which is pale, soft, flabby, easily torn, and of a yellowish color. Under the microscope, oil globules are seen, and the muscular striæ are indistinct.

What are the symptoms?

The symptoms are those of weak heart and circulation. There is a strong tendency to sigh, and the patient easily gets out of breath, suffering severely from dyspnœa and from palpitation upon exertion. Post-sternal pain is often complained of, which is sometimes severe and persistent in character, and even associated with a little tenderness. The pulse is weak and irregular. It may be fast or slow, but is always more or less irregular and compressible. The feeble circulation causes various disturbances, functional and organic, in other organs; thus, vertigo, and even, occasionally, epileptiform seizures may be present. Ulcer of the stomach may occur from malnutrition of that organ, and various fatty degenerations are seen, due to the weak circulation and to the atheromatous changes which occur in the coats of the vessels. An *arcus senilis* (a white ring at the corneo-sclerotic junction, due to fatty degeneration of the tissues) is often seen, and though it may be found without fatty degeneration of the heart, and *vicê versâ*, yet, when present in connection with the other symptoms, it is strong presumptive evidence of this affection. *Cheyne-Stokes respiration* is sometimes seen, consisting in ascending and descending breathing. The respiration becomes slower and slower, until it apparently stops altogether; then, after a pause, it begins again, at first very slowly, becoming faster and faster, until the breath is a succession of quick shrieks, when it becomes slower again, and so on. The respiration is not only decreased and increased in frequency, but also in depth.

What are the physical signs of this disease?

On palpation, there is a weak cardiac impulse.

On auscultation, the first sound is toneless and feeble, sometimes almost inaudible.

Other signs of weak circulation are present, but the feeble impulse and the toneless first sound are the most characteristic.

What is the diagnosis?

The disease is recognized by the symptoms of feeble circulation; by the patient's age, or other cause; by the toneless first sound and feeble impulse, associated with other signs of failure of the general or of the portal circulation.

DIFFERENTIAL DIAGNOSIS.

From dilatation. In the latter disease there is an increase in percussion dulness, and dropsy, both of which are rare in fatty heart unless dilatation is superadded.

What is the prognosis?

The prognosis depends upon the kind of life led by the patient. The danger being, first, from rupture of the heart due to over-exertion, and second, from paralysis of the heart from over-excitement.

What is the treatment?

These cases must rest or die. They *must* tone down their lives, taking very little exercise and avoiding all gymnastics. Their time must be devoted to taking care of themselves, and, if possible, they should give up all business and especially should they not occupy themselves actively with politics.

The diet should be nourishing, and a little wine or whiskey should be taken at meal-times.

They may take cold baths if they are accustomed to them and react well. By these hygienic measures life may be prolonged for many years.

The medicinal treatment consists in the administration of tonics, as Basham's mixture with strychnine.

Persons with fatty heart should never take chloroform, nor should they take chloral, except very cautiously and in extremely small doses.

If digitalis is given at all, it should only be used for a temporary purpose and with extreme care, as the forcible contractions which it causes may lead to rupture of the heart. Convallaria, caffeine or cocaine may be used as cardiac stimuli, but strychnine is probably the best cardiac tonic for continued use.

For the dyspnœa (*so-called cardiac asthma*) ammonium carbonate or Hoffman's anodyne or morphine hypodermically give the most relief.

Endocarditis.

What is endocarditis?

It is an inflammation of the lining membrane of the heart, which may be either acute or chronic, the latter usually merging into valvular disease of the heart.

Acute Endocarditis.

Describe acute endocarditis.

It is an acute inflammation of the endocardium, attended with plastic exudation and is characterized by disturbed cardiac action, shortness of breath, nausea and vomiting, cough and fever, terminating in permanent alterations in the cardiac valves or the orifices which they guard.

What are its causes?

It is usually caused by some alteration in the blood, as rheumatism which often results in this affection. Bright's disease also is a common cause, acting upon the blood in a similar manner. Any pyæmic condition of the blood may produce this disease. It occasionally follows blows on the chest.

What is the pathological anatomy of this disease?

The inflammation usually takes place in the neighborhood of the valvular outlets, particularly on the left side, fastening itself upon the mitral or aortic valve-leaflets. There is at first a congestion of the membrane, followed by a soft exudation which in the earlier stages is easily scraped off. These points of exudation become firmer, and are the site for the deposit of fibrinous coagula, which, by roughening the surfaces of the valves, prevent them from closing entirely, or, by agglutinating their leaflets, produce a constriction of the orifices. These vegetations may become detached by the friction of the blood current, and form emboli in the smaller vessels.

Ulceration of the endocardium sometimes occurs but is not a frequent sequence.

Occasionally the inflammatory process extends down into the

muscular substance of the heart (*acute myocarditis*) but this also is of very rare occurrence.

What are the symptoms?

The symptoms of this affection are usually very obscure at first, being masked by the disease with which it is intercurrent.

The patient suffers with an undefined sense of uneasiness in the præcordial region; dyspnœa; gastric irritability, and fever, the temperature reaching 104° to 105°. The action of the heart is tumultuous and excited.

As the inflammation proceeds the cardiac action becomes less frequent and there is a tendency to venous stasis.

Short cough is a very frequent symptom; the pulse is quick and tense, becoming irregular, and there is an anxious expression of face.

Should myocarditis occur, the cardiac action becomes feeble and irregular, and intense pain is developed, associated with greatly-increased dyspnœa.

If, during an attack of endocarditis the lips become blue, there is coldness of the extremities, cold sweats, and great oppression, coming on suddenly, a *heart clot* is probably forming.

If the patient should become suddenly paralyzed, it is due to *embolism*, part of the clot having become separated and being washed into some of the cerebral vessels.

Should ulcerative endocarditis occur, there are chills, followed by very high fever, the temperature even reaching 107°, delirium, and symptoms of a low typhoid state. Enlargement of the spleen associated with tenderness is common in this form of the affection. Jaundice sometimes occurs.

What are the physical signs?

On inspection and palpation there is an excessive irritability of the heart, associated with some irregularity.

On auscultation the first sound of the heart is muffled, or there may be two first and two second sounds.

A murmur is soon developed, most distinct upon the left side, and systolic in time, the intensity of which varies from day to day. It is usually soft at first.

What is the duration of this affection?

It is never less than six nor more than twenty days, the average duration being eleven days.

How is it diagnosed?

It is diagnosed by the dyspnœa; nausea and vomiting; cough and fever; disturbed cardiac action; muffling of the cardiac sounds, and the development of a murmur soft in character and varying in intensity at each visit.

Differential Diagnosis.

(1) *Endocarditis from pericarditis.* In the first stage of the latter disease a friction sound follows but does not replace the cardiac sound. It is more superficial, is influenced by the pressure of the stethoscope and is not transmitted into the vessels. In the second stage the increased area of percussion dulness, the absence of the cardial sounds at the apex, and the friction sound above the line of fluid, serve to distinguish them.

(2) *From a blood murmur*, perhaps developed after a low fever. This is distinguished from the murmurs of endocarditis by never taking the place of the second sound, and by being preceded by gradual loss of power in the heart and especially in the first sound of the heart. The impulse also is diminished. These murmurs are not as distinct in the recumbent posture, while in endocarditis posture does not influence the murmur.

(3) *From old murmurs* which may be heard for the first time during an acute affection. These are not always easy to differentiate if nothing is known of the antecedent history of the patient. A recent murmur, however, is soft and low pitched, while old murmurs are harsh. An old valve trouble will not exist for any length of time without producing some hypertrophy or dilatation; therefore, in these cases the murmur will be associated with an increase in the area of percussion dulness.

What is the prognosis?

The prognosis of acute endocarditis is favorable as regards immediate danger; but there is usually left behind habitual palpitation and, very probably, various valve troubles depending upon the products of inflammation.

Acute myocarditis is very fatal, the patient generally dying very rapidly from exhaustion or cardiac paralysis.

Ulcerative endocarditis is an exceedingly grave affection.

How should it be treated?

The patient must be kept absolutely quiet in bed. A few wet-cups or leeches applied to the præcordium do much good, early in the disease. They may be followed by applications of ice, cautiously used, or by warm poultices, the latter being preferable. The action of the heart may be regulated by aconite or veratrum viride, if it is strong and tumultuous; but these remedies should be employed with great caution, and must never be given if ulcerative endocarditis is present, or if there is danger of embolism. In these cases digitalis is preferred.

The alkalies may be used, as the ammonium salts, to render the blood more fluid, thus preventing heart-clot, and perhaps fibrinous deposits on the valve.

Mercury may be administered in small doses to aid the absorption of inflammatory products, but should not be given in myocarditis, ulcerative endocarditis, if Bright's disease or rheumatism is present, or where the patient is weak. In these cases potassium iodide is much more serviceable. *Myocarditis and ulcerative endocarditis* should be treated in much the same manner as is pyæmia, giving iron, quinine, digitalis and stimulants.

Chronic Endocarditis and Valvular Disease.

What is chronic endocarditis?

Chronic endocarditis is a chronic inflammation of the endocardium usually resulting in a valvular affection of the heart and generally preceded by an acute attack, or coming on slowly as a subacute or chronic inflammation from its beginning, not preceded by any active inflammation. The lesions produced are of two kinds.

(1) *Insufficiency*, where the valve is insufficient for its office and, as a result, regurgitation of the blood is permitted.

(2) *Stenosis or constriction*, consisting in a narrowing of the orifice, preventing the blood from flowing freely through it. These two conditions may exist together or separately.

What is the result of these conditions?

No valve trouble can exist for any length of time without giving rise to some alteration in the heart's structure, as is shown by the hypertrophy or dilatation, either separate or combined.

These changes are, to a certain extent, physiological, the hypertrophy being compensatory to the increased resistance which the heart has to overcome in supplying the tissues with blood. Dilatation, on the other hand, occurs from gradual overstretching of the cavities of the heart, caused by the increased amount of blood which they are forced by the disease to contain, and is a sign that the power of the heart is inadequate to fulfill its function. The most frequent seats of disease are the mitral or aortic valves. Occasionally the tricuspid and very rarely the pulmonary valves are affected. Insufficiency allowing of regurgitation is the most common affection. The diseases of the left side of the heart, especially of the aortic valve, are most frequently complicated with hypertrophy, while those at the right side, especially the pulmonary artery, are associated with dilatation at a much earlier period.

What are the general symptoms of valvular disease?

The symptoms vary much. They are principally due to the accompanying dilatation or hypertrophy. When the right side of the heart is affected, venous congestions may follow. Dyspnœa is more generally seen in all valvular diseases than any other symptom.

Dropsy bears no relation to the valve trouble, but depends upon the accompanying dilatation.

What are the physical signs of valvular trouble?

The most important sign is a *persistent murmur*, which is usually rough and has a point of greatest intensity to which it should be traced. Murmurs are usually transmitted all over the heart, no matter where the seat of the disease may be, but are heard most distinctly near their point of development.

How is valvular disease diagnosed?

It is diagnosed by the murmur, the history and the accompanying hypertrophy or dilatation.

DIFFERENTIAL DIAGNOSIS.

From an anæmic murmur. Anæmic murmurs are always soft,

and accompanied by signs of anæmia. They occur with the impulse, are transmitted into the vessels of the neck, are often associated with a venous hum in the jugular veins and are not transmitted to the axillary line, or to the back, as is the case with mitral murmurs.

Describe the symptoms and signs of diseases of the mitral valve.

In all diseases of the mitral valve the point of maximum intensity of the murmur is under or near the apex of the heart. Shortness of breath is a common symptom, as is also dropsy due to dilated hypertrophy. Lung complications are commonly present, as congestion, hemorrhage, etc., and there is some irregularity of pulse.

Mitral Regurgitation is the most common form of disease attacking this valve. *The murmur occurs with the first sound of the heart*, the second sound being distinct.

Mitral Stenosis. It is sometimes very difficult to tell the first from the second sound, and *the murmur is presystolic or post-diastolic.*

Describe the symptoms and signs of diseases of the tricuspid valve.

Regurgitation through this valve is the only disease which is at all common on the right side of the heart. As this portion of the heart is connected more especially with the venous system, general fulness of the veins, considerable dropsy from venous stasis and dilatation of the heart quickly ensue. Occasionally dilatation is the primary disease, the auriculo-ventricular opening becoming so stretched that the valves cannot close it, and hence allow of regurgitation. There is frequently a venous pulsation in the neck, because the blood is forced back through the valvular orifice with each contraction of the ventricle. In aggravated cases this pulsation is very distinct. It is sometimes also plainly seen in those organs which are connected closely with the heart by the venous system, as the liver.

Albuminuria, jaundice and various kinds of indigestion are common symptoms from venous stasis in the kidneys, liver and stomach.

Physical Signs.—A short, hoarse *murmur*, *systolic in time* and usually not transmitted, is *heard to the right and a little above the ensiform cartilage.* The signs of dilatation are present, as increased

area of percussion dulness, and a wavy, comparatively feeble impulse.

STENOSIS. This is the rarest of all heart diseases, and soon leads to dilatation and thickening of the auricles. Venous stasis occurs and the symptoms are similar to those of tricuspid regurgitation.

Physical Signs.—Murmur is heard preceding or taking the place of the second sound of the heart, its point of maximum intensity corresponding to that of the regurgitant murmur.

What are the symptoms and signs of diseases of the aortic valve?

AORTIC REGURGITATION. The pulsation of the temporal, radial and other arteries is distinct. There is a secondary current caused by the blood flowing back from the aortic opening into the cavity of the ventricle when the contraction of the latter is over, producing a *receding pulse* in all the arteries and causing an almost pathognomonic sphygmographic tracing, the lever rising abruptly when the hypertrophied ventricle contracts, making a little quiver resembling a hook at the top of the tracing, and then descending abruptly. The pulse is forcible, but recedes from the finger. It is called *the water-hammer or the Corrigan pulse.* When dilatation supervenes, dropsy and other characteristic symptoms will be added.

Physical Signs.—The *murmur begins at mid-sternum and passes to the right second costal cartilage.* It is transmitted into the arterial system at large and *replaces the second sound of the heart.* There is often a double murmur due to the roughness or vegetations on the aortic valve; but the murmur which takes the place of the second sound is always loud and distinct.

AORTIC STENOSIS. This gives rise to much the same symptoms, except that the significant receding pulse is not present, and the *murmur is with the first sound* owing to the difficulty which the blood meets in getting out of the ventricle. Its point of maximum intensity being the same as in aortic regurgitation.

What are the symptoms and signs of diseases of the pulmonary valves?

IN PULMONARY REGURGITATION the symptoms are those of dilatation of the right side of the heart, congestion of the lungs, and venous

stasis. *The physical signs* are a loud blowing *murmur taking the place of the second sound of the heart, the point of maximum intensity being the junction of the third left costal cartilage with the sternum.* An increased area of percussion dulness shows the presence of dilatation.

In pulmonary stenosis the symptoms are at first hypertrophy, with, later, those of dilatation superadded. The seat of maximum intensity of *murmur* is the same; but it is *associated with the first, not the second sound of the heart.* It is invariably a congenital disease.

What is the prognosis of these affections?

They are not curable, though life may be considerably prolonged by care and proper treatment.

Aortic stenosis is for the most part a favorable disease, the patient living for years, with proper care.

In aortic regurgitation, the heart becomes enormously hypertrophied, and apoplexy may ensue.

In mitral regurgitation, the patients may enjoy a moderate amount of good health, with occasional attacks of pulmonary congestion, dyspnœa, etc., as the compensatory hypertrophy yields to dilatation. They will not, as a rule, die suddenly.

Mitral stenosis; the prognosis here depends upon the hypertrophy or dilatation.

Tricuspid disease is the worst of all, dilatation of the heart coming on quickly, and the patient usually dying within a year.

Pulmonary regurgitation; death results from dilatation.

Pulmonary obstruction is always congenital, death usually resulting within a year.

How are valvular diseases of the heart treated?

The treatment depends upon the condition of the heart muscle. If hypertrophy is present, and not in excess of the needs of the system, *it must not on any account be interfered with.* When it begins to fail, digitalis or other cardiac tonics, and stimulants, must be administered, to retard the progress of dilatation as much as possible.

In aortic regurgitation there is usually much hypertrophy and fulness of the vessels of the head. An unstimulating diet, consisting of vegetables, fish and milk, may be given. He should use no

alcohol or tobacco and do nothing to increase the hypertrophy of the cardiac muscle, which should not be interfered with, but simply kept within bounds unless it is excessive, when aconite should be administered and the case treated as one of hypertrophy.

In mitral disease, and especially in regurgitation, hypertrophy exists, but not to so great an extent, not being so necessary for the comfort of the patient. If the hypertrophy is too great aconite or veratrum viride may be given.

When dilatation occurs, as it will later in the disease, digitalis is preëminently of value.

Nitroglycerin and belladonna are also of service.

Treatment of Special Symptoms.

For dyspnœa. Dry cups are a most valuable means of removing this symptom. Hoffman's Anodyne may be given internally in f℥j doses every fifteen to thirty minutes until some relief is experienced. The aromatic spirits of ammonia are also of service, and, as a last resort, hypodermic injections of morphine may have a good effect.

For general dropsy the best treatment is diuretics, as scoparius, juniper, and usually digitalis; dry cups should be applied over the kidneys and the bowels should be regulated by an occasional dose of blue pill or podophyllin followed by a saline.

For sleeplessness opium is the best remedy; chloral should never be used when there is dilatation.

Angina Pectoris.

What is angina pectoris?

It is probably often a neuralgia of the cardiac plexus of nerves, as a result of which the cavities of the heart distend, a tendency to failure in the contraction of the ventricle is present and death may result in a paroxysm. It is frequently associated with organic disease of the coronary arteries. There is often fatty heart also. It is rarely seen with valvular disease, though occasionally there may be disease of the aortic valves.

What are the symptoms?

There is an agonizing pain in the cardiac region; profuse sweat breaks out on the brow; he presses his hand to his heart, and starts

up with severe dyspnœa, great depression and intense anxiety. There is pain also in other regions of the body, particularly running down the left arm, usually as far as the elbow. The attacks are of short duration.

How is this affection diagnosed?

This disease is recognized by the paroxysmal character of the attacks, the great pain, the dyspnœa, the cold sweat, and the fact that the cardiac action is not generally accelerated nor associated with palpitation.

What is the prognosis?

It is a very dangerous disease.

The patient may die in the first attack, or may have a series of attacks, or may suffer from a number of light attacks, being in almost constant pain.

How should it be treated?

His occupation should be such as to require no mental or physical strain, even where no organic disease is perceptible. If he is of a gouty diathesis the treatment for lithæmia should be employed. Bromides or arsenic in small doses long continued are often of service. Nitroglycerin (gtt. j of a 1 per cent. solution increased to gtt. iij) given for a long time until it produces headache and other symptoms which require it to be discontinued, is one of the best remedies in this disease.

During the attack the inhalation of five drops of amyl nitrite affords most rapid relief.

Morphine may be used, or ammonia or brandy given.

Exophthalmos.

What is exophthalmos?

It is also called *exophthalmic goitre*, *Grave's disease*, or *Basedow's disease*, and is characterized by prominent eyeballs, enlargement of the thyroid gland and extreme irritability of the heart, sometimes eventuating in hypertrophy.

What is its cause?

There is no known cause.

It is more common in women, and appears to be due to some disease of the nervous system, and probably of the sympathetic ganglia.

What are its symptoms?

The eyeballs are prominent, giving a fierce, unpleasant expression to the face. It is probably due to increased vascularity at the base of the eyeball, the enlarged vessels pushing it forward. The pupils may be dilated or contracted, but, as a rule, are not normal. Little specks are seen before the eye, and the ophthalmoscope shows an increased blood supply to the eye fundus.

The thyroid gland is enlarged like goitre; but never grows to a very great size. The right side is more especially enlarged. On listening with the stethoscope over the gland a murmur is heard, which is never present in ordinary goitre. These goitres tend rather to diminish than to increase in size.

Irritability of the heart is frequent, and probably precedes the other symptoms. As a rule, there is nothing abnormal to be detected in connection with the heart. If the disease lasts, hypertrophy ensues.

Anæmia may be present, but is not a constant symptom.

Sometimes one of the three symptoms is absent. The irritability of the heart is never absent.

What is the prognosis?

Many recover entirely. Some die from the hypertrophy of the heart which ensues.

How should it be treated?

The patient should be placed in a recumbent position and kept there as long as possible. If this is not practicable, or if hypertrophy is present, a long-continued course of aconite, given in very minute doses, should be employed. In weakly people, where there is no hypertrophy, and the disease is seen in its incipiency, digitalis is of service. Ice-bags applied intermittently over the region of the heart are valuable. An occasional saline laxative does good. It is common in this malady to prescribe iron, but its use had better be limited to those cases associated with anæmia. Ergot is of some use, as is also arsenic sometimes. Iodine is practically useless. Galvanism has been applied to the sympathetic nerve, but without much result.

Cardiac Palpitation.

What is this affection?

It is the most common expression of functional disorder of the heart, characterized by increased frequency of its action and irregularity of its rhythm, sometimes amounting to intermittency.

SYNONYMS.

Irritable heart.

CAUSE.

Dyspepsia is a very common cause, as is also the excessive use of tobacco, or a too free indulgence in tea, coffee, or alcohol. Sexual excesses are also common causes. Over-exercise, particularly in walking, running, or dancing, is a common cause. Disorders of the generative organs will also produce it, as will long-continued depressing emotions, such as grief, anxiety or fear.

If the cause continues, the affection finally results in hypertrophy of the heart.

Palpitation may be a symptom of organic disease, especially of fatty heart, but this is not common.

What are the symptoms?

The palpitation usually comes on suddenly, and occurs in paroxysms. There is a feeling of oppression, followed by dyspnœa and a rapid, tumultuous action of the heart, increased by the recumbent position and accompanied by various sympathetic disturbances, often of an hysterical nature, such as a sense of choking, vertigo and a feeling of impending death. After the paroxysm has ended the patient often passes a large quantity of pale urine.

What are the physical signs?

In the vast majority of cases there is no murmur whatever. The cardiac outlines are normal and the impulse is not displaced, but is jerky, as if the heart were occasionally bounding or endeavoring to turn over.

What is the prognosis?

It is favorable if properly treated and taken in time before hypertrophy commences.

How should it be treated?

Endeavor to ascertain the exciting cause and remove it; then give some remedy to quiet the over-action of the heart, for which purpose digitalis in some bitter tonic is the best. A belladonna plaster may also be applied over the præcordium. Bromides are often of value to quiet the nervous perturbation. A change of scene, especially travelling, is very useful. In very bad cases rest in bed, with the intermittent application of ice over the heart, will be found serviceable.

When the disease is seen in persons of sedentary habits, moderate exercise is of value, while those who lead an active life are benefited by rest.

DISEASES OF THE PERICARDIUM.

Pericarditis.

What is this affection?

It is an acute or chronic inflammation of the pericardium, characterized by fever, disturbed circulation, dyspnœa and pain, and similar in its pathology to inflammations of other serous membranes.

What are the causes?

The causes are those which produce endocarditis, such as acute rheumatism; not so frequently nephritis, traumatism, inflammation extending from adjacent structures, as from a pleuro-pneumonia, general blood affections, as pyæmia, and various mechanical causes, such as swallowing false teeth (which have been known to perforate the œsophagus and pericardium).

What is the morbid anatomy?

Acute pericarditis consists of three stages:—

First, or dry stage. The pericardium is dry and an exudation of lymph is seen upon its surface, either in shaggy, villous masses, or smeared irregularly on its interior. Adhesions form between the surfaces of the pericardium, which may become adherent or agglu-

tinated permanently in portions, or throughout its entirety. More commonly, however,

THE SECOND, THE WET STAGE, OR STAGE OF EFFUSION results, and the surfaces become widely separated by a serous exudation in which the heart floats.

THE THIRD STAGE, OR STAGE OF ABSORPTION. As this fluid is absorbed the lymph is usually absorbed with it, and leaves a somewhat roughened pericardium, or adhesions may form, interfering somewhat with the action of the heart and usually resulting in dilatation, with hypertrophy.

Or the fluid may remain, the disease resulting in chronic pericarditis.

What are the symptoms?

In the first stage there are usually chills, some fever, a feeling of oppression and weight about the heart, and acute shooting pains, increased by pressure and movement; there is a slight hacking cough, difficulty in breathing, and in severe cases, a great difficulty in swallowing, with great anxiety and palpitation, all of which are more marked than in endocarditis.

During the second stage nausea and vomiting are seen, with irregular, feeble pulse; pain in the epigastrium increased by taking food; increased dyspnœa and dysphasia, hiccough and embarrassed cardiac action.

In some cases choreic movements are present, irregular motions of the arm or leg. Symptoms resembling those of irritation at the base of the brain, sometimes even fierce, active delirium, are seen. Melancholia occasionally occurs, or symptoms of other forms of insanity.

Sometimes there are extraordinary aberrations of temperature, the thermometer even registering 110°.

As absorption takes place the symptoms all gradually disappear, leaving an irritable heart, which may remain for some time.

The first stage usually rapidly passes into the second stage. The stage of absorption occupies several weeks, as a rule.

If the disease becomes chronic (occasionally it is chronic from the beginning) and the fluid remains, the symptoms of disturbed circula-

tion will continue, with dyspnœa, irregular and feeble pulse, and pain and distress in the cardiac region.

Similar symptoms are experienced in cases of adherent pericardium, varying in degree with the amount of agglutination.

What are the physical signs?

FIRST STAGE. *A friction sound* is heard, limited to the cardiac region and uninfluenced by breathing, uninfluenced by pressure either of the hand or stethoscope, usually most distinct at the base, and being much modified, even almost disappearing, when the effusion occurs.

On inspection and palpation the *impulse* is tumultuous and excited; occasionally a pericardial *friction-fremitus* is felt.

SECOND STAGE. On *percussion* there is *dulness of triangular form*, the base of the triangle being below on a line with the sixth rib, and extending from the right of the sternum slightly to the left of the left nipple line. The dulness varies in extent, depending upon the amount of effusion present.

On inspection and palpation a feeble, fluttering and diffused *impulse* is found.

On auscultation the *sounds of the heart are absent* at the lower part over the area of dulness, but are heard above the line of effusion, where they are often loud and distinct. A *friction sound* is sometimes heard above the line of effusion.

IN THE THIRD STAGE, *when absorption occurs*, the *percussion* dulness gradually disappears, the *sounds* become more and more distinct, and later the *friction sound* is heard.

After the fluid is absorbed, if the membrane is almost normal, no physical signs of disease will be present; *if adhesions have formed* the physical signs of dilatation, of hypertrophy, or of both supervene, and later on, as the adhesions become firmer, a sinking in of the chest wall will occur with the apex beat.

If the effusion remains, the physical signs of the second stage still continue.

How is pericarditis diagnosed?

The diagnosis is based on the physical signs.

DIFFERENTIAL DIAGNOSIS.

(1) *From endocarditis.* In the latter disease there is a murmur; excited cardiac action; slight, if any, increase of percussion dulness; a strong impulse, and the sounds are normal or more distinct except at the site where the murmur is heard. In the former, during the first stage, there is a friction sound and excited action of the heart; in the stage of effusion the area of percussion dulness is marked and extended, the impulse is wavy and feeble, and the cardiac sounds feeble and muffled, or absent below. There is no murmur.

(2) *From pleurisy.* In this disease the friction sound in the first stage will cease if the patient stops breathing. In the second stage the dulness is not limited to the region of the heart, is not triangular in form and does not interfere with the cardiac action and the impulse.

(3) *From dilated or fatty heart.* In these diseases the first sound of the heart is feeble everywhere, there is no triangular dulness preceded by a friction sound. Later in dilated heart the presence of dropsies will aid in the diagnosis.

What is the prognosis?

The majority of cases of acute pericarditis recover.

If it is due to blows on the chest wall, the prognosis is favorable.

It is unfavorable if it follows rheumatism or Bright's disease.

If the effusion is large there is danger of cardiac failure.

Unfavorable signs are delirium, vanishing pulse, jactitation and high temperature.

If adhesions remain, the danger of dilated hypertrophy should be borne in mind.

What is the treatment of pericarditis?

In the dry stage, if the patient is not too weak, and the case is seen in its incipiency, a few leeches should be applied over the præcordium. The application of ice-bags is also valuable in this stage.

An effort should be made to diminish the volume of blood by the use of saline laxatives, thus drawing serum from the intestine, and tending to prevent its effusion into the pericardium.

These laxatives should be given early and continued throughout

the disease, taking care, however, not to disturb the patient too much, and insisting upon his remaining in bed and using a bed-pan.

Alkalies, as potassium acetate, are also of great service. Opium or morphine in small doses may be given to steady the heart and tranquillize the nervous system. If, however, the disease follows nephritis opium should be employed cautiously.

When an effusion is present the alkalies should be continued and diuretics employed. The patient should not be purged too actively. He *must* use a bed-pan now, as there is great risk of cardiac failure should he get out of bed.

The action of the heart should be steadied by alcohol in large doses, by caffeine and perhaps by cocaine. Digitalis is not good in this affection after effusion has taken place.

Small repeated blisters should be applied over the præcordium.

Opium must be continued in small doses, the action of the kidneys maintained by potassium acetate or bitartrate, and iodide of potassium administered to aid absorption.

If the fluid lingers, sustain the system with tonics, give cardiac stimulants to maintain the action of the heart, and endeavor to produce absorption by active treatment.

If the fluid remains, and the case becomes chronic, or if the effusion is so large that there is danger of a cardiac paralysis, and there is not time for absorbents to act, *paracentesis pericardei* must be performed.

In hydrops pericardei associated with hydrothorax, the pleuritic effusion should first be withdrawn by tapping, as the pericardial effusion will frequently become absorbed when the former is removed.

DISEASES OF THE VESSELS.

Thoracic Aneurism.

What is thoracic aneurism?

It is a pulsatile tumor of the thorax, filled with blood, connecting with a vessel whose coats form its walls.

What are the causes?

It is usually produced by degeneration of the walls of the vessel, similar to what is seen in the case of external aneurism; occasionally, however, rupture of one of the coats may lead to dilatation of the others, and thus the aneurismal sac will form.

What are its symptoms?

Pain is almost universally present. It varies in character, sometimes being dull and transient, on the other hand it may be boring and persistent and, finally, in some cases, sharp and neuralgic.

Pressure on various nerves, on the trachea or œsophagus may produce characteristic symptoms. Thus there may be a peculiar noise on breathing or a loss of voice. In some cases the voice is changed from a deep bass to a shrill squeak. There is often difficulty in swallowing, the pupils may be unequal and the pulse often differs on the two sides of the body. Dyspnœa is frequently present. Aneurism does not necessarily terminate in death. Recovery sometimes takes place by clots gradually forming, and filling up the sac, which shrivels and degenerates into a fibrous mass connected with the vessel. The greater number of patients, however, die from exhaustion or from rupture of the aneurismal sac, either externally or into the pericardium, œsophagus or bronchial tubes. There is nothing to indicate when rupture is about to take place. Thoracic aneurism often produces absorption or caries of the vertebræ, ribs or sternum, by the pressure which it exerts upon them.

What are the physical signs?

Thoracic aneurism may occur in any part of the aorta, but in the

great majority of cases it is situated on some portion of the arch; in rare instances it is found in the innominate artery or other vessels.

Physical signs are present in the majority of instances,—almost universally when the aneurism affects the cardiac two-thirds of the arch,—but are frequently absent when the third part of the arch is the seat of the disease.

On inspection, a local bulging will be seen, one rib appearing more prominent than the rest. A pulsation is present which is distinct from the heart-beat. Not only is there a pulsation, but a thrill may be felt, which is generally, however, indistinct in internal aneurisms.

On percussion over the swelling, a dull sound is elicited.

On auscultation, there may or may not be a murmur. The first sound of the heart is dull and heavy, the second is well defined.

How is thoracic aneurism recognized?

The diagnosis is made from the pressure symptoms, from the impulse, which is distinct from that of the heart, there being two separate centres of pulsation, and from its occurring over the situation of the vessel.

Differential Diagnosis.

(1) *From diseases of aortic valves and dilatation of the aorta above them*, causing pulsation to the right of the sternum, distress in the chest, and sometimes dulness to the right of the sternum, but also producing a distinct, blowing, double murmur, transmitted to the heart; receding water-hammer pulse, and cardiac hypertrophy, but no pressure symptoms.

(2) *From mediastinal tumors*, causing dulness on percussion and bulging of the chest wall. In these cases there is an absence of strong pulsation; no murmur or peculiar hoarse sound, but frothy expectoration; hectic fever; cachexia and death from exhaustion.

(3) *From malformation of the sternum*, causing a prominence of the chest-wall and, frequently, displacement of the heart, consequently an altered position of the impulse. Here, however, there are no pressure signs, the pulsation is not great over the suspected seat of aneurism, and only occurs in one centre.

What is the prognosis?

Generally the disease is very slow, years elapsing before death

takes place ; occasionally, however, it is very rapid, and one case has been reported in which death occurred in fifteen days (Bartholow). In exceptional cases recovery may take place as has been already stated.

What is the treatment for thoracic aneurism ?

An endeavor must be made to favor coagulation of blood in the sac of the aneurism. The patient must therefore not exert himself in any way, and must avoid all excitement.

Local Treatment.

The constant application of ice over the seat of aneurism lessens the pain and the feeling of distention at all events, if it does not often result in cure.

The introduction of foreign bodies, as horsehair or needles, or electrolysis even, has been attempted, but these agents are all useless or dangerous.

General Treatment.

The best treatment consists in lessening the circulation, decreasing the volume of blood, and altering, if possible, its character.

In Valsalva's method, the patient is confined to bed on his back for a prolonged period, and only supplied with a limited amount of food, giving about four ounces of solid and two ounces of liquid food a day to begin with.

Conjoined with this treatment, aconite may be given to depress the action of the heart. This gives the best results.

Iodide of potassium has also been given in gr. x–xx doses, three times a day, occasionally intermitting the medicine for a few days, but prolonging the treatment almost indefinitely. It undoubtedly does good.

Ergot, or ergotine given hypodermically, once or twice a day, is also sometimes useful, but the results are not so favorable as in the two preceding methods of treatment.

Barium chloride has also been used successfully in doses of gr. $\frac{1}{5}$ gradually increased.

Treatment of Special Symptoms.

For the pain, ice applied over the seat of pain, or morphine administered hypodermically, are the best remedies.

To prevent or delay rupture; entire rest of mind and body, avoiding all excitement of every kind, regulating the bowels and preventing straining, are the best means to combat this symptom.

DISEASES OF THE NERVOUS SYSTEM.

DISEASES OF THE BRAIN.

Cerebral Hyperæmia.

Describe cerebral hyperæmia.

Congestion of the brain is really a rare disease. There are two varieties:—

Active hyperæmia is that form where the blood is sent to the brain in greater amounts or more forcibly than is compatible with health. It follows over-eating; is seen in plethoric subjects, particularly where the heart is over-acting; in cardiac hypertrophy; where too much mental work is done, conjoined with too little exercise; or is sometimes produced by a sudden check to the cutaneous action, such as may follow cold baths when no reaction takes place.

Passive congestion is that variety in which too much blood is in the brain from interference with the passage of the blood through or from the brain, and is seen especially where some pressure occurs on the veins of the neck.

The Symptoms of either variety are headache, which is generally dull; vertigo; dreams, or wakefulness; a jerking motion of the tendons during sleep (which is seen also in certain spinal diseases); confusion of ideas; irritability of temper; muscæ volitantes, a symptom of but little importance; and in the active form a flushed face and injected conjunctiva, usually accompanied with contracted pupil. The affection usually comes on slowly, but should it begin abruptly and the congestion be intense, rapid unconsciousness may supervene with symptoms resembling those of apoplexy, which, however, soon pass away.

The Prognosis is favorable, unless there is some organic lesion.

THE DIAGNOSIS is easy, and is arrived at from the history, the headache, vertigo and cause.

THE TREATMENT consists in attending to the state of the bowels, especially using saline purgatives, and occasionally administering a mercurial.

In the active form the diet should be restricted. The patient should avoid meat; he should be subject to no mental strain nor brain work, but must lead a more active out-of-door life.

Counter-irritation may be applied by dry cups or mustard to the nape of the neck, and, if the subject is plethoric, local depletion by means of wet cups is frequently of service.

Bromides should be administered, and if there is an over-acting or hypertrophied heart, small doses of aconite may be advantageously given. In the sudden, acute cases with symptoms resembling those of apoplexy, venesection may be resorted to with advantage.

In passive congestion remove the cause, if possible, and strengthen the circulation by means of digitalis, strophanthus, etc.

Cerebral Anæmia.

Describe cerebral anæmia.

Cerebral anæmia results most frequently as a part of a general condition of anæmia. It may also be caused by some obstruction to the vessels, producing a localized anæmia, as in the case of thrombosis or embolism.

SYMPTOMS.

In the former variety there will be headache and vertigo, both relieved by the recumbent position, thus differing from similar symptoms seen in cases of congestion of the brain. Pallor and other symptoms of general anæmia are usually marked, and attacks of fainting are frequent.

In partial anæmia the symptoms are those of thrombosis or embolism.

PROGNOSIS.

The prognosis depends upon the cause. It is usually favorable.

TREATMENT.

The treatment consists in giving a good nourishing diet, plenty of fresh air, and moderate exercise if the patient is strong enough.

Strychnine, wine and iron should be prescribed, with attention to the state of the bowels.

Meningitis.

What is acute meningitis?

It is an inflammation of the arachnoid and pia mater, characterized by headache, fever, delirium, and often followed by convulsions, hemiplegia, collapse and death.

What is its causation and pathology?

IT IS CAUSED by traumatism; over-work; exposure to the sun; extension of inflammation from adjacent parts, as from the internal ear; syphilis; erysipelas, and the eruptive fevers. It is a disease particularly of adults.

THE PATHOLOGICAL CHANGES consist in congestion and inflammation of the arachnoid and the pia mater, with effusion between them and into the ventricles, the convexity of the brain is most frequently affected, though in some cases, especially following otitis media, the base may bear the brunt of the disease.

What are its symptoms?

(1) *Prodromic period.* There is headache; vertigo; vomiting, with a clean tongue, and some fever lasting from a few hours to a few days. This is followed by—

(2) *The stage of invasion*, which comes on suddenly with a chill, followed by a high fever and a full, tense pulse, from 100 to 110; flushed face; injected eyes; headache and vertigo.

(3) *The stage of excitation.* All the symptoms become very much aggravated, and there is jerking of the muscles, photophobia, and violent delirium.

(4) After a week or ten days this is followed by *the stage of depression*, when the patient gradually becomes more quiet. The delirium subsides and is replaced by stupor; the pulse is irregular; the pupils sluggish; partial palsies occur, and death takes place, sometimes preceded by general convulsions.

If the patient recovers, a gradual reaction takes place, with head aches or occasional convulsions lasting perhaps for years.

How is this disease recognized?

It is recognized by the history, the cause, the fever, the intense headache, fierce delirium, contracted pupils, the causeless vomiting and the stages.

Differential Diagnosis.

From cerebro-spinal fever, by the absence of the spinal symptoms, eruption and epidemic influences and by the cause.

What is the prognosis?

The prognosis is never very favorable. When it occurs from a blow, injury or excessive work, it is better than when occurring after sunstroke. It is most grave when due to extension of inflammation from the internal ear, as in these cases an abscess is apt to form, and pyæmia or septicæmia result.

Syphilitic meningitis gives the most favorable prognosis.

How should it be treated?

The patient must be placed thoroughly at rest in a dark, cool, quiet room. The diet should consist of milk or weak broths, and an active purgative should be administered at once.

Local bloodletting by means of leeches applied to the back of the ear or temples should be employed, provided the inflammation has not extended from adjacent organs and is not purulent in character.

Cold applications to the head by means of an ice-bag, and the administration of bromide of potassium and chloral internally; or, if these fail to relieve the violent headache, morphine hypodermically will give the best results.

The circulation should be controlled by small doses of aconite, and ergot is sometimes employed with the idea of contracting the vessels.

After the effusion has taken place small doses of mercury or iodide of potassium may be administered to produce absorption. The latter is especially valuable in syphilitic cases.

If the patient is losing ground, if the brain appears to be overwhelmed by a large effusion oppressing it, or if the stage of depres-

sion is very profound, his strength must be kept up by alimentation, and chloral, opium, and even small amounts of stimulants, and digitalis, administered.

Tubercular Meningitis.

What is this disease?

It is an inflammation of the meninges of the brain, caused by a deposit of miliary tubercle, particularly at the base and along fissures.

What is its cause and pathology?

CAUSATION.

It is most frequent in children under six years of age, especially in those who inherit a tendency to consumption.

PATHOLOGY.

It consists in the deposit of disseminated tubercle at the base of the brain and along the fissures, although not limited to these regions. This produces inflammation, and effusions occur between the pia mater and the arachnoid and into the ventricles, which sometimes are greatly distended with fluid (acute hydrocephalus).

The lungs are generally full of disseminated tubercle also, and the bowel and spleen are sometimes involved.

What are the symptoms?

The symptoms usually come on gradually.

(1) There is a *marked prodromic period*, during which the child loses flesh, is peevish, and ordinarily has an irritative cough and irregular slight attacks of fever, with headache coming and going. This attack lasts for a few weeks or for months and is followed by

(2) *A second stage, that of excitement.* There is violent, distressing headache; the temperature is very irregular, usually high in the evening; the pulse is moderately frequent and irregular; vomiting occurs, with a clean tongue; photophobia; contracted pupils; spasmodic movements of the muscles; retraction of the abdomen, and constipation. Upon drawing the finger-nail lightly over the forehead a red line is produced, which is more marked in this than in any other cerebral disease.

(3) *The third stage, or that of depression*, follows the stage of excitement. Partial or general convulsions occur; partial palsies ensue; optic neuritis takes place from deposit of tubercle in the choroid coat of the eye; the pulse is very irregular, often extremely slow; it varies very much in the course of twenty-four hours, but is very intermittent and irregular; the vomiting continues, and the patella-tendon-reflex is well preserved, often exaggerated.

The child becomes worse and worse; he is heavy and dull, and has violent and frequent convulsions, with rigid fixation of certain muscles. Bedsores occur and add to the suffering of the little patient, who finally dies worn out by the pain and overwhelmed by the brain irritation and the effusions. Recovery is said to have occurred, but in these cases, as no post-mortem could be made, the diagnosis may be fairly called in question.

How is this disease recognized?

It is recognized by the long prodromic period, with the irregular attacks of fever, the headache and the loss of flesh; by the stage of excitement, with intense headache, irregular temperature and pulse, vomiting, with a clean tongue, retracted abdomen and constipation; by the stage of depression, with the convulsions and palsies, the irregular intermittent pulse, rigid fixation of groups of muscles and exhaustion.

What is the prognosis?

The prognosis is very unfavorable; few, if any, cases recover.

Duration from one to four months.

What is the treatment?

But little can be done to retard the progress of this disease. Cold may be applied to the head and laxatives administered; bromides, chloral and morphine have also been given, as in acute meningitis; iodide of potassium and mercury are administered in large doses, but without much effect, and the nutrition of the patient preserved by small amounts of food, cod-liver oil and stimulants.

Apoplexy.

What is apoplexy?

Apoplexy, or cerebral hemorrhage, is the sudden rupture of a cerebral vessel, with extravasation of blood into the brain substance, causing sudden unconsciousness, stertorous breathing, and a slow pulse, lowered temperature and complete relaxation of the muscular system.

What are its causes?

It appears to be strongly hereditary, and occurs especially after forty years of age, more frequently in the early morning hours, and in the spring of the year. The principal *predisposing causes* are arterial degeneration, syphilis, and Bright's disease.

The exciting causes are mental worry, gormandizing, and frequent stooping.

What is the pathology?

A fatty or granular degeneration has taken place in the walls of the smaller vessels, often giving rise to miliary aneurisms. These rupture, and blood is effused into the brain substance, membranes, or both. The corpus striatum or optic thalamus are usually the seat of the hemorrhage; sometimes both corpus striatum and optic thalamus are involved. The hemorrhage sometimes occurs in the convolutions of the parietal or temporo-sphenoidal lobes, or in the cerebellum; occasionally the pons or medulla oblongata are the seats of the effusion, and in rare cases the convexity of the brain is involved. Double apoplexies are rare. The clot may vary in size from that of a small pea to that of a hen's egg. It may become entirely absorbed in two or three months, may be encysted, or may lead to inflammation, softening and abscess in the surrounding brain substance.

What are the symptoms of apoplexy?

In some cases the attack is preceded by such *prodromic symptoms* as headache, vertigo, tingling sensations in the extremities, some confusion of ideas, and the dread of impending disease, lasting for a day or two, or more, before the actual hemorrhage.

The attack then comes on, suddenly, usually at night or in the

early morning. The patient falls to the ground bereft of consciousness and sensation ; his respiration full, labored and stertorous from paralysis of the buccinator muscle ; his pulse full and slow ; his face usually flushed, turgid with blood ; his temperature much below the normal, even as low as 93° ; his pupils sluggish, perhaps dilated or contracted ; the reflexes altered, often exaggerated on one side and deficient on the other, or absent on both, and the muscular system entirely relaxed.

The stage of reaction comes on usually within twenty-four hours, the first symptom of which is a rise in temperature. The unconsciousness lessens, and hemiplegia is now seen to be present. The temperature may now rise as high as 104° (from inflammatory changes developing around the clot), with tonic contractions of the paralyzed muscles, and severe neuralgic pains ; or, instead of reaction, the coma may deepen and death ensue.

How is this disease recognized ?

The diagnosis is made from the history of sudden unconsciousness ; the characteristic respiration, temperature and pulse ; the change of reflexes ; the sluggish pupils ; the reaction and the subsequent paralysis.

Differential Diagnosis.

(1) *From alcoholic coma.* The profound unconsciousness in the former, from which it is impossible to rouse the patient ; the altered reflexes ; the sluggish pupils, usually dilated and contracted, and the hemiplegia render the diagnosis clear.

(2) *From uræmic coma.* The history of kidney disease in the latter ; the preceding convulsions, normal reflexes, absence of palsies, the absence of secondary fever and the presence of tube-casts in the urine should prevent a mistake.

(3) *From opium poisoning* it is known by the sudden onset, the different temperature, the alteration in the reflexes and the one-sided palsies.

What is the prognosis ?

The prognosis as regards the attack itself is good, if the temperature rises within twenty-four hours, or if he shows early symptoms of returning consciousness.

It is unfavorable if the respiration or pulse are irregular ; if there

are convulsions; if there is vomiting, or great contraction of the pupils.

He is, however, apt to have a second attack, as the degenerated vessels which allowed the first to occur still remain.

If sensation returns to the paralyzed parts but there is no return to motion within a week or two, the case will be long and tedious, and probably recovery will never be complete.

How should the attack be treated?

If a patient has complained of prodromal symptoms, and it is feared that he will suffer from apoplexy, venesection, followed by a brisk purgative, should be resorted to if he is sufficiently robust.

If for any reason bleeding is feared, leeches may be employed over the mastoid process, and large doses of potassium bromide and chloral administered internally.

During the attack, if the patient is comparatively young and has a strong pulse conjoined with regular respiration, and if he is seen within twenty-four hours, bleeding or leeching should be resorted to. He should also be freely purged, and for this purpose croton oil is the best remedy, given in doses of gtt. j, mixed with a little olive oil or glycerin, and repeated every three or four hours until its effects are observed. Its action may also be aided by turpentine enemata.

When reaction comes on, the circulation should be controlled by aconite or veratrum viride, the patient kept perfectly at rest and as little food as possible given. If the temperature is high, a few leeches may be applied behind the ears, and the bromides administered. When the patient has completely passed out of the attack, the aconite should be withdrawn, the purgation reduced and iodide of potassium and mercury administered to promote absorption of the clot.

When the pulse is irregular and slow, and the skin cold (signs of a very large clot), the patient should not be bled nor purged, but small doses of ammonia should be administered at frequent intervals, and, if the cardiac action is weak, digitalis should be given hypodermically, followed by the administration of ergot.

To guard against future attacks he should lead a quiet life,

should avoid any mental or physical strain, should attend scrupulously to the condition of his bowels, should dress warmly, and, if possible, should avoid extremes of temperature.

Thrombosis and Embolism.

What are these affections?

Cerebral thrombosis is the occlusion of a vessel in the brain by the formation of a thrombus or clot.

Cerebral embolism is the occlusion of a vessel by the lodging of an embolus.

What are the causes and pathological anatomy?

CAUSATION.

Thrombosis is usually the result of chronic changes in the walls of the vessel, as atheroma with weakened force of the circulation. *An embolus* usually results from valvular disease of the heart, although it may occur from disease of the vessels also.

PATHOLOGICAL ANATOMY.

When the cerebral vessels are occluded there is anæmia in the parts of the brain corresponding to the distribution of those vessels. The collateral circulation is often rapidly reëstablished and the brain returns to its normal condition. Should this not take place, that portion of the brain which is deprived of its nourishment will undergo softening from want of nutrition. Embolism most frequently occurs in branches of the middle cerebral artery.

What are the symptoms of cerebral thrombosis?

The symptoms of cerebral thrombosis come on gradually, and are headache and vertigo, varying in severity, but persistent.

The patient becomes irritable, absent-minded, suffers from impairment of memory, and often hesitating speech. Muscular weakness, followed by hemiplegia, comes on, the patient usually passing from bad to worse and dying from exhaustion, although recovery may take place even after hemiplegia has occurred.

What are the symptoms of cerebral embolism?

The symptoms are sudden vertigo and headache; confusion of mind; mumbling speech; often vomiting, followed by hemiplegia.

Sometimes the symptoms are similar to those of apoplexy, the patient falling to the ground completely unconscious, usually with flushed face, followed by death or a gradual return to consciousness with hemiplegia. These cases frequently recover rapidly, a point distinguishing them from cerebral hemorhage.

What is the diagnosis and prognosis of these affections?

DIAGNOSIS.

The diagnosis of thrombosis is often very difficult, the patient's age or changes in the vessels and the weak circulation with the cerebral symptoms suggest the nature of the case.

Embolism in its gravest form resembles apoplexy. When the patient is young, and is suffering from valvular disease of the heart; when consciousness rapidly returns, and the hemiplegia disappears, he is probably suffering from the former complaint.

PROGNOSIS.

Cerebral thrombosis rarely terminates in recovery. *Cerebral embolism* may end in the restoration of perfect health. Frequently some hemiplegia remains permanently or the patient's mind may be enfeebled. Sometimes chronic softening results in the neighborhood of the embolus, and sometimes the patient dies without a return to consciousness when a large vessel is involved.

How should this disease be treated?

The heart's action should be sustained by hypodermic injections of digitalis and by ammonia. No depressing treatment of any kind should be employed. The bowels must be kept open without active purgation, and the patient kept perfectly at rest and sustained by a light nutritious diet. Iodide of potassium may be given in order to favor absorption. Alcoholic stimulants may be required.

Aphasia.

What is aphasia?

Aphasia (amnesic) is the loss of memory of words constituting articulate language. It is a symptom, not a disease. *Amnesic aphasia* is usually meant when the latter word only is used. *Ataxic*

aphasia is the inability to articulate, not from the loss of memory of words, but from trouble in producing combined action of the different parts of the vocal apparatus.

In the latter case the patient is usually able to write, but he cannot speak. *Agraphic aphasia* is the loss of memory of written words.

What are the causes and pathological anatomy of this affection?

Aphasia is caused by cerebral embolism, by cerebral congestion, by cerebral softening, or by tumors or other lesions affecting the third left frontal convolution and neighboring parts. It is usually associated, at least at first, with a right-sided hemiplegia.

What are its symptoms?

There is more or less complete loss of memory of words, the patient may simply clip words or may call things by the wrong name, knowing well that he is saying the wrong word, but unable to remember the right one; sometimes if he is told the right word he can repeat it, but almost instantly forgets it again. Some patients become very profane.

What is the diagnosis of this affection?

(1) It can be diagnosed *from aphonia due to laryngeal disease* by the use of the laryngoscope.

(2) *From diseases affecting the articulation, as of the tongue and lips*, by the fact that the tongue is well protruded. The patient can whistle, and often says a few words well.

What is the prognosis?

The prognosis varies according to the cause. If *from tumors or softening*, bad; if *from apoplexy*, the clot may undergo absorption and slow recovery follow; if *from congestion* of the brain, or *cerebral syphilis*, the prognosis is favorable.

How should it be treated?

The cause should be diligently sought for and proper treatment instituted. The patient frequently has to be trained in the use of words in the same manner that a baby would be taught to speak, and thus he can gradually acquire the power of expression.

Acute Cerebral Softening.

What is acute cerebral softening?

It is an acute granular degeneration of the nerve elements of the brain, characterized by fever, headache, mental failure and symptoms depending on the part of the brain structure which is involved.

What are its causes?

It may result from embolism or thrombosis of the cerebral vessels, or from acute inflammation of the brain substance. The former conditions have already been described, and it now remains to consider the last.

Acute inflammation of the brain is caused by traumatism; by the extension of the inflammatory process from contiguous parts, or by septic conditions of the blood.

What is the pathology of acute cerebral softening?

In the inflammatory form of softening the brain is much redder than normal (*red softening*), due to its increased vascularity and to minute extravasations of blood into its substance. It is softer than normal, owing partly to effusion and partly to proliferation and subsequent granular degeneration of the nerve cells.

What are the symptoms?

Headache is a frequent symptom, often accompanied by fever; sometimes vomiting, convulsions and delirium are seen, and sometimes it begins almost like an attack of cerebral hemorrhage, with delirium rapidly followed by unconsciousness. These cases are usually due to embolism or thrombosis. Paralysis, usually limited in extent, involving groups of muscles rather than an entire extremity, usually supervene, and the mental faculties are generally permanently impaired. Aphasia is often seen in this disease. Occasionally the symptoms resemble those of the chronic affection, differing only in running a more acute course.

What is the prognosis?

The prognosis of acute cerebral softening, no matter what the cause, is unfavorable.

How is it treated?

The treatment is purely symptomatic; the aphasia, hemiplegia, etc., being treated on general principles.

Chronic Cerebral Softening.

What are the causes of chronic cerebral softening?

It is often produced by occlusion of a cerebral vessel; by syphilis; prolonged mental work, especially in old people; extension of inflammation from adjacent structures, and by cerebral hemorrhage; therefore, by much the same causes as those which produce the acute disease.

What is the pathological anatomy of this affection?

The substance of the affected portion of the brain is softer than normal; can be easily washed away by a gentle stream of water running upon it. It is yellowish or whitish in color, and has thus given rise to the name "yellow" or "white softening." Under the microscope the nerve elements are seen to have undergone granular and fatty changes.

What are the symptoms?

There is gradual and progressive mental decay, impaired memory, headache, vertigo, debility, disordered cutaneous sensations, as numbness and tingling or formication, hyperæsthesia or anæsthesia, difficulty in articulation and slight palsies are often seen. Hyperæsthesia of the senses of sight, hearing and smell are often experienced, and the sense of taste perverted.

The disease progresses slowly and steadily, and the patient finally dies of some intercurrent affection or of old age, with its diseases.

The duration is about two years.

What is the treatment?

But little can be done for these patients, except to take care of them. They should be well nourished and given tonics, as iron and the bitters. Phosphorus in small doses sometimes seems to answer well for a time. Cardiac tonics should be administered, and for this purpose strychnine is probably the most efficient.

Moderate amounts of alcohol should be given at meals.

Abscess of the Brain.

What are the causes of cerebral abscess?

Cerebral abscess results from traumatism; from extension of inflammation from adjacent parts, as in otitis media, or in diseases of the cranial bones; also from apoplexy, thrombosis, embolism and in pyæmia.

The abscess most frequently affects the left side and consists of a circumscribed cavity, varying in size, and containing pus and cerebral detritus, the surrounding tissue being healthy but hyperæmic.

What are the symptoms?

The patient suffers from violent headache; vomiting, with a clean tongue; mental hebetude, frequently stupor or coma; irregular pulse; irregular rises in temperature; often recurring chills; palsies depending upon the seat of the abscess, and sometimes recurring convulsions.

What is the diagnosis, prognosis and treatment?

DIAGNOSIS.

It is often very difficult to make a diagnosis, which may be possible only by a close study of the case and the history. The recurring chills, the dull headache, the hemiplegia and the irregular rises in temperature may lead to the diagnosis of cerebral abscess.

From cerebral tumors, it may be differentiated by the more rapid course, the more acute beginning, and the temperature.

PROGNOSIS.

It usually terminates fatally.

TREATMENT.

The pain should be relieved by morphine. The strength should be sustained by food and small doses of stimulants, and the patient should be kept perfectly quiet. If it is the result of traumatism, if the abscess is superficial and can be located, the patient should be trephined and the pus evacuated, using antiseptic precautions.

Cerebral Tumors.

What varieties of tumors are found in the brain?

Of the various tumors, *gliomata* are the most frequent. *Sarcomata*, *fibromata*, *tubercular tumors*, *cancer*, *cysts* and *aneurisms* are also found. They may be situated in the bones of the skull, in the membranes or in any part of the brain.

What are the causes of cerebral tumors?

They sometimes arise from syphilis, sometimes from meningitis; from changes occurring in the vessels; after a protracted labor or during or after puerperal septicæmia, or without any assignable cause.

What are the symptoms?

Headache is the most constant symptom. It is sharp, agonizing, paroxysmal in the frontal or occipital region.

Epileptiform convulsions occur very frequently when the tumor is situated in the membranes or in the superficial portions of the brain structure.

Intellectuality is usually well preserved, although, later, the memory becomes impaired.

Vomiting, with a clean tongue, and without apparent cause, is a frequent symptom, especially when the base of the brain, or the cerebellum, is involved in the tumor. The special senses are often disturbed; local palsies occur, depending on the seat of the tumor, facial palsy being the most frequent. Examination of the eye with an ophthalmoscope shows a choked disc.

What is the prognosis, diagnosis and treatment?

PROGNOSIS.

The prognosis depends first upon the cause. If it is syphilitic, the prognosis is favorable under proper treatment.

It also depends upon the situation. If it is embedded in the brain and not pressing on an important ganglia, the duration may be long. If at the base of the brain or in the cerebellum, death takes place quickly, from exhaustion due to vomiting.

The variety is also an important point in the prognosis, but cannot be diagnosed from the symptoms.

DIAGNOSIS.

The disease is recognized by the headache, convulsions, affection of the special senses, the vomiting, palsies and the ophthalmoscope.

The situation of the tumor may often be diagnosed by the symptoms.

Tumors situated in the cortical structure produce convulsions and often local palsies. Should the convulsive movements affect one leg, the *upper parietal convolutions* about the fissure of Rolando are involved in the tumor; should the arm be affected, the parietal convolutions about the fissure of Rolando, but lower down than when the leg is convulsed or paralyzed, are involved; convulsive movements or paralysis of the muscles of the face denote a lesion situated near the lower end of the fissure of Rolando. When speech is involved, the island of Reil and parts near the division of the Sylvian fissure are pressed upon or diseased. One-sided vision indicates a lesion of the optic tracts; great nausea and extreme vomiting is seen when the base near the medulla is involved, spastic contractions when the base near the peduncles are irritated, and vomiting with disturbed locomotion and sometimes jaundice when the cerebellum is affected.

TREATMENT.

Large doses of potassium iodide should be given, and will frequently effect a cure, if the tumor is due to syphilis.

For the headache, bromides may be prescribed and blisters applied to the scalp or nape of the neck.

Cerebral Syphilis.

Describe cerebral syphilis.

Syphilis may produce gummata of the brain, may cause meningitis or disease of the cerebral vessels, giving rise to inflammation in the walls of the arteries, with thickening of their coats and narrowing of their calibre. It may also cause tumors of the skull or dura mater, which, by pressing on the brain substance, produce cerebral symptoms. It may be inherited; the symptoms may not appear for years after the primary affection.

SYMPTOMS.

The symptoms vary according to the form of disease. Thus the

patient may suffer from symptoms of tumor, from symptoms of meningitis, or disease of the vessels may cause such symptoms as may be produced by plugging the vessels and cutting off the blood supply to certain portions of the brain.

DIAGNOSIS.

It usually occurs in young persons. These affections are recognized by the great variability of the symptoms, which are apt to change rapidly without apparent cause, and which disappear rapidly under treatment.

PROGNOSIS.

The prognosis is favorable under early and proper treatment. If softening occurs from long-continued plugging of the vessels, the prognosis is, of course, bad.

TREATMENT.

Large doses of potassium iodide, alone or combined with mercury, must be given when cerebral syphilis is suspected. Mercurial inunctions are also of value.

Acute Hydrocephalus.

What are the causes of acute hydrocephalus?

It is most common before the fifth year of age. It may be produced by dentition, cholera infantum, tubercular meningitis (the most common cause), traumatism and acute fevers, or cardiac disease or diseases of the kidneys.

What is its pathological anatomy and symptoms?

PATHOLOGICAL ANATOMY.

A serous effusion occurs into the ventricles, and often in the subarachnoid spaces, which, by pressure, may produce softening of the brain. If the fontanelles have not become ossified the bones may become separated by the pressure of the fluid.

SYMPTOMS.

The patient may rapidly pass into a condition of coma, and death may result in a few hours; or there may be headache, nausea and vomiting and convulsions preceding the coma. In most cases, how-

ever, there is a feverishness, headache and vertigo, restlessness, twitching of the muscles, photophobia, and nocturnal delirium, followed by convulsions, stupor, coma and death.

What is the prognosis and treatment?

PROGNOSIS.

The prognosis is unfavorable, recovery rarely occurring.

TREATMENT.

The treatment consists in the administration of diuretics and diaphoretics, and keeping the bowels open, giving large doses of iodide of potassium, and nourishing, and often stimulating, the patient, should the latter not be contraindicated.

Chronic Hydrocephalus.

Describe this affection.

Chronic hydrocephalus, called also water on the brain and Congenital Hydrocephalus, occurs usually in very young children. The child may be born with it, or it may come on gradually after birth. It is due to a chronic inflammation starting near or around the choroid plexus and occasioning serous effusion into the ventricles, the amount of fluid varying from three to twenty-seven pints. This enormous amount of fluid causes atrophy of the brain substance by pressure, thins the bone of the skull and presses the sutures apart. It consists of water, salts and a very large amount of albumen, and under the microscope is seen to contain compound-granule cells. If the child survives, secondary ossification occurs in the sutures, and they thus become closed, the head remaining enlarged.

SYMPTOMS.

The child has a large head, the sutures are separated and there is an elastic fluid beneath which fluctuates on pressure.

Percussion elicits a cracked-metal sound. The head is not only large, but heavy, and often rolls from side to side, the face looks small in comparison and the intelligence is usually very limited. The ophthalmoscope sometimes shows a choked disc. Convulsions occur, or a tendency toward them is manifested. Idiocy is apt to

result, or the child passes into a condition of prostration, in which, probably from atrophy of the brain substance, voluntary motion appears impossible except under the strongest excitement. Death usually results. The disease may be arrested and sometimes the cerebral functions return, and even the highest grade of mental activity may result, if there is no atrophy of the brain substance.

DIAGNOSIS.

The characteristic appearance of the child is such that a mistake is hardly possible except in those cases where the amount of fluid is small, when it may be confounded with enlargement of the head due to rickets. In the latter disease, however, the fontanelles are not open, and there is no elasticity on pressure. The appearance of the head is different and there is an enlargement of the joints.

PROGNOSIS.

The prognosis is unfavorable.

TREATMENT.

The head may be aspirated and the fluid drawn off, followed by compression by means of elastic webbing or adhesive strips. The latter may be resorted to before aspiration.

Internally, diuretics, as the acetate of potassium, should be given. Occasional purgation should be resorted to and cod-liver oil and iodide of iron administered, at the same time paying great attention to the general nutrition of the child.

Insolation.

What is insolation?

There are two varieties of insolation.

(1) SUNSTROKE PROPER, or *thermic fever*, due to the direct exposure to the rays of the sun, especially when engaged in active exercise.

(2) HEAT STROKE, *or exhaustion*, occurring after a patient has been subjected to prolonged intense heat, either of the sun's rays or of artificial production.

What are the symptoms?

The symptoms of *sunstroke* come on suddenly during exposure to

the sun and are sudden giddiness, splitting headache, and rapid insensibility, sometimes preceded by nausea and vomiting. During unconsciousness the temperature rises even as high as 112°; if it is not over 104 or 105, recovery will usually take place. The skin is harsh and dry; the urine scanty; the bowels constipated; the reflexes are probably preserved, and the limbs relaxed; there is never any palsy.

In some cases the cerebral symptoms predominate, and the similarity to apoplexy is marked; the pupils being insensible to light and vessels of the head and neck full, the respiration labored and stertorous, the pulse labored and full. Albumen is sometimes found in the urine, and convulsions may take place, which are probably, however, not uræmic. Recovery is usually not complete, the attack being followed by sequelæ and the patient being subject to a recurrence on exposure to the sun.

The symptoms of *heat exhaustion* are giddiness, weakness, and feeble pulse coming on when the patient is not exposed to the direct rays of the sun, continuing for some hours and followed, perhaps, by unconsciousness, the attacks simulating faintness. The face is pale, the insensibility is not so marked as in the case of sunstroke, the respiration is uninfluenced and the temperature also is not so high.

How are these conditions recognized?

Thermic fever is recognized by the history of exposure, the sudden attack, with high temperature, dry skin, preserved reflexes and absence of palsy.

Heat exhaustion is known by the history of continued exposure to a high temperature, by the giddiness, pale face, weak pulse and insensibility.

Differential Diagnosis.

(1) They are diagnosed from *apoplexy* by the high temperature, the absence of palsy and unaltered reflexes.

(2) *From drunkenness* by the high temperature and different history.

What is the prognosis?

It is very difficult to give a general prognosis. No case is neces-

sarily fatal. Bad prognostic symptoms are the very high temperature, convulsions, profound coma, stertorous respiration and a full, labored pulse.

What are the sequelæ?

(1) The patient is usually unable to make any severe mental exertion for months or years after the attack.

(2) There is persistent vertigo.

(3) Irritability of the bladder.

(4) Sleepless nights.

(5) Giddiness.

(6) Confusion of mind.

All the sequelæ are aggravated by hot weather.

What is the pathology?

The vessels of the brain are intensely engorged. Sometimes a serous effusion is found in the cavity of the ventricles, which may, however, have occurred after death. The lungs are also congested. The blood is black and diffluent; the red corpuscles are altered in shape (a similar condition of the blood, brain and lungs, attended with similar symptoms, can be produced in animals by artificial heat); the heart is probably softened (?); the kidneys are sometimes congested.

How should these conditions be treated?

The patient should not be sent to a hospital unless it is within a very short distance. He must be removed from direct exposure to the sun, and care should be taken that there is no constriction of the circulation. If his temperature is high, he may be rubbed down with ice, or buckets of cold water may be thrown over him and cold-water enemata administered.

In congestive cases, leeching, cupping or general blood-letting may be resorted to. Should the temperature be very high, it must be lowered by the administration of antipyrine, either by the mouth or bowel.

If convulsions occur, morphine should be administered hypodermically, or small amounts of chloroform inhaled.

The bowels should be opened in all cases by turpentine enemata, by croton oil, or by a small injection of glycerin.

In cases of heat exhaustion, a stimulating, rather than depleting, treatment is called for, and ammonia and digitalis should be administered by hypodermic injection.

In any case, the patient must rest from mental and physical labors for a long time after the attack, and should pay great attention to his bowels and avoid direct exposure to the sun.

Delirium Tremens.

What is delirium tremens?

Delirium tremens is an affection occurring in persons habitually addicted to the use of alcohol, and particularly seen in those who have been drinking steadily for some time and taking but little food. It is characterized by sleeplessness, delirium and tremor.

It differs from mania-a-potû, which is often used as a synonymous term, by occurring after a prolonged debauch, the delirium usually being of a low muttering type, whereas the latter is an acute affection, occurring in persons rather unaccustomed to the effects of alcohol, and usually characterized by maniacal excitement.

What are its symptoms?

Insomnia is the earliest symptom. The patient soon becomes tremulous, is fanciful, has visions of all sorts, associated usually with a high degree of gastric irritability, a coated tongue, and a pulse at first full and large, but soon becoming small, more frequent (110–150), feeble and compressible. These visions are not always of an unpleasant kind; but sometimes they are associated with a fixed idea, as of the fear of bodily injury. In these cases the expression of the patient's face well accords with his terror. He is usually timid, easily impressed and cowed. Fever is always present except in the mildest cases. Delirium soon supervenes. It is not fierce or maniacal, but is associated with a great deal of movement, a constant "busy delirium" (Watson). During the stage of delirium there is a strong craving for alcohol, and it is only when the gastric irritability is very marked indeed that this craving is absent. Occasionally the patient is frantic and the pulse tense. These cases are

usually associated with active congestion of the brain or with meningitis, and are generally mania-a-potû rather than delirium tremens.

Sometimes a patient seems to be doing well, the symptoms ameliorate, he gets up from bed and suddenly collapses, death occurring very rapidly. This is probably due to cardiac paralysis.

Very rarely is the first sleep followed by entire cessation of the symptoms, although they are commonly ameliorated.

OTHER AGENTS BESIDES ALCOHOL will produce these symptoms. It will follow the withdrawal of opium in persons accustomed to its use, or from sudden breaking off of the chloral habit.

The author has seen several cases in which these symptoms appeared in middle aged or old men who had been excessive smokers all their lives, and who, for some temporary reason, had discontinued the use of tobacco.

How is this disease recognized?

It is recognized by the history, the insomnia, the tremor, and the delirium.

DIFFERENTIAL DIAGNOSIS.

(1) *From acute mania.* The characteristic tremor and the preponderance of visual hallucinations serve to distinguish it from this affection.

(2) *From acute meningitis.* The absence of headache and paralysis renders the diagnosis easy.

(3) *From general paralysis of the insane with muscular tremor.* In these cases the exaggerated delusions and the mental complacency are very different from the symptoms of delirium tremens, and when depression occurs, it is of gradual development.

What is the prognosis?

Mania a potû usually terminates favorably if the patient is prevented from doing himself bodily injury. In delirium tremens each recurrence becomes more dangerous, because of the organic changes produced in the viscera by the alcoholic habit; occasionally a patient, upon recovering from the attack, will be insane temporarily or permanently.

How is acute alcoholism treated?

No restraint should be used with the patient unless he exhibits

homicidal or suicidal tendency, as sometimes occurs in cases of mania a potû.

He requires good nursing and systematic feeding at regular intervals. A small amount of capsicum in pill will aid his digestion in cases associated with great irritability of the stomach. In addition to food, opium, morphine, or hyoscine should be administered. Bromide of potassium is also of service, especially when the face is flushed and the pulse strong; he should be kept perfectly quiet in bed and should use a bed-pan. The bowels should be opened by mercurial purgatives, but excessive purgation is to be avoided. In the majority of cases some stimulant must be administered, but the amount should be limited. Food is of more importance than stimulation in this affection. Whiskey, f℥iv in 24 hours, is usually enough to begin with, and as soon as the nourishment which he is taking tells on his exhausted vital forces, the amount of alcohol should be gradually diminished.

If the excitement continues in spite of the treatment, and especially if the delirium is fierce and active, he may be placed in a warm bath and cold water poured on his head.

After Treatment.

During convalescence bitter tonics should be administered and cocoa and oxide of zinc (gr. ij–iv *t.i.d.*) administered. An endeavor should be made to wean him from the alcoholic habit.

Chronic Alcoholism.

What is chronic alcoholism?

It consists in the changes produced in the various viscera by the habitual indulgence in alcohol; thus—

(1) *The brain* may undergo certain changes, the dura mater being thickened, the arachnoid opaque and the brain substance itself hardened, owing to the hyperplasia of the connective tissue. There is loss of memory and self-respect, with usually an inordinate craving for drink.

(2) The same changes may occur in *the cord*, producing the symptoms of sclerosis.

(3) *The nerves* also suffer, multiple neuritis being sometimes seen.

(4) *In the lungs* a low grade of inflammation, resulting in hyperplasia of the connective tissue, may be developed, causing chronic catarrhal pneumonia or fibroid phthisis. Chronic pleurisy is also often seen.

(5) *The heart* undergoes fatty and granular degeneration.

(6) *The liver* is indurated, the fibrous tissue increases, producing the hob-nail liver (cirrhosis).

(7 and 8) *The spleen and kidneys* undergo changes similar to those seen in the liver.

The treatment of chronic alcoholism has been discussed in connection with the various organs which are diseased.

The oxide of zinc, in gr. ij doses three times a day, has been highly lauded.

DISEASES OF THE SPINAL CORD AND ITS MEMBRANES.

Spinal Hyperæmia.

What is spinal hyperæmia?

Spinal hyperæmia is congestion of the vessels of the spinal cord, which may be either active or passive, and is due usually to cold, exposure, injuries, alcoholism, excessive venery, rheumatism or malaria.

What are the symptoms, diagnosis and treatment?

SYMPTOMS.

Dull pain in the back, increased by pressure, tingling sensation in the extremities, constrictive feeling around the abdomen, dragging sensation in the limbs on walking, all these symptoms being exaggerated by a recumbent position. The reflex and electro-muscular contractility are not impaired, but may be exaggerated.

DIAGNOSIS.

(1) *From anæmia.* In the latter the symptoms are somewhat

similar, but there is a different history, general pallor and weakness, and the symptoms are aggravated by the erect position.

PROGNOSIS.

The prognosis is usually favorable. If the case proves obstinate it generally terminates as a meningitis or a myelitis.

TREATMENT.

Rest, cups or leeches along the spine, followed by the application of ice, and active purgation by means of salines; ergot or belladonna with potassium bromide is given to diminish the amount of blood in the cord. *In the passive form* active depletion should be avoided. Digitalis and tonics may be administered and an occasional laxative given.

Spinal Meningitis.

What is spinal meningitis?

Spinal meningitis, also called *lepto-meningitis*, is an inflammation of the arachnoid and pia mater, caused by exposure, lying on damp ground, severe blows and injuries, rheumatism, phlebitis, or the extension of inflammation from the surrounding parts.

What are its pathology and symptoms?

The pathology is similar to that of inflammation generally. There is at first hyperæmia, followed by swelling, with a grayish exudation of lymph on the meninges of the cord, particularly the pia mater. It is usually a diffuse meningitis; the lymph often becomes organized, pressing upon different nerves and agglutinating the membranes together. Sometimes it may be a purulent inflammation.

SYMPTOMS.

There is pain and tenderness along the spine, increased by motion or pressure, and aggravated by the application of heat. The patient therefore lies absolutely still on his side or abdomen. There is marked girdle-pain around the waist; marked hyperæsthesia, the situation depending upon the seat of the inflammation; spasms in the muscles supplied by the part of the cord affected, usually clonic in character, coming and going. On close examination, quiverings may be detected in the fibrillæ of the muscles. The reflexes are usually

exaggerated. Electro-muscular contractility is good and may be exaggerated. There is moderate fever, temperature of 101° to 102°, being higher in the early part of the disease. Irritability of the bladder and rectum, the discharges at first being incomplete, later on becoming involuntary. If the upper part of the cord is affected, there will be difficulty in breathing without obvious cause. These symptoms all increase as the disease gets worse. There are marked alterations in the cardiac rhythm, the neurotic pain becomes more severe as the lymph organizes, the pupils are sometimes dilated, but otherwise there are no symptoms referable to the cerebellum.

What is the diagnosis, prognosis and treatment?

DIAGNOSIS.

(1) *From spinal congestion* by the fever, the greater severity of the symptoms, tendency to spasm, respiratory difficulty, alterations of cardiac rhythm, etc.

PROGNOSIS is favorable under treatment, especially if treated in the early stages.

TREATMENT.

The patient should be confined absolutely to bed, lying more particularly on the side and face, and, if seen in the earlier stages, cups or leeches should be applied along the spine, followed by ice. He should be freely purged, especially by salines; ergot, or belladonna combined with bromide of potassium, may be used to contract the vessels of the cord. As the acute stage subsides, a slightly mercurial impression is of advantage; it produces absorption and prevents organization of the lymph; if the mercury is not well borne, iodide of potassium may be substituted for it, which, if the case is of rheumatic origin, is more serviceable than the mercury.

In the later stages the application of iodine along the spine, or even of the galvano- or thermo-cautery, may prove advantageous.

Pachymeningitis.

What is this disease?

It is an inflammation of the dura mater of the spinal cord, characterized by violent pains, hyperæsthesia, followed by anæsthesia, caused by cold and exposure, excessive venery, syphilis, rheumatism

or caries of the vertebræ. It may become chronic and extend to the cord. It occurs usually in young males; is rare after thirty-five years of age.

What are the symptoms, prognosis, diagnosis and treatment?

SYMPTOMS.

The symptoms come on gradually with chills, fever, severe pains, hyperæsthesia of some parts and anæsthesia of others, pains shooting along the nerves which come from the affected parts of the cord, and the muscles supplied by these nerves undergoing constant contractions, which are often spastic in character.

The reflexes are rather exaggerated. When it is high up in the cervical region the pupil and heart are affected, and the spastic contraction may be such as to give rise to the condition known as "claw-hand." There are neurotic pains in the limbs, chest and abdomen, which are aggravated by motion. Gastralgia, with nausea and vomiting are common. There is some loss of power, but no actual paralysis.

PATHOLOGICAL ANATOMY.

Inflammation and exudation occurs on the under surface of the dura mater. This may organize, forming a mass of connective tissue, which, by pressing upon the spinal cord and nerves, gives rise to myelitis, neuritis and atrophy of the muscles supplied by the nerves coming from the parts affected. The new tissue may become vascular, producing hæmatoma.

DIAGNOSIS.

(1) *From congestion of the membranes*, by the severe pains, spastic contractions and fever.

PROGNOSIS.

If promptly treated in the early stages the prognosis is favorable, provided the cause does not continue.

TREATMENT.

Rest in bed, with frequent change of posture, ice-bags to the spine in the early stages, good nutritious diet and occasional laxatives. If the more acute symptoms have passed, blisters over the affected part of the spine frequently are of service. Potassium iodide and mercury should be administered, as in the other varieties of meningitis. When it is of syphilitic or rheumatic origin, the iodides are the best remedies.

Myelitis.

What is myelitis?

It is an inflammation of the substance of the cord itself, resulting in softening caused by an extension of the inflammation from the meninges, exposure to cold and damp, traumatism, phlebitis or pyæmia, or malnutrition of the cord, such as may be the result of low forms of fever.

It may be either an *interstitial myelitis* in the intercellular substance, or *parenchymatous*, where the cells themselves are affected. There is, however, no practical importance in the distinction.

What are the symptoms, diagnosis, prognosis, and treatment?

SYMPTOMS.

The symptoms are similar to those of meningitis, but are much more aggravated. There is more fever, more pain and more severe girdle pains in the early stages; spasms of the muscles and muscular twitching are at first prominent symptoms, but these, as well as the pain, rapidly disappear, giving place to palsies and to anæsthesia, which is often complete in parts below the portions of the cord affected. The reflexes are lost, the rectum and bladder beyond the control of the patient, and the nutrition of the parts is so affected that bedsores and sloughs take place and form one of the worst features of the disease. The temperature in the affected limbs is from 3 to 4 degrees below the normal.

The urine becomes highly alkaline, is apt to decompose in the bladder and give rise to chronic cystitis.

Electro-muscular contractility is absent.

PATHOLOGY.

The nerve-fibres are much swollen and their axis cylinders are beaded. Many granular and fatty cells are seen, and the perivascular spaces are infiltrated with leucocytes. The smaller veins are distended and varicose, and the "small arteries are often the seat of hyaline thrombi." The ganglion cells are swollen and distorted, their protoplasm granular and vacuolated, their nuclei if present are often undergoing division, and their polar processes are frequently invisible.

DIAGNOSIS.

The history of symptoms resembling meningitis, but much more severe and rapidly followed by complete paraplegia with loss of reflexes and electro-muscular contractility, render the diagnosis easy.

PROGNOSIS.

Prognosis is very unfavorable. It is a long disease under the most favorable circumstances. Sometimes extraordinary lulls occur, lasting for weeks and months, but recovery is next to impossible.

TREATMENT.

In the early stages it should be treated like meningitis, with ice, local bleeding, purgatives, mercurials, potassic iodide, and ergot and belladonna. Later, when paraplegia sets in and bedsores occur, the patient's strength should be sustained by food at short intervals; cod-liver oil may be given if it is well borne by the stomach, and iron iodide should be alternated with potassic iodide. *To prevent bedsores* the alternate application of heat and cold may be resorted to to keep up the nutrition and circulation of the part, and alcohol, pure or diluted with water one-half, or carbolized oil, may be applied, to harden the integuments.

The patient may be placed on a water-bed to equalize the pressure.

Spinal Apoplexy.

What is spinal apoplexy?

Spinal apoplexy, or spinal hemorrhage, is a sudden extravasation of blood occurring between the membranes or into the substance of the cord, generally following myelitis, traumatism or blood poisons, such as purpura, scurvy or pyæmia.

What are the symptoms, prognosis and treatment?

SYMPTOMS.

The symptoms occur suddenly, and are rapidly developed palsy, dependent upon the seat of the lesion; severe pain in the spine, radiating in the direction of the distribution of the nerve pressure, and more severe when the hemorrhage is superficial. There is no

fever at first except in those cases where a fever preceded the hemorrhage. The temperature in the palsied limbs is at first low; secondary fever occurs as the result of inflammation, but is not persistent; the reflexes and electro-muscular contractility are preserved, never being impaired except when the lesion is in the deeper parts of the cord.

PROGNOSIS.

If there is no complication, such as myelitis, which is in itself unfavorable, recovery will take place and the palsies readily yield to treatment.

TREATMENT.

Local bleeding in the early stages, followed by cold; the patient must be put absolutely at rest, laxatives must be given and ergot should be freely used. *For the resulting palsy*, massage, to keep up the nutrition of the parts, and iodide of potassium to produce absorption of the clot, is the best treatment in the early stages. Electricity should not be employed, nor strychnine administered, until after the inflammatory symptoms have subsided, and this treatment should be persisted in for a long time. For the pain morphine may be administered, combined with atropine, while belladonna or chloral liniment may be applied to the spine.

Acute Poliomyelitis.

What is this affection?

Acute poliomyelitis, or infantile paralysis, is an inflammation in the large ganglion-cells of the anterior horns of gray matter of the cord, which preside over the nutrition as well as the motion of the parts to which their nerves are distributed. It usually occurs in children between the age of three and six or eight years, and may be due to cold or damp, injuries to the spine, dentition, or may be developed as a sequela of an acute exanthemata.

It sometimes arises spontaneously. Occasionally it is seen in adults.

What are its symptoms and pathology?

SYMPTOMS.

It usually begins suddenly with a mild attack of fever lasting from two to four days, rarely a week. The child complains of some head-

ache and vertigo; occasionally convulsions occur. Within a week all these symptoms disappear to be followed by paralysis of one or both sides of the upper or lower extremities, one leg being usually much more affected than the other. The palsied limb wastes rapidly and the muscles feel soft and flabby. The reflexes are impaired or palsied. The electro-muscular contractility is lost to the faradaic current, and the galvanic current when applied shows the reactions of degeneration. Bedsores occur in paralyzed parts, but there is absolutely no pain in the limbs or elsewhere.

PATHOLOGY.

The large ganglion-cells of the cornua are at first inflamed, but rapidly undergo granular and fatty degeneration, beginning in the nuclei and spreading to the cell itself, which then atrophies. The interstitial connective tissue is increased. The degeneration spreads along the nerves arising from the cells, and the muscles which they supply also become granular or fatty and atrophied.

What is the diagnosis of this disease?

The diagnosis is easy except in the very early stage.

The physician rarely sees the child before paralysis takes place, when the diagnosis is clear.

What is the prognosis?

It is favorable if early treated.

What is the treatment?

In the early stages leeches should be applied over the spinal region, followed by cold; bromide of potassium must be given in decided doses, and, subsequently, iodide of potassium administered. Laxatives are given throughout the disease.

TREATMENT OF SUBSEQUENT PALSY.

The child should be placed under the best possible hygienic conditions; he should be well fed. A change of air and scene are desirable. Nutrients should be administered, as ferrous iodide, cod-liver oil, or phosphorus (the phosphorated oil of the Pharmacopœia is a serviceable preparation). Massage should always be used; muscle-beating is of service; the galvanic current should be applied to the atrophied muscles, and occasionally, as their electrical reaction improves, the faradaic current may be employed.

If deformities result, a suitable apparatus should be applied.

When this disease occurs in adults (rare), it differs in no respect in pathology or treatment from that just discussed. The cerebral symptoms and fever, however, are more pronounced and last for a longer time.

Chronic Poliomyelitis.

What are the synonyms of this affection?

It is also called progressive muscular atrophy and wasting palsy.

What are the causes?

It usually occurs between twenty-five and thirty-five years of age, and more frequently in males; exposure to cold and wet may produce it. Anxiety and mental distress are also causes. Occasionally traumatism plays a part in the production of the disease. Syphilis and an inherited neurotic tendency are also supposed to be factors in its production. In many cases no cause can be traced.

What is the pathological anatomy?

The muscles are wasted, and under the microscope the fibres are seen to have undergone granular and fatty degeneration, or to have undergone a vitreous degeneration, sometimes resulting in an almost entire disappearance of the fibre.

The peripheral nerve-fibres also degenerate, especially the axis cylinders connected with the anterior roots of the nerve, the posterior roots remaining normal. The spinal cord shares in the affection, particularly the anterior cornua of gray matter, and the antero-lateral white columns, especially in the cervical enlargement. The large cells in the anterior cornua undergo granular degeneration, and many disappear entirely, their processes also having degenerated; the pyramidal tracts of the gray matter, as also the anterior and lateral columns, undergo degeneration, and sclerosis occurs, sometimes traceable into the medulla oblongata.

What are the symptoms?

The disease usually commences in the arm, often preceded by

aching pain, and afterward wasting, accompanied by loss of power, which is usually first noticed in the shoulder-muscle, wasting being first seen in the muscles of the hand. The disease spreads from the first part affected to other parts of the same arm, and soon to the corresponding limb of the other side, and paresis or actual paralysis results, in proportion to the atrophy. In the forearm the flexors are usually involved before the extensors.

The muscles of the back become affected early in the case, and the muscles of respiration are usually involved sooner or later, constituting a source of great danger; wasting of the legs is not nearly as common as in the arms, although occasionally it may begin in this situation. If the wasting progresses slowly, there is a diminution of electro-muscular irritability both to the galvanic and faradaic currents; and, finally, as the wasting progresses the irritability is entirely lost, first to the faradaic, afterward to the galvanic current. The mechanical irritability of the muscles is increased, causing *fibrillation*, occurring spontaneously or when the fibres are lightly struck first in one bundle and then in another. Reflex action is abolished.

Pain is sometimes experienced, especially at the commencement of the malady, usually dull and rheumatic in character, sometimes accompanied by numbness; the muscular sense is preserved. The atrophied parts are usually cold and may be pale or livid.

The disease usually progresses steadily, although the rate of progression may vary; sometimes it is actually arrested for a time, particularly in the later stages of the disease. The chief danger to life is from the development of pulmonary diseases, which are very fatal on account of the weakness of the muscles of respiration. Death sometimes occurs from paralysis of the muscles of deglutition, sometimes from exhaustion due to bedsores or septicæmia.

How is this disease diagnosed?

It is recognized by the muscular atrophy occurring without uniformity in groups of muscles successively; by the abolition of the reflexes in the atrophied part; by the diminution of the electric reaction; by the fibrillation, and, perhaps, by harpooning a portion of the muscle and its subsequent examination under the microscope.

What is the prognosis?

The prognosis is very unfavorable where the two sides are nearly

equally affected; if the case is seen early the prognosis is more favorable. If the atrophy has existed for six months it will probably continue.

What is the treatment?

It is important that the patient have the best hygienic surroundings; gentle exercise should be taken, but fatigue of the body or mind avoided. Arsenic and strychnine are of service. Electricity does little good, except, perhaps, to keep up temporarily the nutrition of the muscles. Massage has been employed combined with passive movements without much effect.

Spinal Sclerosis.

What is this disease?

It is an interstitial myelitis producing an increase of the connective tissue of the spinal cord with atrophy of the nerve-structure from pressure. It may affect the antero-lateral columns particularly, or the posterior columns, or it may disseminate throughout the cord and even the brain.

What are its causes?

It occurs in persons of middle age and is often hereditary. It may be due to syphilis, mineral poisons, traumatisms, exposure to cold and damp, excessive venery and alcoholism.

What is the pathological anatomy?

There is a hyperplasia of the connective tissue of the cord, with granular degeneration and atrophy of the nerve-structure. The disease spreads in a longitudinal direction and the cord becomes atrophied and indurated; atrophic changes occur in the parts supplied by the nerves, which take their origin from the indurated portion of the cord.

Describe the varieties of spinal sclerosis, with their treatment.

(1) Antero-lateral Sclerosis.

Symptoms.

As the antero-lateral column of the cord and the great motor tracts are affected, this disease interferes with motivity chiefly.

In the early stages there is jerking and twitching of the muscles, with spasms and stiffness, followed eventually by entire loss of motion of the lower extremities.

During the progress of the disease the gait of the patient is characteristic. He walks on his toes bending his body forward, and developes a tendency to lose his equilibrium on the slightest push. Sensation is unaffected, the reflexes are preserved. Electro-muscular contractility is gradually impaired and finally abolished.

(2) Posterior Spinal Sclerosis.—Locomotor Ataxia.

Symptoms.

This disease comes on gradually, with darting pain in the limbs, spoken of as *lightning pains;* impaired sensation to contact in the feet, the patient being unable to distinguish between hard and soft objects when walking. This is gradually increased until there is entire abolition of sensation to contact, heat or pain in the lower extremities. The fingers are usually somewhat affected also, the patient having difficulty in buttoning his clothes. There is loss of coördination. He is unable to walk in a straight line with his eyes closed, and can only do so with his eyes open when he watches each step closely. He cannot stand with his feet together, if he closes his eyes or looks at the ceiling, without swaying from side to side, and finally falling. He cannot hop on one foot, and invariably walks with the aid of a cane. The sight is impaired, and even as an early symptom the Argyll Robertson's pupil is seen (a myotic pupil which responds to accommodation, but not to light).

The reflexes are abolished and severe girdle pains encircle the body, and are complained of at irregular intervals. The muscular strength is well preserved, although the coördination is lost.

(3) Multiple Sclerosis.

Symptoms.

It is also called cerebro-spinal sclerosis, longitudinal or disseminated sclerosis, and Charcot's disease. There is loss of power in the extremities, irritability of temper, slight delusions, loss of memory, and tremor occurring particularly when attempting any voluntary motion, as in raising a glass of water to the mouth. It affects one extremity at a time, is absent during sleep and while at perfect rest. There is constant oscillation of the eyeballs—a characteristic symp-

tom—and when the patient talks he dwells on his syllables, speaking in a monotone (*scanning speech*) ; this is also thoroughly characteristic. Later, contractions occur in the muscles, resulting in deformities resembling rheumatoid arthritis.

Diagnosis of Sclerosis.

Antero-lateral sclerosis. It is recognized by a jerking and twitching, followed by cramps in the muscles and the affected part, by the peculiar gait, final paraplegia, the preservation of the reflexes, and the abolition of the electro-muscular contractility.

Posterior sclerosis (locomotor ataxia), by the lightning pains, the impaired sensation, loss of coördination, abolished reflexes, Argyll Robertson's pupil, girdle pains and preservation of the muscular forces.

Multiple sclerosis, by the tremor on attempting voluntary motion, absent during sleep, by the rolling of the eyeballs and scanning of speech, the paresis and the contractures of the muscles.

Prognosis.

These affections may be retarded in their progress at times, but invariably terminate fatally. If due to syphilis, the prognosis is more favorable.

Treatment.

In all the varieties of sclerosis the patient should be put as completely at rest as possible, the diet should be nutritious and easily digested,—a milk diet is often of advantage. The oxide or nitrate of silver appears to have more effect upon this disease than other internal medication. Iodide of potassium, corrosive sublimate or the chloride of gold and sodium at times, especially in syphilitic cases, will retard the course of the malady.

The galvanic current applied along the spine, faradism of the muscles, the feet, limbs, etc., is sometimes of use. Prolonged hot baths have a tendency to prevent contractures both in the antero-lateral and in disseminated sclerosis ; in locomotor ataxia *suspension treatment* for a few minutes at a time in the very early stages appears to afford relief. The lightning pains may be relieved by antipyrine or other anodyne, opium being held in reserve. In the disseminated sclerosis hyoscyamine may be administered for the tremor.

Paralysis Agitans.

What is this affection?

Paralysis agitans, or shaking palsy, is a disease of the nervous system, more often seen among women than men. It is characterized by trembling, which is present all the time. The head may be still and the limbs tremble, or *vice versâ*.

This occurs even while the patient is quiet in bed, and affects the body generally, not one limb at a time, as in spinal sclerosis; there is no rolling of the eyeballs nor scanning speech. The patient walks on tiptoe as in antero-lateral sclerosis, and a slight push will upset his equilibrium; the head and body are usually bent forward. The handwriting is peculiar, the patient being unable to make a straight perpendicular stroke with pen or pencil. There is a progressive loss of power, and, as the disease progresses, entire paralysis.

TREATMENT.

Everything should be done to build up the nervous system. Good hygiene and nourishing food must always be insisted upon. Arsenic is of service. Phosphorus in pill or in capsule, if it is well borne, does good. Phosphates and cod-liver oil are also of use. Nutrition of the muscles may be kept up by weak faradization. For the trembling, hyoscyamine is the best agent.

Bulbar Paralysis.

What is bulbar paralysis?

Bulbar paralysis, or glosso-labio-laryngeal paralysis, is a progressive loss of power of the muscles of the tongue, lips, palate, pharynx and larynx.

What are its causes and symptoms?

CAUSES.

The causes are not well understood. It usually occurs in middle age and is believed to be the result of syphilis, rheumatism, gout, exposure to cold, and traumatism.

It is supposed to be due to the atrophy of the gray nuclei in the

floor of the fourth ventricle, which preside over the parts involved in the palsy.

SYMPTOMS.

The disease begins gradually with difficulty in articulation due to progressive paresis of the muscles of the tongue and lips. The soft palate and pharynx next become involved, causing difficulty in deglutition. The general health suffers, the paralyzed parts become atrophied, there is regurgitation of fluids through the nose and paralysis of the muscles of the palate, and the application of electricity shows the reaction of degeneration; the respiration is interfered with, and the patient dies usually from starvation.

The case lasts from one to five years.

TREATMENT.

Symptomatic. Iodide of potassium is occasionally of use.

Varieties of Tremor.

Describe the varieties of tremor.

In addition to the tremors occurring in paralysis agitans and multiple sclerosis, a variety of tremor is seen in the *atheroma of the vessels* resulting in malnutrition of the corpus striatum and of the anterior columns of the cord.

It is diagnosed from the former affections by the absence of paralysis, pain and the changes in the joints, there being a simple tremor, nothing else.

Treatment.

Phosphorus, good, nutritious diet, strychnine and alcohol should be administered.

TREMOR FROM ABUSE OF TOBACCO occurs mostly in middle-aged subjects, sometimes in young men. The daily amount of tobacco necessary to cause tremor may be very small, some persons being more susceptible to its influences.

Symptoms.

The tremor affects the upper extremity mostly; the heart is irregular; patient sleeps badly at night; is annoyed by sounds. The

tongue may be affected also, and the mucous membrane of the mouth altered, from the constant irritation of the poison.

The prognosis is good if habit is given up.

Treatment.

The cure is aided by exercise in the open air.

TEA DRINKERS' TREMOR.—The *symptoms and prognosis* are like those of tremor in tobacco.

Treatment consists in stopping the habit and the administration of tonics.

TREMOR FROM METALLIC POISONING.—This occurs mostly in persons who work in metals, particularly in mercury.

Symptoms.

In addition to the tremor, if it be due to mercury, the gums will be spongy, the patient salivated, and there will be a tendency to diarrhœa. If it is due to lead, there will be a blue or black line on the gum, the patient will be obstinately constipated, the subject of violent cramps, and perhaps have wrist-drop or other symptoms of lead poisoning.

The treatment consists in preventing further poison from entering the body, and eliminating that which is present by iodide of potassium and large amounts of diluents, while administering strychnine as a tonic.

Writer's Cramp.

What is the cause of writer's cramp?

It is more common in men than in women; it is particularly seen between fifteen and twenty years of age; there is often a neurotic family history and it is predisposed to by anything lowering the tone of the nervous system. It occasionally follows a local disease or injury; but the principal causative agent is the habit of excessive writing, particularly when the pen is held so that the small muscles are used in this act. It is also seen in telegraph and in type-writer operators.

What are the symptoms?

The symptoms vary in different cases. Usually there is muscular spasm, sometimes accompanied by pain, occasionally only a tremor

without any spasm, which discontinues as soon as the patient ceases to write. The spasmodic form is the most frequent and characteristic. After writing for some time he finds that the pen does not move as he intended it should; that every now and then the strokes are irregular; that he is grasping the pen too tightly, and that the index finger particularly has a tendency to slip from the pen.

The hands feel tired and there is pain in the first finger or thumb, or wrist, or forearm.

These symptoms continue for weeks, with a gradual impairment in the power of the muscle, gradually increasing in degree, with now and then an acute spasm which cannot be controlled. As the spasm increases it extends and involves more and more of the muscles of the forearm until finally all these muscles may be affected to such a degree as to render the movements of the hand impossible. Occasionally the symptoms develop acutely and rapidly. The spasm is usually tonic. It is frequently limited to the act of writing. Electric reactions may be normal or slightly increased or diminished. Occasionally there is a tingling sensation in the fingers in attempting to write. Sometimes the pain is severe and neuralgic in character. The spasm, pain and tremor occasionally affects the other arm also. The sensory symptoms have a greater tendency to extend than the motor. Sometimes if the patient accustoms himself to write with the left hand, it also may be involved. Occasionally the foot of the same side is involved in the cramp and pain.

What is the pathology of this disease?

No anatomical changes have been found. It is generally supposed to be due to a derangement of the action of the centres concerned in the act of writing.

What is the diagnosis?

The diagnosis is based on the spasm, pain and tremor, particularly the former, accompanying attempts at writing.

What is the prognosis?

The prognosis is uncertain when the disease is well developed, particularly if the patient continues to write.

What is the treatment?

(1) PREVENTIVE TREATMENT.

In writing, the characters should not be formed by motions of the fingers or of the wrist. If all persons wrote freely from the shoulder this disease would be much less frequent.

(2) FOR THE DISEASE ITSELF the treatment should be commenced early. There must be absolute rest from writing for a prolonged period, and when it is begun it should be commenced gradually. A fountain pen with a rubber handle, or a cork pen-holder, or a large quill should then be used. If the patient must write, he should learn to write with his left hand, or he should use a type-writer.

Strychnine and other nerve tonics must be administered.

In the neuralgic form morphine may be necessary. The application of ointments of belladonna or of aconite are also useful. Cocaine is efficient in some cases.

If tenderness exists over the nerve, blisters should be applied over their course. Galvanism is sometimes of use in either variety. Massage and regular gymnastic exercises of the fingers do good.

Convulsions.

Describe convulsions.

Convulsions generally occur between two and five years of age, and are produced by reflex causes, as dentition or the presence of worms in the alimentary canal; by the onset of some acute febrile malady, in which case there will be a high febrile temperature; by uræmia, when there will be albumen and casts in the urine, with more or less dropsy, or at least œdema of the eyelids, or there may be tetanoid convulsions which are epileptiform. They may be also due to hysteria or to certain poisons, as strychnine. Among other reflex convulsions may be mentioned those occurring in hydrophobia or tetanus.

Convulsions recurring after puberty without elevation of temperature, and with no casts in the urine and only temporary albuminuria, are usually due to epilepsy.

SYMPTOMS.

The face becomes dusky, the facial muscles jerk spasmodically.

There is rolling of the eyes, perhaps a squint or other departure from the natural condition, frothing at the mouth, the head and neck are drawn backwards or turned toward one side, the muscles of the back are rigid and the extremities are violently thrown about, consciousness and sensation are lost, the eye is insensible to light, and the pupils either dilated or contracted, at any rate immovable, the breathing is hurried and labored, the pulse quick, the heart irregular, the urine and fæces are discharged unconsciously, and a clammy moisture breaks out over the entire body. The above condition may last from a minute or two, to an hour or two, when the child falls asleep, or cries loudly on regaining consciousness, or sinks into coma.

PROGNOSIS.

If the child lives twenty-four hours after the attack he is apt to recover; if he inherits a tendency towards convulsions and suffers from them repeatedly, his condition is dangerous. In older persons, however, recurring convulsions are generally not fatal.

Convulsions occurring in whooping-cough, in croup, and in epilepsy, or after long exhausting diseases, are rarely fatal.

TREATMENT.

Remove the cause. In children look at the teeth, and lance the gums if necessary. If worms are suspected santonin and calomel should be given. If it is due to indigestion free purgation gives the best result. In any event laxatives should be promptly administered and cold applied to the head.

The child should be placed in a warm bath, and if its temperature is high, cold water may be poured over its head until the heat of the body is reduced, or antipyrine may be administered internally; the bromides or chloral alone, or combined, are of the greatest service. In uræmic convulsions chloral is the most efficacious remedy, with the exception of the inhalation of chloroform.

When convulsions occur in children who have been the subject of summer complaint or other protracted disease, or appear to be due to anæmia of the brain, chloroform or the bromides may be used temporarily to control the convulsions, and in case of diarrhœa, the discharge should be promptly arrested by small doses of opium, after which the nutrition of the child should be carefully attended to and stimulants should be administered.

Paralysis.

What is paralysis?

Paralysis is a loss of the motor function of a part, and is a symptom of disease, not a disease itself.

(1) It may be HEMIPLEGIA—loss of power of one side of the body. In this case the face at first is paralyzed, but soon regains its power. The arm and leg of the same side are more or less paralyzed; sensation, as a rule, is not impaired. Atrophy of the paralyzed muscles and cutaneous disturbances occur on the injured side, showing trophic changes. The patellar reflex and electrical reactions are preserved. *It is generally of cerebral origin.*

(2) PARAPLEGIA is double and complete paralysis, generally affecting the lower limbs, but the arms may also be involved. The bladder and rectum are affected. The reflexes are abolished. Electric reactions are wanting. Atrophy and cutaneous eruptions occur, and there is a tendency to the formation of bedsores.

It is generally of spinal origin.

(3) MONOPLEGIA is a local paralysis due to the affection of the motor centre, which presides over the motivity of the part.

(4) FUNCTIONAL PARALYSIS may occur from pressure on the nerves or from poisons circulating in the blood, as lead, etc.; from nerve exhaustion, as after long exertion and in hysteria.

TREATMENT.

Remove the cause, if possible, and keep up the nutrition of the patient. Guard against bedsores, and prevent contractures of the paralyzed part by frequent soaking in hot water. The muscles and nerves should not be stimulated by electricity too early in the case. It is better to wait until the cases become chronic, or, in other words, until the irritation due to the central lesion has subsided. As a rule, they should not be employed until the third month.

TABULAR VIEW OF PARALYSIS.

SYMPTOMS.	SEAT OF LESION.
Inability to move leg and arm of one side. Sensation unimpaired, or slightly impaired. Incomplete paralysis of muscles of face; mouth drawn toward healthy	Corpus striatum, involving internal capsule, both on side opposite to the palsy.

Symptoms.	Seat of Lesion.
side. Electro-muscular contractility preserved; may be increased; so may be the reflex excitability of the tendons.	
Same symptoms, but less palsy; some impairment of sensation; absence of vasomotor symptoms; early tonic and clonic spasms in hand, face and neck.	Optic thalamus.
Same symptoms, but less paralysis of face on opposite side to that of arm and leg, and usually marked loss of sensation on one side of face, and unilateral anæsthesia or hyperæsthesia of limbs; giddiness; nausea; heightened temperature; convulsions; contracted pupil. Urine may contain sugar or albumen. Early rigidity of paralyzed muscles.	Pons varolii, on side opposite to palsy of limbs. The part affected is below decussation of facial nerve.
Same symptoms, but face paralyzed on both sides.	Pons varolii, and at level of decussation of facial nerve.
Paralysis of arm and leg on one side; slight paralysis of face; third nerve paralyzed on other side; defective sensation; higher local temperature.	Crus cerebri on side corresponding to paralysis of third nerve.
Paralysis of motion of arm and leg, incomplete and transitory, soon followed by rigidity; no loss of sensation; reflexes, superficial and deep, preserved or increased; localized pain in head; convulsions.	Cortical part of brain in motor zone on side opposite to palsy.
Motion more or less completely affected on both sides of body; sensibility diminished or lost on one side, increased on the other; higher temperature on one side.	Medulla oblongata on side of increased sensibility and temperature, and at level of decussation of anterior pyramids.
Both legs and lower part of trunk paralyzed as to motion; loss of sensation; some wasting of muscles; loss of power over bladder and rectum; reflex excitability in legs heightened; trunk reflexes impaired; electric contractility diminished or lost; trophic changes; paralysis of muscles of respiration in some instances.	In the cord throughout its sections, above the lumbar enlargement, as in transverse myelitis of the dorsal cord.
Both legs paralyzed: muscles of legs flaccid; feet extended; anæsthesia; incontinence of urine from the start; superficial and deep reflexes lost; rapid wasting of muscles; reaction of degeneration; trophic changes.	In the cord, in lumbar enlargement, as seen in myelitis of these parts.
Arms as well as legs paralyzed, otherwise symptoms much the same; affection of pupils.	Cervical region of the cord, as in cervical myelitis.
Both legs rapidly paralyzed as to motion; relaxation of muscles; sensation unimpaired; only transient loss of control over bladder and rectum; marked lowering, or extinction of reflex excitability in the palsied muscles and tendons; lost electro-muscular contractility to faradic current; rapid muscular atrophy; no bedsores; if disease becomes chronic, muscular contractions.	Anterior horns of the cord, as in degeneration of the cells in acute poliomyelitis.

(From Da Costa's "Medical Diagnosis.")

Facial Paralysis.

Describe facial paralysis.

Facial paralysis, or Bell's palsy, is due to inflammation of the portio dura nerve from cold, exposure, rheumatism, pressure, etc.

Symptoms.

Complete paralysis on one side of the face. Patient cannot close his eye because the orbicularis palpebrarum is paralyzed. The face is drawn towards the opposite side; he laughs and cries on one side of his face. The corner of the mouth on the paralyzed side is depressed, and there is dribbling of saliva.

If the deeper portion of the nerve is paralyzed also, ringing in the ear from involvement of the tympanic branch, and paralysis of the uvula may occur, and deglutition be seriously affected.

The reflexes are abolished and electro-muscular contractility is impaired. A palsy of cerebral origin may involve the facial nerves, but in these cases the reflexes are preserved, as is also the electro-muscular contractility.

Prognosis.

As a rule, the prognosis is favorable. If the electro-muscular contractility is preserved, it is favorable. If it reacts only to a feeble galvanic current, recovery will be slow. If there is any reaction to electricity, it is favorable.

Treatment.

When due to cold and exposure or rheumatism, if seen early, leeches should be applied over the nerve behind the ear; if not seen until later, blisters should be employed.

Early in the case diaphoretics are indicated; afterwards, iodide of potassium should be administered. Later, strychnine should be employed hypodermically, and the potassic iodide treatment continued.

The faradaic current may be applied daily over the affected nerves and the muscles. If the faradaic current does not act, or the disease is very deep-seated, the galvanic current gives better results.

Syphilitic Paralysis.

How is this variety recognized and treated?

The diagnosis of this form of paralysis is extremely important, as it renders the prognosis more favorable, and indicates the line of treatment. Syphilitic paralysis is recognized by its history.

Paralysis occurring in young men, as a general thing, is from syphilis. When it occurs in women, the disease has frequently been contracted from their husbands. The presence of syphilides, even though slight, should always be sought for.

Osteocopic pains are generally present. The palsies are irregular, shifting and multiple. There is a rapid improvement under treatment, which confirms the diagnosis.

The infection may have occurred in early life and paralysis not develop until middle life or old age.

The treatment consists in large doses of mercury and potassic iodide.

Neurasthenia.

What is neurasthenia?

Neurasthenia, called *nerve-exhaustion*, is characterized by symptoms rather of depression than exhaustion of the nerve centres of the brain and the cord.

THE CEREBRAL TYPE.

This is the most common variety in men.

What is the cause?

Long-continued overwork or mental worry: it often follows influenza.

What are the symptoms?

The memory is defective; he forgets the names of objects and does not speak fluently, although previously he may have spoken well. His temper is irritable; he suffers from headache on reading or performing any brain work. His mind is perfectly good for a short time, but the nervous depression is shown by the fact that the cerebral powers soon give out.

His nights are sleepless or restless; he suffers from indigestion,

is sleepy after meals; his limbs are numb; his tread not so firm as formerly, and any slight pressure on the nerves causes numbness and tingling, persisting for some time.

He is hypochondriacal and loses his morale; he is apt to indulge to excess in any taste which he may have, such as for smoking or drink.

What is the prognosis?

It is favorable under proper treatment.

SPINAL TYPE.

This is the most common variety in women. It may occur among males also.

What are the symptoms?

There is sensitiveness on pressure along the spine, the slightest pressure producing pain. The hands and feet are cold, the menstrual flow disordered; she is peevish and irritable, and becomes a household tyrant.

There may even be some loss of power in the extremities.

The symptoms are sometimes the same as in the cerebral variety, with the addition of irritable spine and hysteria.

What are the causes common to both varieties of this affection?

It is often produced by sexual excesses, by the abuse of alcohol, opium, or other nerve-sedatives, as potassium bromide, chloral or cocaine. A nervous diathesis probably underlies the affection and acts as a strong predisposing cause.

How is it treated?

Change of surroundings is a prominent element in the cure, provided the patient will leave his business cares behind him.

He must have no worry of any kind, must not study or read to fatigue; the diet should be good, beginning with milk and feeding him up.

Passive motion and massage is of great value; electricity may be employed, especially the static form of electricity.

Good mental and moral influences play an important part in the treatment. Tonics, as arsenic, strychnine, ignatia and the bitters, must be administered.

THE NEUROSES.

Chorea.

What is chorea?

Chorea is a disorder of the nervous system characterized by irregular muscular movements. It has been called "insanity of the muscles." It is classed among the functional nervous disorders, as the seat of the affection has not been ascertained. It has been thought by some to be situated in the corpora striata and ganglia, connected with motion at the base of the brain, due to capillary embolism in this situation. Others think the peduncles or medulla oblongata are the seat of the disease.

Clinically, however, neither view is substantiated.

What are its synonyms and causes?

It is also called *St. Vitus's dance.* The causes are rheumatism, inherited or acquired; fright; sexual excesses, etc., or it may be of reflex origin, as from masturbation, elongated prepuce, dentition and intestinal disorder.

It is usually a disease of childhood, not infrequently occurring about the age of puberty. Occasionally it is seen in infants, sometimes in adults.

What are its symptoms, diagnosis, prognosis and treatment?

SYMPTOMS.

Chorea differs much in violence in different cases. In the mild cases there may be only a tendency to jerk or twitch various groups of muscles, aggravated by the knowledge on the part of the patient that she is being observed. The irregular motions may be confined to the hand, face or leg of one side or both sides may be affected. The child becomes cross and irritable, and is apt to drop anything she picks up. The hands and arms are usually first affected, then the face, and lastly the legs. In the more severe forms the disease becomes general, and any or all of the muscles of the body may be affected so that the patient is completely helpless, being unable to feed herself and even to talk. Mania is sometimes present, more frequently in adults than in children, and especially in preg-

nant women, when the disease is often truly frightful, often ending in abortion and death.

Muscular weakness is a common symptom and anæmia is often present.

DIAGNOSIS.

The diagnosis is easy. The age, the history and incessant muscular action, and the absence of other nervous trouble, make the case clear.

PROGNOSIS.

The prognosis is favorable, though the disease may be of long duration.

TREATMENT.

In the treatment of chorea, *sleep* is of the utmost importance. The patient should go to bed early and get up late.

If sleep does not come naturally hypnotics should be administered, as bromides or chloral.

The diet should consist of easily digested food, avoiding meat, as a rule, unless anæmia is a pronounced feature in the case. The patient should have plenty to eat.

During the waking hours he should lead a life in the open air, with systematic exercise, but not to fatigue. Cold baths may be used in addition to the tonic treatment.

Arsenic is one of the best remedies. Hyoscyamine is also of use.

Epilepsy.

What is epilepsy?

Epilepsy is a chronic disease characterized by a sudden and complete loss of consciousness, attended with convulsions followed by a deep sleep and temporary albuminuria, manifesting a tendency to recur at intervals of variable length.

What are its causes?

Idiopathic epilepsies are generally inherited. The causes are fright, overwork, sexual exhaustion, syphilis or reflex causes, as disordered digestion or uterine disease. The irritation due to an elongated prepuce is sometimes an exciting cause.

Symptomatic epilepsies are due to an injury of the brain, to sun-

stroke, or to pressure caused by a tumor or by thickening of the membrane of the brain.

The seat of epilepsy has not been ascertained.

What are its symptoms?

All cases of epilepsy may be classed under one of two heads, which, however, are different in degree rather than in kind.

In the more severe form, the grand mal, there is usually a sensation preceding the attack as of something passing from the extremities or from any part of the body towards the head, which is called the *aura epileptica*.

During the attack the patient frequently utters a peculiar cry and falls to the ground in convulsions, usually unconscious and foaming at the mouth. The face is pale, and at first the muscles are in a state of tetanic rigidity, succeeded in a few minutes by general clonic convulsions, during which the patient frequently bites his tongue. The pupils are fixed.

After a longer or shorter interval he comes to himself in an exhausted condition, entirely unconscious of what has occurred, and falls into a heavy sleep from which he awakes heavy and dull, and frequently with a headache which may last for some time.

The urine will contain albumen for several days after the attack.

In the interval between the attacks the patient is well, but if they recur too frequently his intellect is apt to suffer, the memory being gradually destroyed.

The number of attacks varies greatly. Some cases are reported in which over 2000 fits occurred in a year. In others there may be only one or two in the course of several years, while sometimes, from some passing cause, the patient may have an attack which may never be repeated.

In the lighter variety, le petit mal, the loss of consciousness is of but momentary duration. The patient pauses in what he is about, feels momentarily dizzy and sometimes experiences a slight convulsive twitch.

How is this disease diagnosed?

The diagnosis is not always easy. The aura, the cry, the unconsciousness, convulsive movements, foaming at the mouth, biting of the tongue, the sleep and temporary albuminuria, and the fact that

the patient in falling frequently hurts himself, are the points on which a diagnosis is based.

DIFFERENTIAL DIAGNOSIS.

(1) *From uræmic convulsions.*

The diagnosis is based upon the history—the dropsy with albumen and tube casts as a constant feature—the stupor between the convulsions, and the frequency of the recurring paroxysm in the latter disease.

What is the prognosis and treatment?

PROGNOSIS.

If the cause can be ascertained and removed the patient may be cured. Usually the frequency of the paroxysms may be materially lessened, although they cannot be entirely prevented.

It is a chronic disease.

TREATMENT.

The attack may be sometimes averted by the inhalation of a few drops of amyl nitrite, when the aura is experienced, or by compressing the body between the aura and the head.

During the attack the clothing should be loosened and a folded cloth placed in the mouth to prevent the patient biting his tongue, and he himself placed in such a position as will not interfere with his respiration nor allow him to injure himself during the convulsions.

When he is coming to, throw a little cold water in his face and let him smell ammonia.

After the paroxysm allow the patient to sleep as much as he wants, giving him a brisk cathartic when he wakes.

To prevent the return of the attack remove the cause if possible and avoid all irritating articles of food, placing the patient as nearly as possible on a diet of milk and vegetables.

The best remedies in epilepsy are the bromides, which are better given combined than separate. They should be given at first in doses sufficiently large to destroy the reflex irritation of the pharynx, as shown by the absence of vomiting when a feather is applied to the fauces and the palate. When this effect is obtained the dose should be lessened, but should be sufficiently large to maintain the effect for a long continued period.

If it is desirable to give one of the bromides only, at a time, instead of combining several of them, it is better to change the preparation occasionally, as in this way the effect will be better and there will be less likelihood of bromism.

DISEASES OF THE NERVES.

Neuralgia.

What is neuralgia?

Neuralgia is a nervous affection, characterized by sudden, sharp pain following the course of the sensory nerves, usually confined to the left side, and unattended with inflammation of the nerve structure. It may affect any nerve in the body.

What are the causes?

It is often hereditary, or may be due to anything which depresses the nervous system or weakens the constitution of the patient, as anæmia, syphilis, metallic poisoning, malaria, exposure to cold and damp, and traumatism.

What are the symptoms, diagnosis, prognosis and treatment?

Symptoms.

A darting, stabbing pain, sudden in its onset, is felt in the course of the affected nerve. There is no swelling or redness, but there is often a feeling of vague tenderness.

Points of tenderness on pressure are found where the nerve leaves its bony canal, where it enters the muscular tissue, and where its filaments become cutaneous. In chronic cases, nutritive changes are produced in the part to which the nerve is distributed.

Diagnosis.

It is recognized by pain limited to the course of the nerve, by the points of tenderness, by the absence of fever or of inflammation, by the paroxysmal nature of the affection, and the fact that the pain is not increased on motion or by pressure.

Prognosis.

When promptly treated the prognosis is favorable.

There is, however, a tendency to recurrence as long as the cause exists.

TREATMENT.

The patient should be well nourished and the cause, if possible, removed. The condition of the blood should be looked into, and iron or arsenic administered should anæmia exist.

In rheumatic or gouty diatheses potassic iodide, colchicum or ammonium chloride, should be given.

If there is any suspicion of malaria, quinine and arsenic are the remedies to be employed.

To relieve the pain, veratrine ointment, belladonna plasters or ointment, counter-irritation by mustard plasters or solutions of menthol may be applied, while antipyrine or its congeners, cannabis indica, atropine, tincture of aconite, or morphine may be administered, or morphine or cocaine given hypodermically. The continuous current applied over the course of the nerve is often of advantage in obstinate cases.

Cerebral Neuralgia.

What is this affection?

Cerebral neuralgia, *sick headache*, *hemicrania* or *migraine*, is usually one-sided, and consists of a severe pain, lasting for twelve to forty hours. The eyes are injected; usually there is nausea and vomiting; the attacks tend to recur, in women, generally coming on at the menstrual period. In these cases the pain is not confined to the head, but is felt in various parts of the body. The intervals between these attacks is usually about four weeks.

The cause is unknown. It is said to be due to vasomotor disturbance with a cerebral tendency.

TREATMENT.

During the attack the bromides and chloral, in decided doses, with brisk purgation to relieve the abdominal circulation, are sometimes efficacious. Fluid extract of guarana, half a drachm to a drachm every hour until relief is obtained, has been highly recommended. Cocaine and various other drugs are sometimes of use. Hot

mustard foot baths and hot-water bags or mustard should be placed to the back of the neck. In the interval between the attacks endeavor to build up the patient's health with arsenic, strychnine or zinc, good hygiene, exercise and rest.

Neuralgia of the Fifth Pair of Cranial Nerves.

Describe this variety of neuralgia.

The upper branches of the fifth pair of nerves are usually thus affected, but one or all of them may be the seat of neuralgia.

The pain is generally confined to the left eye, is very violent, extending to the forehead and cheek, and is intermitting in character. There is great tenderness over the foramen of exit, the teeth and other parts supplied by this nerve; lachrymation; altered nutrition of the parts may cause the hair to turn gray on that side, or cutaneous eruptions to develop. Sometimes there are also painful spasmodic twitchings, from reflex irritation of the facial nerve, to which the name of *tic douloureux* has been given.

CAUSE.

The causes are those of all neuralgia generally; pressure from thickening of the bony foramen of exit often produces it.

TREATMENT.

The condition of the blood should be promptly attended to, and quinine, arsenic or iron administered. Gelsemium is very useful, especially if there is any tenderness of the nerve. For this purpose the tincture should be employed, in doses of five drops every three hours until physiological effects are obtained. Antipyrine is also recommended.

Locally, an application of equal parts of croton chloral and camphor, or of aconite often gives relief. Menthol is often of service. If all else fail morphine may be used. In very obstinate cases neurotomy may be performed, or nerve-stretching may be practiced.

Cervico-Occipital Neuralgia.

Describe this variety.

It manifests itself by a severe paroxysm of pain in the cervico-occipital region, with spots of tenderness over the nerves, and often herpes due to trophic changes in the skin; spasmodic twitching in the muscles occurs.

It is usually seen in women, and follows superlactation or other debilitating causes connected with parturition.

TREATMENT.

The treatment is the same as that of neuralgia generally.

Cervico-Brachial Neuralgia.

Describe this variety.

SYMPTOMS.

Patient suffers from paroxysm of pain in the arms and fingers, with hyperæsthesia to touch or to contact, and a subjective feeling of numbness and tingling. There are sensitive spots along the affected nerve, at the point of exit from the spine, at the deltoid and at the elbow. Motor disturbances also usually occur, on account of the mixed character of the nerve.

CAUSES.

The causes are those of neuralgias in general; fractures or other traumatism often produce this variety of neuralgia.

TREATMENT.

The treatment is that for the neuralgias in general. The iodide of potassium may often be of service; blisters over the course of the nerve should not be neglected.

Dorso-Intercostal Neuralgia.

Describe this variety.

SYMPTOMS.

The symptoms of dorso-intercostal neuralgia are pain and tenderness, as in other varieties, affecting the parts around the shoulder

and side. Herpes zoster, which consists of a series of painful vesicles developing along the course of the nerve, due to nutritive disturbances, is a frequent symptom.

CAUSES.

Dorso-intercostal neuralgia is especially seen in men, and often in those who are subjects of a gouty, lithæmic or rheumatic diathesis.

TREATMENT.

Treat the cause as in the other varieties of neuralgia and give quinine; locally, the herpes may be powdered with bismuth subnitrate or zinc oxide, or some soothing unguent applied.

Intercostal Neuralgia.

Describe intercostal neuralgia.

SYMPTOMS.

There is pain in one or two of the intercostal spaces, with painful points at the foramina of exit, at the sides of the chest, in the axillary line and in front where the nerves become superficial. The breathing is often shallow and quickened and the heart may become embarrassed.

CAUSE.

This variety is seen usually in women suffering from uterine or vaginal disease. Anæmia is a common cause.

TREATMENT.

The treatment is the same as that of neuralgia generally, and locally, in obstinate cases, tincture of iodine may be applied over the sensitive spots, or blisters may be used.

Lumbo-Abdominal Neuralgia.

Describe lumbo-abdominal neuralgia.

SYMPTOMS.

It consists of a paroxysmal pain following the course of the upper branches of the lumbar plexus. Herpes is sometimes seen.

CAUSE.

Malaria is the most frequent cause.

TREATMENT.

The same as of neuralgias in general is indicated.

Sciatica.

Describe sciatica.

SYMPTOMS.

Sciatica is usually one-sided, affecting generally the left side. There is violent, shooting pain, darting along the course of the nerve, and more or less continuous. Sensitive spots are found along the course of the nerve, near the trochanter, in the popliteal space, along the tibia and fibula and in the foot, and there is also tenderness over the dorsal vertebræ. The patient is irritable, and there is usually more or less loss of motion. Nutritive changes, as dwindling of the muscles, clubbing of the toe nails, and herpes frequently occur.

CAUSES.

Exposure to cold and damp, rheumatic diathesis, local injuries, and pressure from accumulation of fæces or an intra-abdominal tumor. In the latter case, one or both sides may be affected.

PROGNOSIS.

Usually good, if properly treated.

DIAGNOSIS.

(1) *From muscular rheumatism* by the sensitive points, and by not being increased on motion.

(2) *From hysterical joint* by the history and the pain along the course of the nerve only. In the latter affection the sensitive points disappear when the patient's attention is distracted.

TREATMENT.

General treatment of neuralgias. Iodide of potassium is of service, as is blistering over the nerve and between the trochanter and the tuberosity of the ischium. The application of points of hot metal (previously heated by dipping in hot water) along the course of the affected nerve is sometimes of service. Acupuncture is recommended by Hammond: hypodermic injections of chloroform, by Bartholow. Phenacetin or antipyrine are sometimes successful. Hypodermic injections of atropine and morphine generally give relief, which may be permanent. The injections should be deep-seated. When due to impacted fæces, very active purgation with croton oil, aloes, and colocynth should be resorted to.

Neuritis.

What is neuritis?

It is an inflammation of the trunk of a nerve, due to traumatism or cold, attended with pain and loss of power in the parts supplied by the affected nerve.

PATHOLOGICAL ANATOMY.

Congestion, followed by inflammation of the nerve tissue, with proliferation of cells and migration of the corpuscles are found, as in inflammation generally.

SYMPTOMS.

There is slight fever, with an intense burning pain, increased by pressure and motion, felt along the course of the nerve and at its distribution. If a motor nerve is affected, spasmodic contraction of the muscles, followed by paresis, occurs. Should the nerve be destroyed by the inflammatory action, there will be atrophy of the muscular tissues to which it is distributed. Various trophic changes also occur, as cutaneous eruptions and clubbing of the nails. Electro-muscular contractility is impaired and finally lost.

PROGNOSIS.

Generally favorable under proper treatment.

TREATMENT.

Repeated blistering along the course of the nerve, with full doses of potassic iodide, should be given.

As the acute symptoms subside, weak currents of electricity may be employed.

For the intense pain, hypodermic injections of morphine should be used.

When due to cold and exposure, in the very early stages, prolonged warm baths, or profuse diaphoresis from pilocarpine, are of decided advantage.

Multiple Neuritis.

What is multiple neuritis?

It is a disease usually due to chronic alcoholism, characterized by symmetrical paralysis of the extremities, particularly affecting the

muscles supplied by the musculo-spiral and anterior tibial nerves, associated with pain and tenderness, localized in the nerves, particularly in their peripheral endings, and accompanied by wasting of the muscles and impaired sensation over the distribution of these nerves.

What are its synonyms?

It is called *polyneuritis* and (a bad name) *disseminated neuritis.*

What are its causes?

Primary multiple neuritis may affect either sex, but is more common among females, particularly when due to chronic alcoholism. It occurs especially between the ages of thirty and fifty years. It may also be caused by grief, exposure, long-continued heat, or altered conditions of the blood, as in septicæmia. It sometimes occurs associated with the debility which follows acute or chronic diseases.

What are the symptoms?

It may begin either as an acute or subacute disease, but is rarely chronic from the start. If it begins acutely, there will be chills, fever, temperature from 102° to 104°, and general constitutional disturbance, lasting from two to four weeks, associated with tingling in the fingers and toes, sometimes preceded with a vague rheumatic pain.

Soon actual pain occurs, dull at first, but increasing and becoming acute, burning and darting, generally increased by motion, and felt along the course of the nerves or in the parts to which they are distributed. In subacute or chronic cases there is not much pain.

Tenderness of the nerves and muscles is experienced, and there is progressive muscular weakness, with loss of power, affecting the extensors and supinators of the forearm, the extensors of the toes and the flexors of the ankle. The other muscles of the limbs are also affected, but to a lesser degree. The muscles become flabby and atrophied, the nerves lose their electrical excitement, and the reactions of degeneration are present in the muscles.

Anæsthesia to contact often exists even when hyperæsthesia to painful impression is present.

The reflexes are lost early in the disease; incoördination of mus-

cular movement is sometimes present, and trophic changes in the skin, nails and hair usually occur. In cases of moderate severity, the symptoms increase for from four to six weeks, then become stationary for several months and slowly improve. Sometimes the muscles least affected undergo secondary contracture; relapses are apt to occur if the cause of the disease is still in operation.

In severe cases, complete paralysis of the muscles of the limbs rapidly ensues, and the muscles of the trunk also becoming rapidly affected, paralysis of the muscles of the diaphragm and death may occur. Occasionally cardiac failure takes place.

What is the pathological anatomy of this affection?

In recent and acute cases the nerves are red and swollen, and sometimes minute hemorrhages are seen in the connective tissue and nerve-sheath. The sheath, and also the septa between the fasciculi, are infiltrated by embryonal cells. The nerve fibres undergo acute granular degeneration. These changes are especially peripheral, and are confined especially to the nerves of the limbs, the musculo-spiral nerve of the upper extremity and the anterior tibial of the lower, being the first to suffer and being more severely affected than the other nerves of the limbs, which also become involved. The muscles which these nerves supply degenerate and become atrophied. It is a peripheral disease, the nerve trunks being rarely affected, and the anterior roots being healthy. The spinal cord, although generally healthy, sometimes shows signs of chronic myelitis.

How is the disease recognized?

Diagnosis is formed from the combination of motor and sensory symptoms, their localization to the periphery of the limbs, and the tenderness along the nerve trunk.

What is the prognosis?

If the muscles of the trunk and of respiration are not involved, the prognosis is good in uncomplicated cases. Entire recovery usually takes place within a year, if the patient will avoid the cause of the disease.

What is the treatment?

In the acute stage absolute rest, a light, nutritious diet, and the

occasional use of purgatives are indicated. Alcoholic drinks should either be avoided entirely or their amount much reduced.

Warm baths and warm applications to the limb are often of value.

Gentle massage may be employed from the first, and afterward passive motion practiced, to lessen the tendency to contraction.

If there is much fever, the usual febrile treatment should be employed, and when this stage is passed, iron and quinine, with small doses of nux vomica, administered. Cod-liver oil and attention to general nutrition are important adjuvants in chronic cases. A mild galvanic current may be applied to the muscles after the acute stage is past.

INDEX.

ABSCESS, hepatic, 179
splenic, 191
Addison's disease, 119
Ætiology, definition of, 18
Ague-cake, 58
Alcoholism, chronic, 329
Anæmia, 113
cerebral, 306
essential, 115
idiopathic, 115
progressive pernicious, 115
Aneurism, thoracic, 302
Angina, 128
pectoris, 293
Aphasia, 315
agraphic, 316
amnesic, 315
ataxic, 315
Apoplexy, 311
spinal, 335
Appendicitis, 163
Arthritis deformans, 105
rheumatoid, 105
Asthma, 235
cardiac, 235
renal, 235
Ataxia, locomotor, 341
Atrophy, acute yellow, 183
progressive muscular, 338
Auscultation, 216

BACILLUS, malaria, 47
Bile-ducts, acute catarrh of, 176
Bilious attack, 134
Blood, diseases of the, 113
Brain, abscess of the, 319
tumors of the, 320
Bright's disease, acute, 194
chronic, 196
Bronchial tubes, diseases of the, 229
Bronchitis, 229
acute catarrhal, of the larger tubes, 229
Bronchitis, capillary, 232
chronic, 234
of the smaller tubes, 232
plastic, 231
Bulbar paralysis, 343

CACHEXIA, malarial, 58
Cancer, gastric, 138
Catarrh, acute bronchial, 229
epidemic, contagious, 28
suffocative, 232
Cerebral softening, acute, 317
chronic, 318
syphilis, 321
tumors, 320
Cerebro-spinal fever, 43
Chest, diseases of, 213
Chicken-pox, 80
Chiragra, 106
Cholera, 92
bacillus of Koch, 92
morbus, 149
Chorea, 354
Circulatory system, diseases of the, 273
Colic, 146
from constipation, 147
from mechanical irritation, 147
lead, 147
metallic, 147
of copper, 147
Colitis, 157
Coma, diabetic, 111
Constipation, 148
Consumption, 257
Contagious disease, definition of, 19
Convulsions, 347
Cramp, writer's, 345
Crisis, 18
Croup, 226
false, 226
spasmodic, 226
true, 226

DANCE, St. Vitus's, 354
Death, definition of, 18
mode of, 18
Defervescence, 24
Delirium tremens, 327
Dengue, 84
Diabetes, 109
insipidus, 112
Diagnosis, definition of, 19
differential, definition of, 20
Diarrhœa, acute, 151
chronic, 152
Diathetic diseases, 99
Dickinson, on the tongue before death, 22
Digestive organs, diseases of, 123
Dilatation, cardiac, 279
acute, 279
chronic, 279
Diphtheria, 85
laryngeal, 86
nasal, 86
Disease, Addison's, 119
Basedow's, 294
causes of, 18
Charcot's, 341
contagious, definition of, 19
definition of, 17
endemic, definition of, 19
epidemic, definition of, 19
functional, 17
Graves', 294
Hodgkin's, 118
infectious, definition of, 19
organic, 18
physiognomy of, 21
pulse of, 21
sporadic, definition of, 19
termination of, 18
Diseases, diathetic, 99
exanthematous, 25
general, 25
zymotic, 25
Diuresis, chronic, 112
Duodenitis, 152
Dysentery, acute, 157
catarrhal, 158
chronic, 161
croupous, 158
diphtheritic, 158
gangrenous, 158
ulcerative, 158
Dyspepsia, 144
acid, 145
Dyspepsia, atonic, 145
flatulent, 145
hysterical, 145
nervous, 145

EGYPTIAN sore throat, 85
Ehrenberg, spirochæte of, 42
El vomito, 59
Embolism, 314
cerebral, 314
Emphysema, 237
Endocarditis, 285
acute, 285
chronic, 288
Enteritis, 156
catarrhal, 154
croupous, 154
membranous, 154
Epilepsies, idiopathic, 355
symptomatic, 355
Epilepsy, 355
Eruptions in essential fevers, 26
Erysipelas, 81
ambulans, 82
cerebral, 82
gangrenous, 82
migrans, 82
phlegmonous, 81
simple, 81
Exanthematous diseases, 25
Exhaustion, 324
Exophthalmos, 294

FAUCES, diseases of, 123
Febricula, 27
Februs recurrens, 42
Fever, 25
abdominal typhus, 31
autumnal, 31
bilious relapsing, 42
break-bone, 84
camp, 39
catarrhal, 28
cerebro-spinal, 43
characteristics of, 25
congestive malarial, 55
diet, definition of, 26
enteric, 31
ephemeral, 27
epidemic catarrhal, 28
famine, 42
gastro-intestinal, 31
hemorrhagic malarial, 53
hospital, 39

Fever, infantile remittent, 31
intermittent, 48
double tertian, 48
duplicated quotidian, 48
quartan, 48
quotidian, 48
tertian, 48
jail, 39
Mediterranean, 59
mild yellow, 42
pernicious malarial, 55
relapsing, 42
remittent, 50
quartan, 51
quotidian, 51
tertian, 51
rheumatic, 97
scarlet, 64
seven days', 42
ship, 39
simple continued, 27
slow nervous, 31
splenic, 191
spotted, 43
thermic, 324
treatment, definition of, 26
typhoid, 30
typhus, 39
yellow, 59
Fevers, continued, definition of, 27
eruptive, 63
essential, 26
classification of, 27
periodical, 47
treatment of, 25

GALL-STONES, passage of, 178
Gangrene, pulmonary, 253
Gastralgia, 143
functional, 143
neuralgic, 143
Gastric catarrh, acute, 134
chronic, 135
ulcer, 137
Gastritis, acute, 133
chronic, 135
General diseases, 25
Gin-liver, 181
Glossitis, 125
acute, 125
chronic, 126
Glycosuria, 109
Goitre, exophthalmic, 294
Gonagra, 106
Gout, acute, 106
chronic, 108
of the brain, 107
of the heart, 107
of the stomach, 107
rheumatic, 105

HÆMATEMESIS, 140
Hæmaturia, 207
Hæmoptysis, 241
Heart, aneurism of, 279
diseases of, 276
fatty degeneration of, 282
irritable, 292
physical examination of, 273
valvular disease of, 288
Heartburn, 145
Heatstroke, 324
Hemiplegia, 349
Hemorrhage, cerebral, 311
spinal, 335
Hepatitis, acute, 175
chronic, 176
interstitial, 181
Herpes zoster, 361
Hodgkin's disease, 118
Hydrocephalus, acute, 322
chronic, 323
Hydronephrosis, 206
Hyperæmia, acute hepatic, 174
acute renal, 193
cerebral, 305
chronic hepatic, 175
chronic renal, 193
pulmonary, 239
spinal, 330
splenic, 191
Hyperpyrexia, rheumatic, 100
Hypertrophy, cardiac, 276

ICTERUS, 173
Ilitis, 154
Incubation period, definition of, 18
periods in essential fevers, 26
Infectious disease, definition of, 19
Influenza, 28
Inoculation, 78
Insolation, 324
Inspection, 213
Intestinal obstruction, 166
tract, diseases of, 146

JAUNDICE, 173
catarrhal, 176

KIDNEY, albuminoid, 200
amyloid, 200
cancer of, 210
cirrhosis, 199
cystic degeneration of, 201
fatty, 196
floating, 211
gouty, 199
large white, 196
small granular, 199
tuberculosis of, 209
waxy, 200
Kidneys, diseases of the, 193

LA COQUETTE, 28
La folette, 28
La grippe, 28
Laryngitis, acute, 223
chronic, 225
Larynx, diseases of the, 223
Laveran, corpuscles of, 52
Lepto-meningitis, 307
Leucocythæmia, 117
Leukæmia, 116
Lithæmia, 109
Lithiasis, 109
Liver, acute parenchymatous degeneration of the, 183
albuminoid, 184
cancer of the, 185
cirrhosis of the, 181
diseases of the, 173
fatty, 183
functional diseases of the, 188
hob-nail, 181
hydatid cyst of, 187
sclerosis of, 181
waxy amyloid, 184
Lung, œdema of, 241
tissue, diseases of, 237
tubercular diseases of, 254
Lungs, hemorrhage from the, 241
Lympho-sarcoma, 118
Lysis, 18

MALARIA, definition of, 47
Malarial cachexia, 58
Malignant sore throat, 85
Mania-a-potû, 327
Measles, 69
black, 70
camp, 70
French, 72
German, 72
Measles, malignant, 70
Meningitis, 307
cerebro-spinal, 43
spinal, 331
tubercular, 309
Mensuration, 214
Metastasis, 18
Muguet, 125
Monoplegia, 349
Morbilli, 69
Mouth, diseases of the, 123
Muco-enteritis, 154
Mumps, 89
Myelitis, 334
interstitial, 334
parenchymatous, 334

NEPHRITIS, acute parenchymatous, 194
acute tubal, 194
chronic interstitial, 199
chronic parenchymatous, 196
Nerves, diseases of the, 358
Nervous system, diseases of the, 305
Neuralgia, 358
cerebral, 359
cervico-brachial, 360
cervico-occipital, 361
dorso-intercostal, 361
lumbo-abdominal, 362
of the fifth pair of nerves, 360
Neurasthenia, 352
cerebral, 352
spinal, 353
Neuroses, the, 354
Neuritis, 364
disseminated, 364
multiple, 364

ŒSOPHAGITIS, 130
Œsophagus, diseases of the, 130
organic stricture of the, 131
spasmodic stricture of the, 131
stricture of the, 131
Oïdium albicans, 125
Oligæmia, 114

PACHYMENINGITIS, 332
Pain, varieties of, 20
Palpation, 213
Palpitation, cardiac, 296
Palsy, Bell's, 351
shaking, 343
wasting, 338

Pancreas, cancer of, 190
cysts of, 189
diseases of, 189
Pancreatitis, 189
chronic, 189
Paralysis, 349
agitans, 343
bulbar, 343
facial, 351
functional, 349
glosso-labio-laryngeal, 343
infantile, 336
syphilitic, 352
Paraplegia, 349
Parotitis, 89
Pathological anatomy, definition of, 17
chemistry, definition of, 17
Pathology, definition of, 17
general and special, 17
Percussion, 214
respiratory, 216
Pericarditis, 297
Pericardium, diseases of, 297
Perinephritis, 210
Periodical fevers, 47
Periproctitis, 166
Peritoneum, diseases of, 169
Peritonitis, acute, 169
chronic, 172
Perityphlitis, 163
Pertussis, 91
Pharyngitis, 128
Pharynx, diseases of, 123
Phthisis, 254
fibroid, 265
pulmonary, 264
Physical diagnosis, 213
Plasmodium malariæ, 47
Pleura, diseases of, 266
Pleurisy, acute, 266
chronic, 269
Pleuritis, acute, 266
Pleurodynia, 103
Pneumonia, 244
acute lobar, 244
catarrhal, 250
chronic, 252
croupous, 244
lobular, 250
Pneumonitis, 244
Pneumothorax, 271
Podagra, 106
Poliomyelitis, acute, 336
chronic, 338
Polyneuritis, 364
Polyuria, 112
Principles and practice of medicine, definition of, 17
Proctitis, 165
Prodromic period, definition of, 18
Prognosis, definition of, 20
general, 20
special, 20
Progressive pernicious anæmia, 115
Pseudo-leukæmia, 118
Pulse, normal, 21
of disease, 21
Purpura, 122
hæmorrhagica, 122
simplex, 122
Pyelitis, 205
Pyrosis, 145

QUINSY, 126

RELAPSING fever, 42
Renal calculi, formation of, 203
mulberry, 203
passage of, 202
phosphatic, 203
uric-acid, 203
Rheumatism, acute, 99
acute articular, 99
cerebral, 100
chronic, 104
muscular, 103
Rheumatoid arthritis, 105
Rötheln, 72
Rubella, 72
Rubeola, 69
sine catarrho, 72

SCARLATINA, 63
anginosa, 64
latens, 65
maligna, 64
simplex, 64
Sciatica, 363
Sclerosis, amyotrophic lateral, 340
antero-lateral, 340
cerebro-spinal, 340
disseminated, 341
multiple, 341
posterior, 341
spinal, 340
Scorbutus, 120
Scurvy, 120

Small-pox, 73
black, 75
Sore throat, chronic, 129
clergymen's, 129
Special organs, diseases of, 123
Spinal cord, diseases of, 330
membranes, diseases of, 330
Spirochæte of Ehrenberg, 42
Spleen, acute affections of the, 190
chronic diseases of, 192
diseases of, 190
Splenitis, acute, 191
Sporadic disease, 19
Sprue, 125
St. Anthony's fire, 81
Stomach, dilated, 142
diseases of, 130
Stomatitis, 123
catarrhal, 123
follicular, 124
ulcerative, 124
Submaxillitis, 90
Sunstroke, 324
Symptomatology, general, definition of, 20
Symptoms, definition of, 18
objective, 18
subjective, 18
Synanche maligna, 85
Synocha, 27
Syphilis, cerebral, 321

TEMPERATURE, how ascertained, 23
normal, 23
variations of, 23
of the mouth, 23
prognostic value of, 24
variations compatible with life, 23
Temperatures, characteristic, 24
Thermometer, surface, 24
Thrombosis, 314
cerebral, 314
Thrush, 125
Tic douloureux, 360
Tongue, appearance in disease, 22
in health, 22
Tonsillitis, 126
Tonsillitis, acute, 126
chronic, 128
Torticollis, 103
Treatment, abortive, 20
definition of, 20
expectant, 20
palliative, 20
prophylactic, 20
radical, 20
restorative, 20
Tremor, 344
from abuse of tobacco, 344
tea-drinkers', 345
from metallic poisoning, 345
Tuberculosis, 254
acute, 255
chronic, 257
of kidney, 209
Tumors, acute splenic, 190
Typhlitis, 163
Typhoid, abortive, 35
fever, 30
walking, 35
Typhus fever, 39
icterodes, 59

ULCER, gastric, 137

VACCINATION, 78
how performed, 79
Varicella, 80
Variola, 73
confluens, 75
discreta, 75
maligna, 75
Varioloid, 78
Vessels, diseases of, 302

WATERBRASH, 145
Whooping-cough, 91
Writer's cramp, 345
Wryneck, 103

YELLOW fever, 59
Yellow jack, 59

ZYMOTIC diseases, 25

AN APPENDIX

CONTAINING SOME

THREE HUNDRED "ESSENTIAL FORMULÆ,"

SELECTED FROM THE

WRITINGS AND PERSONAL PRACTICE OF THE MOST EMINENT AUTHORITIES OF THE MEDICAL PROFESSION.

COLLECTED AND ARRANGED BY

WILLIAM M. POWELL, M.D.,

AUTHOR OF "ESSENTIALS OF DISEASES OF CHILDREN," ETC

Entered according to Act of Congress, in the year 1891, by

W. B. SAUNDERS,

In the Office of the Librarian of Congress, at Washington.

ESSENTIAL FORMULÆ.

ALBUMINOID KIDNEY.

℞ Ammon. chlor., ... ʒiij.
Aq. menthæ pip., ... fʒiij.—M.

Sig.: fʒj in water three times a day.

ALBUMINOID LIVER.

℞ Syr. ferri iodid., ... fʒij.—M.

Sig.: Ten drops in water three times a day. HUGHES.

ALCOHOLISM.

℞ Tr. nucis vomicæ, ... ♏lxxx.
Tr. gentian co.,
Tr. columbæ co., ... āā fʒij.—M.

Sig.: fʒij before each meal, in water. LOOMIS.

℞ Zinci ox., ... gr. xxiv.—M.

Div. in pil. No. xii.

Sig.: One pill three times a day. MORRIS.

℞ Spt. ammon. aromat., ... fʒij.
Tr. camphoræ, ... fʒiss.
Tr. hyoscyamii, ... fʒiiss.
Spts. lavendulæ co., . . q. s. ad fʒij.—M.

Sig.: fʒj every hour or two until relieved. AITKEN.

ANÆMIA.

℞ Liq. potass. arsenitis, ... fʒj.
Vini ferri amar., ... fʒvj.—M.

Sig.: Tablespoonful three times a day, after meals.

F. P. HENRY.

ANÆMIA (Continued).

℞ Tr. ferri chlor., ... fʒiv.
Acid phosphor. dil., ... fʒvj.
Spts. limonis, ... fʒij.
Syr. simp., ... q. s. ad fʒvj.—M.

Sig.: Dessertspoonful, well diluted, after meals. GOODELL.

℞ Quiniæ sulph., ... gr. xx.
Ferri sulph. exsiccat., ... gr. xl.
Strychniæ sulph., ... gr. ss.—M.

Et div in pil. No. xx.

Sig.: One pill three times a day. BARTHOLOW.

ANEURISM.

℞ Potass. iodid., ... ℥ss.
Syr. simp., ... fʒj.
Aq. menthæ pip., ... ad fʒiij.—M.

Sig.: A teaspoonful three times daily, gradually increased to double the quantity. BALFOUR.

℞ Antipyrin, ... ʒiss.
Syr. tolu, ... fʒiss.
Aquæ, ... ad fʒiij.—M.

Sig.: Tablespoonful from one to four hours, to relieve pain. GERMAIN SEE.

ANGINA PECTORIS.

℞ Sol. nitro-glycerin (1 per cent.), . . fʒss.—M.

Sig.: One to two drops internally. (*When pallor of face exists.*) PEPPER.

℞ Methylal, ... fʒix.
Amyl nitrite, ... fʒj.—M.

Sig.: Drop thirty or forty drops on handkerchief and inhale. RICHARDSON.

APOPLEXY.

℞ Ol. tigli, ... gtt. j.
Glycerinæ, ... ♏xij.—M.

Sig.: Place on tongue.

APOPLEXY (Continued).

℞ Tr. veratri viridis, ... f℥ss.—M.

Sig.: Three to five drops every three or four hours.

ASTHMA.

℞ Tr. sanguinariæ,
Tr. lobeliæ,
Ammon. iodid., ... āā ʒj.
Syr. tolu., ... fʒvj —M.

Sig.: ʒj every two to four hours. BARTHOLOW.

℞ Ammon. brom., ... ℈viij.
Ammon. chlor., ... ʒiss.
Tr. lobeliæ, ... fʒiij.
Spt. æther comp., ... f℥j.
Syr. acaciæ, ... ad f℥iv.—M.

Sig.: ʒij in water every hour or two during paroxysms. PEPPER.

℞ Potass. brom., ... ℥ss.
Ex. grindeliæ rob. fl.,
Syr. ipecac., ... āā f℥j.
Aquæ, ... f℥ij.—M.

Sig.: Teaspoonful every four hours. ROCHESTER.

BRIGHT'S DISEASE.

℞ Auri et sodii chlor., ... gr. iij.
Hydrarg. chlor. corr., ... gr. v.
Ex. gentian, ... q. s.—M.

Ft. pil. No. lx.

Sig.: One pill morning and evening. BARTHOLOW.

℞ Ferri sulphat., ... ℈j.
Ex. nucis vom., ... gr. x.–℈j.
Pil. galbani co., ... ℈ij–iij.—M.

Ft. pil. No. xx.

Sig.: A pill two or three times a day. (*When dyspeptic symptoms are present.*) GOODFELLOW.

BRIGHT'S DISEASE (Continued).

℞ Ol. erigeronitis, ... f℥ss.

Sig.: Five drops on a lump of sugar every three or four hours. (*In chronic forms.*) BARTHOLOW.

℞ Potass. acetat., ... gr. x–xx.
Infus. digitalis, ... f℥ij.
Infus. juniperi, ... f℥ij.—M.

Sig.: Take at one dose every two or three hours.

℞ Mist. ferri et ammon. acetat. (U. S. P.), f℥vj.

Sig.: ℥j–℥ij well diluted three times a day. BASHAM.

℞ Ferri sulph., ... gr. xv.
Magnes. sulph., ... ℥ij.
Potass. bicarb., ... ℥iij.
Infus. buchu, ... f℥viij.– M.

Sig.: f℥ss once or twice daily in water. (*When constipation exists.*) FOTHERGILL.

℞ Pulv. jalapæ comp., ... ℥ss–℥j.

Sig.: Take before breakfast.

BRONCHITIS.

℞ Tr. sanguinariæ, ... f℥j.
Tr. lobeliæ, ... f℥j.
Vini ipecac., ... f℥ij.
Syr. tolu., ... f℥ss.—M.

Sig.: Teaspoonful every three hours. BARTHOLOW.

℞ Vini ipecac., ... f℥ij.
Vini antimonialis, ... f℥j.
Vini xerici, ... ℥iij.—M.

Sig.. Three drops every hour to a child six months old. DESSAU.

℞ Ammon. carb., ... ℈ij.
Spt. chloroform., ... f℥ss.
Infus. senegæ, ... f℥viij.—M.

Sig.: f℥ss every four to six hours. FOTHERGILL.

BRONCHITIS (Continued).

℞ Ammon. carb., ... gr. xxiv.
Syr. tolu., ... fʒvj.
Spt. vini gal., ... fʒiij.
Syr. senegæ, ... fʒiiiss.
Syr. acaciæ, ... q. s. ad fʒiij.—M.

Sig.: Teaspoonful every two hours. (*In capillary form.*)
GOODHART and STARR.

℞ Acid. hydrocyan. dil., ... ♏xvj.
Syr. prun. virg.,
Aq. camphoræ, ... āā fʒj.—M.

Sig.: fʒj every two or three hours. HARTSHORNE.

CANCER, GASTRIC.

℞ Bismuth. subnit., ... ʒij.
Acid. hydrocyanic. dil., . . . fʒss.
Mucil. acaciæ,
Aq. menthæ pip., ... āā fʒij.—M.

Sig.: Tablespoonful three times a day in milk.
BARTHOLOW.

℞ Bismuth. subnit., ... ʒij.
Morph. sulph., ... gr. j.—M.

Et div. in chart. No. vi.

Sig.: One powder three times a day in milk. BARTHOLOW.

CANCER OF LIVER.

℞ Liq. potass. arsenitis, ... fʒj.

Sig.: Three to five drops in water three times a day.
Relieve pain by morphine. MORRIS.

CHOLERA.

℞ Strychniæ sulph., ... gr. ¼.
Acid. sulphuric. dil., ... fʒss.
Morphiæ sulph., ... gr. ij.
Aq. camphoræ, . . . q. s. ad fʒiv.—M.

Sig.: Teaspoonful every hour or two, well diluted.
BARTHOLOW.

CHOLERA (Continued).

℞ Plumbi acetat., ... gr. iv.
Ex. opii, ... gr. j.
Ol. theobrom., ... q. s.

Ft. suppos. No. i.

Sig.: Repeat in one hour if the diarrhœa continues.

MORRIS.

CHOLERA INFANTUM.

℞ Acid. sulphuric. aromat., . . . ♏xxiv.
Liq. morphiæ sulphat., . . . fʒj.
Elix. curacoæ, ... fʒij.
Aquæ, ... q. s. ad fʒiij.—M.

Sig.: Teaspoonful every three hours for a child one year old.

GOODHART and STARR.

CHOLERA MORBUS.

℞ Tr. opii deod., ... fʒij.
Acid. sulphuric. aromat., . . . fʒiij.—M.

Sig.: Twenty drops every hour or two in ice water.

BARTHOLOW.

℞ Acid. nitrosi, ... fʒj.
Tr. opii, ... gtt. xl.
Aq. camphoræ, ... fʒviij.—M.

Sig.: One-fourth to be taken every three or four hours.

HOPE.

CHOREA.

℞ Lobelinæ hydrobrom., ... gr. j.
Aquæ, ... fʒv.—M.

Sig.: Three to fifteen minims hypodermically. BARTHOLOW.

℞ Eserinæ sulphat., ... gr. j.
Aquæ destillat., ... fʒvj.—M.

Sig.: Six minims hypodermically twice daily with tonics.

RIESS.

℞ Liq. potass. arsenitis., ... fʒss.

Sig.: One to five drops three times a day gradually increased.

WOOD.

CHOREA (Continued).

℞ Ferri citrat., ... ʒij.
Syr. simp., ... fʒiv.
Aq. aurant. flor., ... fʒiss.—M.
Sig.: fʒj before or after meals. (*When anæmic.*)

HARTSHORNE.

℞ Ex. cimicifugæ fl., ... fʒij.
Sig.: Half teaspoonful increased to a teaspoonful three times a day. (Six to ten years old.)

JESSE YOUNG.

℞ Zinci valerianat.,
Ex. hyoscyam.,
Bismuth. subnit., ... āā gr. xv.—M.
Et div. in pil. No. xxv.
Sig.: Three to six pills daily.

DESCROIZILLES.

COLIC.

℞ Spt. chloroform.,
Tr. cardamom. co., . . . āā ʒij.—M.
Sig.: Teaspoonful every half hour until relieved.

BARTHOLOW.

℞ Tr. opii deod., ... gtt. xij.
Magnesii calcinat., ... gr. xij.-xxiv.
Sacch. alb., ... ʒj.
Aq. anisi, ... fʒiss.—M.
Sig.: Shake well. One teaspoonful for a child of one year.

J. L. SMITH.

CONSTIPATION.

℞ Aloes purificat., ... gr. xx.
Ex. belladonnæ, ... gr. iv.
Ex. nucis vomicæ, ... gr. v.
Oleo resinæ capsici, ... gr. iv.—M.
Et ft. pil. No. xx.
Sig.: One pill at bedtime.

WAUGH.

CONSTIPATION (Continued).

℞ Ex. stillingiæ fl., ... fʒv.
Tr. belladonnæ,
Tr. nucis vomicæ,
Tr. physostigmat., ... āā fʒj.—M.

Sig.: Twenty drops in water three times a day.

BARTHOLOW.

℞ Ex. belladonnæ, ... gr. ¼.
Pil. aloes et myrrh., ... gr. ix.
Ol. cari, ... gtt. ij.—M.

Et ft. pil. No. vi.

Sig.: One pill at bedtime for a child of six years.

GOODHART and STARR.

℞ Ex. cascaræ sagrad. fl., ... fℨj.

Sig.: Three drops three times a day, to be increased, if necessary, for a child of five years.

℞ Sodii bicarb., ... ʒj.
Tr. nucis vomicæ, ... ♏vj.
Tr. card. comp.,
Syr. simp., ... āā fʒij.
Aq. chloroform (Br. P.) . . . . fℨss.
Aquæ, ... fℨij.—M.

Sig.: Teaspoonful every six hours for infant.

EUSTACE SMITH.

℞ Mannæ opt.,
Magnesii carb., ... āā ʒj.
Ex. sennæ fl., ... fʒiij.
Syr. zingiber., ... fℨj.
Aquæ, ... q. s. ad fℨiij.—M.

Sig.: One or two teaspoonfuls three times a day for a child of two years.

GOODHART and STARR.

℞ Tr. aloes et myrrh., ... fℨj.

Sig.: One to three drops in sweetened water three times a day, according to age of child.

CONSTIPATION (Continued).

℞ Mannæ opt., ... ℥j.
Syr. simp., ... f℥ss.
Aq. cinnam., ... q. s. ad f℥j.—M.

Sig.: Teaspoonful three times a day for an infant. STARR.

CONVULSIONS.

℞ Moschi, ... gr. iij.
Camphoræ, ... gr. xv.
Chloral hydrat., ... gr. viiss.
Vitelli ovi . . . . . No. j.
Aq. destillat., ... f℥iv.—M.

Sig.: Wash out the rectum with a simple enema and then use above as an injection. J. SIMON.

℞ Mist. asafœtidæ, ... f℥ij.

Sig.: Tablespoonful per rectum. WARING.

℞ Moschi, ... gr. xij.
Sacchari, ... ʒij.
Spts. ammon., ... ♏xxx.
Infus. lini co., ... f℥iv.—M.

Sig.: An injection for infantile convulsions. ELLIS.

℞ Ætheris fort., ... ℥iv.

Sig.: To be used as an inhalation until the paroxysm is broken. J. L. SMITH.

℞ Chloral hydrat., ... gr. xv.–xxx.
Syr. acaciæ, ... f℥j.
Aquæ, ... ad f℥iv.—M.

Sig.: Inject a tablespoonful into the rectum and repeat in fifteen or twenty minutes, if required. WIDERHOFER.

CROUP (TRUE).

℞ Acid. lactic, ... ℥iiiss.
Aquæ, ... f℥x.—M.

Sig.: Use with spray or mop. MACKENZIE.

CROUP (TRUE) (Continued).

℞ Hydrarg. chlor. mit., ... gr. ij.
Sodii bicarb., ... gr. xxiv.
Pulv. ipecac., ... gr. j.
Pulv. pepsinæ, ... gr. xxiv.—M.

Et ft. chart. No. xii.

Sig.: One powder every two hours. STARR.

℞ Tr. ferri chlor., ... f℥j.–iss.
Potass. chlorat., ... ℥j.
Glycerinæ, ... f℥j.
Aq. cinnam., ... ad f℥iv.—M.

Sig.: Teaspoonful every two hours for a child of four years. MEIGS and PEPPER.

DELIRIUM TREMENS.

℞ Potass. brom., ... ℥j.

Div. in chart. No. viii.

Sig.: One powder in half tumblerful of cold water every four to six hours. BARTHOLOW.

℞ Ex. cannabis indicæ, ... gr. vj.–xij.

Div. in pil. No. xii.

Sig.: One pill every two or three hours till sleep is procured. PHILLIPS.

℞ Sodii brom., ... gr. xv.
Chloral hydrat., ... gr. x.
Syr. aurant. cort.,
Aquæ, ... āā q. s. ad f℥j.—M.

Sig.: As required. DA COSTA.

℞ Liq. morph. sulph. (U. S. P.),
Ex. valerian. fl., ... āā f℥j.—M.

Sig.: One or two teaspoonfuls as required. HARTSHORNE.

DENGUE.

℞ Tr. aconiti rad., ... ♏xxx.
Syr. limonis, ... f℥ss.
Liq. ammon. acetat., . . q. s. ad f℥iij.—M.

Sig.: Dessertspoonful every three hours. THOMAS.

DENGUE (Continued).

℞ Ex. nucis vomicæ, ... gr. iv.
Quiniæ sulphat., ... ʒss.—M.

Et ft. pil. No. xvi.
Sig.: One pill three times a day. DA COSTA.

DIABETES.

℞ Codeinæ, ... gr. viij.
Glycerinæ,
Aquæ, ... āā fʒj.—M.

Sig.: Half teaspoonful three times a day gradually increased to two teaspoonfuls. PAVY.

℞ Sodii salicylat., ... ʒiij.
Liq. potass. arsenitis, ... fʒj.
Glycerinæ, ... fʒj.
Aq. cinnam., . . . q. s. ad fʒiij.—M.

Sig.: Dessertspoonful three times a day. J. C. WILSON.

℞ Tr. opii, ... fʒj.
Tr. ferri chlor., ... ʒix.—M.

Sig.: Twenty drops well diluted three times daily.
WELLER.

℞ Ex. ergotæ fl., ... fʒij.

Sig.: Teaspoonful three times a day, increased to two teaspoonfuls. DA COSTA.

℞ Pulv. opii, ... gr. iv.
Acid. gallici, ... ʒij.—M.

Et div. in chart. No. xii.
Sig.: One three or four times daily. H. C. WOOD.

℞ Sodii salicylat., ... ʒiv.
Glycerinæ, ... fʒij.
Aquæ, ... q. s. ad fʒiij.—M.

Sig.: Two teaspoonfuls three times daily. DA COSTA.

DIARRHŒA IN ADULTS.

℞ Aq. camphoræ, ... f℥iij.
Spt. lavend. co., ... f℥j.
Sacch. alb., ... ℥j.—M.

Sig.: Tablespoonful every two hours. PARRISH.

℞ Cretæ præp., ... ℥ij.
Tr. catechu, ... f℥ss.
Tr. opii, ... ♏lxxx.
Aq. cinnam., ... ad f℥viij.—M.

Sig.: Two tablespoonfuls after each stool. FOTHERGILL.

℞ Ex. ergotæ aq., ... ℈j.
Ex. nucis vomicæ, ... gr. v.
Ex. opii, ... gr. x.—M.

Et ft. pil. No. xx.

Sig.: One pill every four to six hours. DA COSTA.

DIARRHŒA IN CHILDREN.

℞ Bismuth. subcarb., ... ℥ss–℥iss.
Spt. myristicæ, ... ♏xx.
Spt. vini gal., ... f℥ij.
Syr. acaciæ, ... f℥iss.
Aq. cinnam., . . . q. s. ad f℥iij.—M.

Sig.: (Shake well.) Teaspoonful every two hours.
W. H. BENNETT.

℞ Magnesii sulphat., ... ℥j.
Tr. opii deod., ... gtt. xij.
Syr. simp., ... f℥ss.
Aq. cinnam., ... q. s. ad f℥iss.—M.

Sig.: Teaspoonful every two hours for a child of one or two years. MEIGS and PEPPER.

℞ Argenti nitrat., ... gr. j.
Syr. acaciæ, ... f℥ij.
Aq. cinnam., . . . q. s. ad f℥iij.—M.

Sig.: Teaspoonful every two hours for a child of two years.
STARR.

DIARRHŒA IN CHILDREN (Continued).

℞ Tr. krameriæ,
Tr. opii camph., ... āā f℥ij.
Mist. cretæ, ... q. s. ad f℥ij.—M.

Sig.: Teaspoonful every two hours for a child of two years.

DILATED STOMACH.

℞ Carbo lig., ... gr. xij–xxiv.
Sodii bicarb., ... ℥ss.
Bismuth subnit., ... ℥j.—M.
Et div. in capsul. No. xii.

Sig.: One three or four times daily at meal time.

DIPHTHERIA.

℞ Trypsin (Fairchild's), ... ℥j.
Sodii bicarb., ... gr. xx.
Aquæ, ... q. s. ad f℥ij.—M.

Sig.: Apply with atomizer every hour or two as necessary.

KEATING.

℞ Ol. eucalypti, ... f℥ij.
Ol. terebinthinæ, ... f℥viij.—M.

Sig.: Place in shallow vessel and keep boiling over the stove.

J. LEWIS SMITH.

℞ Acid. boric.,
Sodii borat., ... āā ℥ss.
Sodii chlor., ... gr. xx.
Aquæ, ... Oss.—M.

Sig.: Inject teaspoonful, warm, in each nostril every two hours. (*Nasal form.*)

STARR.

℞ Quiniæ sulphat., ... gr. xij.
Potass. chlorat., ... gr. xlviij.
Tr. ferri chlor., ... f℥j.
Syr. zingiber., ... f℥j.
Aquæ, ... q. s. ad f℥iij.—M.

Sig.: Teaspoonful in water every two hours for a child of six to ten years.

GOODHART and STARR.

DIPHTHERIA (Continued.)

℞ Hydrarg. chlor. corros., . . . gr. j.
Spt. vini rect., ... fʒij.
Elix. bismuth et pepsin, . . ad fʒiv.—M.

Sig.: Teaspoonful every two hours for a child of six years.
J. Lewis Smith.

℞ Tr. ferri chlor., ... fʒ–ʒiij.
Glycerinæ, ... q. s. ad fʒj.—M.

Sig.: Paint tonsils every four hours. Rex.

DYSENTERY.

℞ Cupri sulphat., ... gr. ss.
Magnesii sulphat., ... fʒj.
Acid. sulphuric. dil., ... fʒj.
Aquæ, ... fʒiv.—M.

Sig.: Tablespoonful every four hours. (*In acute form.*)
Bartholow.

℞ Hydrarg. chlor. mit., ... gr. ij.
Pulv. opii., ... gr. iv.
Pulv. ipecac., ... gr. viij.—M.
Et div. in chart No. viii.

Sig.: One powder every two hours. Hazard.

℞ Liq. ferri pernitrat.,
Acid. nitric dil., . . . āā fʒss.
Syr. simp., ... fʒj.
Aq. cinnam., . . . q. s. ad fʒiij.—M.

Sig.: Teaspoonful every three hours for a child. Ellis.

℞ Pulv. ipecac. co., ... gr. vj.
Bismuth. subcarb., ... ʒj.
Pulv. aromat., ... gr. vj.—M.
Et ft. in chart. No. xij.

Sig.: One powder every three hours for a child of three years.
Starr

DYSPEPSIA.

℞ Bismuth. subcarb., ... ʒiij.
Morph. sulph., ... gr. j.
Pulv. aromat., ... ʒj.—M.
Et div. in chart. No. xii.

Sig.: A powder in milk before each meal. BARTHOLOW.

℞ Bismuth. subnit., ... ℈iv.
Mucil. acaciæ, ... fʒj.
Sodii bicarb., ... ℈iv.
Infus. calumbæ, ... fʒviij.—M.

Sig.: Two tablespoonfuls before each meal. FOTHERGILL.

℞ Pepsinæ (Fairchild's) . . . . gr. xxxvj.
Carbo lig., ... gr. xxiv.
Sodii bicarb., ... ʒj.—M.
Et div. in cap. No. xii.

Sig.: One after each meal. STARR.

℞ Tr. capsici, ... ♏xvj.
Tr. nucis vomicæ, ... fʒij.
Tr. gentian. comp., . . . ad fʒij.—M.

Sig.: A teaspoonful in water three times a day. DA COSTA.

℞ Ex. cascaræ sagrad. fl.,
Ex. berberis aquifol., . . . āā fʒj.
Syr. simp., ... fʒij.—M.

Sig.: Teaspoonful three times a day. BUNDY.

EMPHYSEMA.

℞ Ex. convallariæ fl., ... ♏iv.
Syr. tolu, ... ♏viij.
Aquæ, ... q. s. ad fʒj.—M.

Sig.: Take every three hours. ROBINSON.

℞ Tr. opii, ... fʒj.
Ætheris, ... ʒij.—M.

Sig.: Sixty drops every twenty minutes well diluted until relieved. CLYMER.

ENDOCARDITIS.

℞ Tr. aconiti rad., ... fʒss.

Sig.: One drop every hour or two. RINGER.

℞ Lini farinæ,
Aq. bullientis, ... ad q. s.—M.
Ft. cataplasma.

Sig.: Apply over heart as hot as can be borne and renew frequently. DA COSTA.

ENTERITIS.

℞ Liq. potass. arsenitis, ... gtt. l.
Tr. opii, ... gtt. cxx.
Aquæ, ... ad fʒiij.—M.

Sig.: Teaspoonful before meals three times a day.
BARTHOLOW.

℞ Ol. ricini, ... fʒj.
Pulv. acaciæ,
Sacch. alb., ... āā ℈iss.
Tr. opii, ... ♏iij.
Aq. cinnam., ... ʒxj.—M.

Sig.: Teaspoonful every four hours for a child of one year.
TANNER.

℞ Pulv. opii, ... gr. v.
Bismuth subnit., ... ʒij.—M.

Et div. in chart. No. xx.

Sig.: One powder every two to four hours for a child of five years. J. L. SMITH.

℞ Naphthalin, ... gr. xij–ʒj.
Sacch. lact., ... gr. xij–ʒss.—M.

Et ft. chart. No. xii.

Sig.: One powder every three hours. STARR.

EPILEPSY.

℞ Lobelinæ hydrobrom., ... gr. ½–j.
Aq. destill., ... f℥iiss.—M.

Sig.: Teaspoonful three or four times a day. BARTHOLOW.

℞ Ex. conii fl., ... f℥ij.

Sig.: Fifteen to sixty minims not over three times a day.
SPITZKA.

℞ Nickel brom., ... gr. xvj.
Aq. destill., ... f℥ij.—M.

Sig.: Teaspoonful several times daily. DA COSTA.

℞ Ferri brom., ... gr. iv.
Potass. brom., ... f℥j.
Syr. simp., ... f℥vj.
Aquæ, ... ad f℥viij.—M.

Sig.: Tablespoonful twice daily. (*In anæmic patients.*)
BARTHOLOW.

℞ Potass. brom.,
Ammon. brom., . . . āā ℥j.
Ex. ergotæ fl., ... f℥ss.
Aquæ, ... q. s. ad f℥ij.—M.

Sig.: Teaspoonful three times a day well diluted. (*When maniacal excitement follows the attack, or cerebral congestion or hemorrhage is feared.*) CHAS. R. SMITH.

℞ Potass. brom.,
Sodii brom.,
Ammon. brom., ... āā ʒiij.
Potass. iodid.,
Ammon. iodid., ... āā ʒiss.
Ammon. carbonat., ... ʒj.
Tr. calumbæ, ... f℥iss.
Aquæ, ... q. s. ad ℥viij.—M.

Sig.: Teaspoonful and a half before each meal and three teaspoonfuls at bedtime. BROWN-SÉQUARD.

ERYSIPELAS.

℞ Antifebrin, ... ʒj.

Div. in capsulas No. xv.

Sig.: Two capsules as required to reduce temperature.

OSLER.

℞ Tr. ferri chlor.,
Syr. simp., ... āā fʒj.
Aquæ, ... q. s. ad fʒiij.—M.

Sig.: Teaspoonful every two or three hours well diluted.

CHARITY HOSPITAL, N. Y.

℞ Ferri sulphat., ... ʒj.
Aquæ, ... Oj.—M.

Sig.: Apply by compresses, and renew every two or three hours. VELPEAU.

℞ Cretæ præparat.,
Adipis, ... āā ʒj.
Acid. carbol., ... fʒj.—M.

Sig.: Apply to part and cover with lint. DUCKWORTH.

FEVER, CATARRHAL.

℞ Antifebrin., ... ʒj.
Spt. vini gal., ... fʒss.
Elix. simp., ... q. s. ad fʒij.—M.

Sig.: Teaspoonful every four hours. HEINZELMANN.

FEVER, INTERMITTENT. (See RELAPSING FEVER.)

FEVER, RELAPSING.

℞ Quiniæ sulphat., ... ℈iv.
Acid. sulphuric. dil., ... q. s. ut ft. sol.
Spt. æther. nitro., ... fʒss.
Syr. tolu,
Aquæ, ... āā q. s. ad fʒij.—M.

Sig.: Teaspoonful three or four times daily. DA COSTA.

FEVER, REMITTENT.

℞ Acid. carbol., ... f℥j.
Tr. iodinii comp., ... f℥iij.—M.

Sig.: Four drops every four hours well diluted.

BARTHOLOW.

FEVER, SCARLET.

℞ Tr. ferri chlor., ... f℥j.
Potass. chlorat., ... gr. xlviij.
Glycerinæ, ... f℥j.
Aquæ, ... q. s. ad f℥iij.—M.

Sig.: Teaspoonful every two hours for a child of four years.

MORRIS.

℞ Acid. boracic., ... ℥ss.
Potass. chlor., ... ℥ij.
Tr. ferri chlor., ... f℥ij.
Glycerinæ,
Syr. simp., ... āā f℥j.
Aquæ, ... f℥ij.—M.

Sig.: Teaspoonful every two hours for a child of five years.

J. LEWIS SMITH.

℞ Infus. digitalis, ... f℥iv.

Sig.: One-half to one teaspoonful every two or three hours.

BARTHOLOW.

℞ Acid. carbol., ... ♏xx.
Vaseline, ... ℥j.—M.

Sig.: Apply to body night and morning. STARR.

℞ Tr. digitalis, ... f℥ss.
Liq. ammon. acetat., ... f℥iss.
Spt. æth. nit., ... f℥ij.
Syr. tolu, ... f℥ss.
Aq. cari, ... q. s. ad f℥iij.

Sig.: Teaspoonful every two hours for a child of six or eight years.

GOODHART and STARR.

FEVER, SCARLET (Continued).

℞ Ol. menthæ pip., ... ♏xv.
Ol. olivæ, ... fʒiij.—M.

Sig.: Apply to body night and morning. STARR.

FEVER, SIMPLE CONTINUED.

℞ Liq. ammon. acetat., ... fʒiiiss.
Spt. æther. nitros., . . q. s. ad fʒiv.—M.

Sig.: Teaspoonful to tablespoonful every two hours.
HARTSHORNE.

FEVER, SPOTTED. (See also MENINGITIS.)

℞ Morphiæ sulphat., ... gr. ss.
Acid. sulphur. aromat. . . . . fʒj.
Elix. cinchonæ, . . . q. s. ad fʒvj.—M.

Sig.: Teaspoonful every two hours for a child of twelve years.
MEIGS and PEPPER.

℞ Acid. hydrocyanic. dil., . . . ♏xxx.
Sodii bicarb., ... ʒj.
Syr. simp., ... fʒss.
Aquæ, ... q. s. ad fʒiij.—M.

Sig.: Teaspoonful every three or four hours for vomiting.
DELAFIELD.

FEVER, TYPHOID.

℞ Antipyrin, ... ℈viij.
Syr. simp., ... fʒj.
Aquæ, ... q. s. ad fʒij.

Sig.: Two or three teaspoonfuls at first, and one teaspoonful hourly until temperature is reduced. MINOT.

℞ Bismuth subnit., ... ʒiij.
Spt. vini gal., ... fʒvj.
Spt. myristicæ, ... fʒss.
Syr. acaciæ, ... fʒj.
Aq. cinnam., . . . q. s. ad fʒiij.—M.

Sig.: From one to two teaspoonfuls every three or four hours.
W. H. BENNETT.

FEVER, TYPHOID (Continued).

℞ Acid. muriat. dil., ... f℥j.
Syr. rubi idæi, ... f℥vij.
Aquæ, ... f℥iij.—M.

Sig.: f℥ij every two or three hours. GERHARD.

FEVER, TYPHUS.

℞ Quiniæ sulphat., ... ℈iv.
Acid. sulphuric. dil., ... f℥ss.
Syr. simp., ... f℥ss.
Aquæ, ... q. s. ad f℥ij.—M.

Sig.: Teaspoonful every two hours until temperature is lowered. GOLDEN.

℞ Tr. belladonnæ, ... f℥ss.
Tr. aconiti rad., ... f℥ss.—M.

Sig.: Ten drops every two hours. (*For dry tongue and rapid pulse.*) HARLEY.

FEVER, YELLOW.

℞ Pilocarpinæ muriat., ... gr. iij.
Aq. destillat., ... f℥ij.—M.

Sig.: ♏x hypodermically. HEBER SMITH.

℞ Hydrarg. chlor. mit.,
Pulv. jalapæ, ... āā gr. x.—M.

Et ft. pulv. No. i.

Sig.: Use at the onset of the disease. RUSH.

GALL STONES (PASSAGE OF).

℞ Chloroform. purif., ... f℥ij.
Ol. cinnam., ... gtt. viij.
Spt. camphoræ,
Tr. opii deod., ... āā f℥iss.
Spt. vini, ... ℥iij.—M.

Sig.: Five to thirty drops in sweetened water every hour or two. HARTSHORNE.

GALL STONES (PASSAGE OF) (Continued).

℞ Morphiæ sulphat., ... gr. vj.
Atropiæ sulphat., ... gr. $\frac{1}{5}$.
Aquæ, ... f℥ss.—M.

Sig.: Ten minims hypodermically during paroxysm, and repeated if necessary. BARTHOLOW.

℞ Ætheris, ... f℥iij.
Ol. terebinth., ... f℥ij.—M.

Sig.: Ten or twelve drops twice daily. DURANDE.

GASTRALGIA.

℞ Ex. cocæ fl., ... f℥j.
Syr. aurantii flor., ... f℥v.
Aquæ, ... ad f℥ij.—M.

Sig.: Teaspoonful every hour until relieved. D'ARDENNE.

℞ Tr. conii, ... f℥j.
Tr. valerianæ,
Tr. opii camph.,
Aquæ lauro-cerasi, . . . āā f℥ij.—M.

Sig.: Seven drops in milk when the pain appears. MONIN.

℞ Tr. aconite (Fleming), ... f℥j.
Sodii carbonat., ... ℥iss.
Magnesii sulph., ... ℥iss.
Aquæ, ... f℥vj.—M.

Sig.: Tablespoonful when pain is urgent. FLEMING.

GASTRIC CATARRH, ACUTE IN CHILD.

℞ Liq. calcis,
Aquæ cinnam., ... āā f℥j.—M.

Sig.: One or two teaspoonfuls every fifteen or twenty minutes as necessary. STARR.

GASTRIC CATARRH.

℞ Acid. carbol., ... gtt. xxiv.
Aq. menthæ pip., ... f℥iij.—M.

Sig.: Teaspoonful every hour or two.

GASTRIC CATARRH (Continued).

℞ Hydrarg. chlor. mit., ... gr. j.
Bismuth subnit., ... gr xxxvj.—M.

Et ft. chart. No. xii.

Sig.: One powder every three hours. GREEN.

GOITRE.

℞ Potass. brom., ... ℥ss.
Div. in chart. No. xii.

Sig.: Powder well diluted three times a day.
JON HUTCHINSON.

℞ Tr. iodine co., ... f℥j.

Sig.: Apply locally with a brush; also give from five to fifteen drops well diluted three times a day. BARTHOLOW.

℞ Ungt. hydrarg. iodid. rubr., . . ℥j.

Sig.: Rub in a piece the size of a pea and expose to heat.
RINGER.

GOUT.

℞ Colchicini, ... gr. j.
Ex. colocynth. co. . ℥ss.
Quiniæ sulphat., ... ℥iij.—M.

Et ft. pil. No. lx.

Sig.: One pill every four hours. BARTHOLOW.

℞ Magnesii sulph., ... ℥ij.
Potass. bicarb., ... gr. xv.
Tr. colchici sem., ... ♏x.
Infus. buchu, ... f℥j.

Ft. haustus.

Sig.: To be taken every four or six hours, followed by a large draught of water, not too cold. FOTHERGILL.

℞ Tr. colchici sem., ... ♏xv.
Magnes. carb., ... gr. vj.
Magnes. sulp., ... ℥ss.
Aq. menthæ pip., ... ad f℥j.—M.

Ft. haustus. UNIVERSITY HOSPITAL.

HÆMATEMESIS.

℞ Liq. ferri subsulphat., ... gtt. xx.
Aquæ, ... f℥ij.—M.

Sig.: Teaspoonful every half or hour in ice water.
BARTHOLOW.

℞ Tr. hamamelis, ... f℥j.

Sig.: Two to four drops every two hours. RINGER.

℞ Plumbi acetat., ... ℥ss.
Hydrarg. chlor. mit., ... gr. v.
Confect. rosæ, ... q. s.—M.

Et ft. pil. No. x.

Sig.: Pill every two to four hours. ELLIS.

HÆMATURIA.

℞ Tr. ferri chlor., ... ♏xxx.
Tr. digitalis, ... ♏xv.
Aq. menthæ pip., ... ℥iss.—M.

Sig.: Take one dose every four hours. AITKEN.

℞ Acid. gallic., ... ℥ss.
Acid. sulphuric. dil., ... f℥j.
Tr. opii deod., ... f℥j.
Infus. digitalis, ... f℥iv.—M.

Sig.: ℥ss every four hours. DRUITT.

℞ Ex. ergot. fl., ... f℥ij.

Sig.: 20 gtt.–℥j every two hours. MORRIS.

℞ Tr. hamamelis, ... ♏xxiv.
Elix. simp.,
Aquæ, ... āā f℥j.—M.

Sig.: f℥j every two or three hours. RINGER.

HÆMOPTYSIS.

℞ Ext. ergotæ fl., ... f℥j.
Ol. gaultheriæ, ... gtt. iv.—M.

Sig.: f℥j every hour at first; then every four to six hours.
RINGER.

HÆMOPTYSIS (Continued).

℞ Plumbi acetat., ... gr. xx.
Pulv. digitalis, ... gr. x.
Pulv. opii, ... gr. v.—M.

Et div. in pil. No. x.

Sig.: One pill every four hours. BARTHOLOW.

HEART DISEASE.

℞ Tr. digitalis, ... fʒij.
Spt. chloroform., ... fʒv.
Infus. buchu, ... ad fʒxij.—M.

Sig.: ʒj in wineglassful of water three times a day. (*In simple cardiac debility.*) FOTHERGILL.

℞ Pulv. digitalis, ... gr. xxx.
Ferri sulph. exsiccat., ... gr. xv.
Pulv. capsici, ... gr. xl.
Pil. aloe et myrrh., ... ʒij.—M.

Et ft. pil. No. lx.

Sig.: One pill night and morning. (*Chronic heart trouble, with constipation.*). FOTHERGILL.

℞ Tr. strophanthi (1–20), ... fʒj.

Sig.: Five to fifteen drops three times daily. (*In fatty heart and valvular disease.*) FRASER.

℞ Ex. ergotæ fl., ... fʒiiiss.
Tr. digitalis, ... fʒss.—M.

Sig.: Teaspoonful three times a day. (*Enlarged heart without valvular lesion.*) BARTHOLOW.

℞ Ferri redacti,
Pulv. digitalis fol.,
Quiniæ sulphat., ... āā ℈j.
Pulv. scillæ, ... gr. x.—M.

Et ft. pill No. xx.

Sig.: One pill three or four times a day. (*In fatty heart, dilatation of cavities, and mitral regurgitation.*) BARTHOLOW.

HEART DISEASE (Continued).

℞ Tr. digitalis, ... f℥ij.

Sig.: 10♏ three times a day. (*In irritable heart with palpitation.*) DA COSTA.

℞ Tr. veratri viridis, ... f℥ss.

Sig.: Five drops three times daily. (*In hypertrophy.*) BARTHOLOW.

℞ Potass. iodid., ... ʒj.
Potass. bicarb., ... ʒiij.
Infus. buchu, ... f℥xij.—M.

Sig.: f℥j three or four times daily. (*In hypertrophy.*) FOTHERGILL.

HEPATIC HYPERÆMIA.

℞ Hydrarg. chlor. mit., . . . gr. iij.
Sodii bicarb., ... gr. xxxvj.—M.

Et div. in chart. No. xii.

Sig.: One every two hours, to be followed by a saline.

HYDROCEPHALUS.

℞ Potass. iodid., ... ʒss–ʒj.
Syr. aurant. cort., ... f℥j.
Aquæ, ... ad f℥iv.—M.

Sig.: ʒj every two hours to an infant of six months. J. LEWIS SMITH.

℞ Potass. iodid., ... gr. xvj.
Iodini, ... gr. iv.
Aquæ, ... f℥j.—M.

Sig.: ʒj every four hours. Used in connection with—

Ungt. hydrarg. biniod., . . . ʒij.
Cerati simp., ... ℥j.—M.

Sig.: Rub into scalp every four hours. CHRISTIE.

℞ Ol. tiglii, ... ♏ij.
Mucil. acaciæ, ... f℥ij.
Aquæ, ... f℥j.—M.

Sig.: Take the fourth part every four hours. DUNGLISON.

JAUNDICE.

℞ Sodii sulphat.,
Potass. et sodii tart., . . . āā ℥j.
Infus. cascarillæ, ... f℥viij.—M.
Sig.: Two tablespoonfuls three times daily. FOTHERGILL.

℞ Acid. nitro-muriat. dil., . . . f℥j.
Sig.: Ten or fifteen drops, well diluted, before meals.
BARTHOLOW.

KIDNEYS, CONGESTION OF.

℞ Infus. digitalis, ... f℥iv.
Sig.: ℥ij–℥iv three or four times a day.

LARYNGITIS (ACUTE).

℞ Tr. aconiti rad., ... f℥ss.
Sig.: One drop every hour, in water. Best results when following a dose of castor oil. When it has existed several days give—

℞ Vini mariani, ... Oj.
Sig.: Wineglassful every three hours, with absolute rest of voice. SAJOUS.

℞ Tr. pulsatillæ, ... f℥j.
Syr. ipecac., ... f℥j.
Liq. potass. citrat., ... f℥v.—M.
Sig.: f℥ss every three hours. GERHARD.

℞ Argenti nitrat., ... gr. lx.
Aquæ, ... f℥j.—M.
Sig.: Apply locally on cotton; then immediately apply the following:—

℞ Cocaine muriat. (10 per cent. sol.), . f℥j.
Sig.: Apply locally to the larynx. (*Chronic form.*) SEILER.

LARYNGITIS (ACUTE) (Continued).

℞ Hydrarg. cyanidi, ... gr. ij.
Sacch. lact., ... gr. xv.
Mucil. acaciæ, ... q. s.—M.

Et div in pil. No. xx.

Sig.: One pill twice daily. (*Syphilitic form.*)

M. MACKENZIE.

LEUCOCYTHÆMIA.

℞ Acid. arseniosi, ... gr. j.
Pil. ferri carbonat.,
Quinidiæ sulph., ... āā ʒj.—M.

Et ft. pil. No. xl.

Sig.: Two pills three times a day. DA COSTA.

℞ Chinoidinæ, ... ℈ij.
Resinæ podophyll., ... gr. iv.
Ferri sulph. exsiccat., . . . ℈j.—M.

Et ft. pil. No. xx.

Sig.: One pill three times a day. BARTHOLOW.

LITHÆMIA. (See GOUT.)

LIVER, FUNCTIONAL DISEASE OF.

℞ Acid. nitro-muriat. dil., . . . fʒij.

Sig.: Ten drops, well diluted, before meals. WOOD.

LIVER, ALBUMINOID.

℞ Ammon. chlor., ... ʒiij.
Syr. limonis, ... fʒiss.
Aquæ, ... q. s. ad fʒiij.—M.

Sig.: Teaspoonful three times a day, gradually increased to two teaspoonfuls. DA COSTA.

LOCOMOTOR ATAXIA.

℞ Argent. nitrat., ... gr. x.
Confect. rosæ, ... ℈j.—M.

Et ft. pil. No. xl.

Sig.: One or two pills three times a day. Cease giving after a few weeks, to prevent argyria. DA COSTA.

LOCOMOTOR ATAXIA (Continued).

℞ Strychniæ sulph., ... gr. iss.
Syr. hypophos., ... f℥xij.—M.

Sig.: f℥j in water three times a day. (*When the system is saturated with silver.*) DA COSTA.

LUMBAGO.

℞ Atrophiæ sulph., ... gr. i½.
Morph. sulphat., ... gr. xvj.
Aq. destillat., ... f℥j.—M.

Sig.: Inject 5 minims into the muscular tissues. DA COSTA.

℞ Potass. iodid., ... ℥ij.
Vini colchici sem., ... f℥j.
Syr. zingiber., ... f℥iss.
Aquæ, ... q. s. ad f℥iv.—M.

Sig.: f℥ij every three hours. GERHARD.

℞ Potass. iodid.,
Potass. carbonat., ... āā ℥j.
Tr. aconiti rad., ... f℥ij.
Aquæ, ... f℥x.—M.

Sig.: Use locally every three hours. (*Mark poison.*) ERICHSEN.

℞ Ex. cimicifugæ fl.,
Syr. acaciæ, ... āā f℥ss.
Aq. amygdalæ amar., ... f℥iij.—M.

Sig.: Teaspoonful every three hours. BARTLETT.

MEASLES.

℞ Liq. ammon. acetat., ... f℥iiiss.
Spt. æther. nitro., . . q. s. ad f℥iv.—M.

Sig.: Teaspoonful every two or three hours. HARTSHORNE.

℞ Pulv. ipecac., ... gr. vj.
Syr. simp., ... f℥ss.
Aquæ, ... f℥vj.—M.

Sig.: f℥ss three times a day. NIEMEYER.

MEASLES (Continued).

℞ Tr. opii camph., ... fʒiss.
Spt. æther. nitro., ... fℨss.
Syr. ipecac., ... fʒj.
Syr. scillæ, ... fℨss.
Liq. potass. citrat., . . q. s. ad fℨiij.—M.

Sig.: Teaspoonful every two hours for a child of four years.

PENROSE.

MENINGITIS.

℞ Sodii brom., ... ʒij.
Chloral hydrat., ... ʒj.
Syr. aurant. cort., ... fℨj.
Aquæ, ... q. s. ad fℨiij.—M.

Sig.: ʒij well diluted every hour until excitement abates.

HERMANN.

℞ Tr. opii deodorat.,
Ex. gelsemii fl., ... āā fʒj.
Syr. limonis, ... fʒij.
Aq. fœniculi, ... fℨiss.—M.

Sig.: Teaspoonful every two hours. BARTHOLOW.

℞ Hydrarg. chlor. mit., ... gr. iij.
Sacch. lact., ... ʒss.—M.

Et ft. chart. No. xii.

Sig.: One powder every two hours. GERHARD.

℞ Morph. sulph., ... gr. ij.
Aquæ, ... fʒj.—M.

Sig.: Five minims hypodermically every three to five hours. (*In cerebro-spinal form.*) LEYDEN.

℞ Hydrarg. chlor. mit.,
Pulv. jalapæ,
Sacch. alb., ... āā ʒj.—M.

Et div. in chart. No. v.

Sig.: A powder every hour until free purgation occurs. (*In cerebro-spinal meningitis.*) KOBERT.

MENINGITIS (Continued).

℞ Tr. aconiti rad., ... ʒij.
Tr. opii deod., ... fʒv.—M.

Sig.: Seven drops in water every two hours during the stage of excitement. (*Cerebro-spinal form.*) BARTHOLOW.

MUMPS. (See PAROTITIS.)

NEURALGIA.

℞ Quiniæ sulphat., ... ʒj.
Morphiæ sulphat.,
Acid. arseniosi, ... āā gr. iss.
Ex. aconiti, ... gr. xv.
Strychniæ sulph., ... gr. j.—M.

Et ft. pil. No. xxx.
Sig.: One pill three times a day. S. D. GROSS.

℞ Thein,
Sodii benzoat., ... āā ʒj.
Sodii chlor., ... gr. x.
Aq. destillat., ... fʒj.—M.

Sig.: Three to twenty drops, as required. MAYS.

℞ Menthol, ... gr. xxiiss.
Cocaini muriat., ... gr. viiss.
Chloral hydrat., ... gr. ivss.
Vaselini, ... ʒiiss.—M.

Sig.: Apply to the painful part and cover with court-plaster. GALEZOWSKI.

℞ Menthol, ... ʒj.
Lini saponis co., ... fʒij.—M.

Sig.: Use locally. WITHERSTINE.

℞ Aconitiæ, ... gr. iss.
Spt. vini rect. . . . . q. s.
Adipis præp., ... ʒij.—M.

Sig.: To be rubbed in three times daily. BROOKES.

NEURALGIA (Continued.)

℞ Chloral. hydrat.,
Pulv. camphoræ, ... āā ʒiv.—M.

Sig.: Apply with camel's-hair brush. GEORGE BIRD.

℞ Ferri carbonat., ... ʒij.
Quiniæ sulphat., ... gr. vj.
Ex. opii, ... gr. ¾.
Syr. simp., ... q. s.—M.

Et ft. pil. No. xvi.

Sig.: Eight pills during the day. JOLLY.

℞ Methyl chlor. pur., ... fℨj.

Sig.: Apply with brush to the painful parts. DEBORE.

℞ Sol. nitro-glycerin (1 per cent.), . . fℨss.

Sig.: One or two drops on the tongue every four to six hours. TRUSSEWITSCH.

* **PARALYSIS.**

℞ Strychniæ sulph., ... gr. ij.
Aquæ, ... ♏c.—M.

Sig.: Two to four minims hypodermically every second day or daily. (*In all forms of paralysis except cerebral and spinal paralysis.*) BARWELL.

℞ Ammon. iodid., ... ʒj.
Ammon. carbonat., ... ʒij.
Liq. ammon. acetat., ... fℨvj.—M.

Sig.: ℨss three times a day. (*To absorb thrombi in incipient hemiplegic paralysis due to endarteritis deformans.*) BARTHOLOW.

℞ Phosphori, ... gr. ij.
Alcoholis absolut., ... ʒxxiij.
Tr. vanillæ, ... fℨss.
Ol. aurant. cort., ... ♏xij.
Alcoholis absolut., . . q. s. ad fℨiij.—M.

Sig.: Twenty to forty minims two or three times a day. (*In cerebral softening and hysterical paralysis.*) HAMMOND.

PARALYSIS (Continued).

℞ Hyoscyam. sulph., ... gr. ss.
Aquæ, ... fʒvj.—M.

Sig.: Five minims hypodermically once daily or by the stomach twice daily. (*In paralysis agitans.*) SÉGUIN.

℞ Strychniæ sulph., ... gr. ij.
Aq. destillat., ... fʒj.

Sig.: One to five minims hypodermically. (*In infantile paralysis, etc.*) BARTHOLOW.

℞ Ex. physostigmatis, ... gr. j.
Ex. gentian, ... ℈j.—M.

Et div. in pil. No. xxx.

Sig.: One pill every two hours. (*In general paralysis of the insane.*) CRICHTON BROWNE.

PAROTITIS (MUMPS).

℞ Hydrarg. cum cretæ, ... gr. iv.
Sacch. lact., ... ℈j.—M.

Div. in chart. No. xii.

Sig.: One powder three times a day. RINGER.

℞ Tr. belladonnæ,
Tr. opii,
Ætheris, ... āā fʒj.
Liniment. saponis, ... fʒiij.—M.

Sig.: Use locally. HAZARD.

℞ Magnesii sulph., ... ʒiv.
Aq. puræ, ... fʒiv.
Antimonii et potass. tart., . . . gr. j.
Spt. æth. nit., ... fʒiij.
Sacch. alb., ... fʒvj —M.

Sig.: Teaspoonful every three hours, after the bowels have been well moved. Flaxseed poultices locally. CONDIE.

PERICARDITIS.

℞ Hydrarg. chlor. mit.,
Pulv. ipecac., ... āā gr. vj.
Potass. nitrat., ... ʒss–j.—M.

Et div. in chart. No. xii.
Sig.: Powder every three hours. HARTSHORNE.

℞ Antimonii et potass. tart., . . . gr. iv.
Tr. opii, ... ʒj.
Aq. camphoræ, ... fʒviij.—M.

Sig.: fʒss every two hours. (*In acute form.*) GRAVES.

PERITONITIS.

℞ Tr. aconiti rad., ... fʒij.
Tr. opii deod., ... fʒvj.—M.

Sig.: Eight drops in water every hour or two.
BARTHOLOW.

℞ Magnesii sulphat., ... ʒiss.

Div. in pulv. No. xii.
Sig.: A powder in hot peppermint water every hour until the bowels are freely opened. (*Use in beginning of attack.*)
MUNDE.

℞ Morph. sulph., ... gr. iv.
Aq. destill., ... ʒij.—M.

Sig.: Ten to fifteen minims as required, hypodermically, to control the vomiting. TAIT.

PERTUSSIS (HOOPING COUGH.)

℞ Tr. opii camp.,
Syr. ipecac., ... āā fʒj.
Syr. scillæ, ... fʒiij.
Syr. tolu., ... fʒss.
Liq. potass. citrat., . . q. s. ad fʒiij.—M.

Sig.: Teaspoonful every two hours for catarrhal stage.
PENROSE.

PERTUSSIS (HOOPING COUGH) (Continued).

℞ Ex. belladonnæ, ... gr. ss.
Pulv. aluminis, ... gr. xxiv.
Syr. zingiber.,
Aquæ, ... āā f℥iss.—M.

Sig.: Teaspoonful every two hours for a child of one year.
GOODHART and STARR.

℞ Ammon. brom.,
Potass. brom., ... āā ℥j.
Tr. belladonnæ, ... f℥j.
Glycerinæ, ... f℥j.
Aq. rosæ, ... q. s. ad f℥iv.—M.

Sig.: Use as spray from four to six times daily. KEATING.

℞ Quiniæ sulphat., ... gr. xij.
Ol. theobrom., ... q. s.—M.

Et ft. suppos. No. xii.

Sig.: Use one or two three times a day for a child of two years.

PHARYNGITIS.

℞ Cocaine muriat., ... gr. x.
Aquæ, ... f℥ss.—M.

Sig.: Use locally. SAJOUS.

℞ Pilocarpinæ muriat., ... gr. ij.
Aquæ,
Glycerinæ, ... āā f℥j.—M.

Sig.: Teaspoonful three times a day. (*In dry pharyngitis.*)
SAJOUS.

℞ Tr. ferri chlor., ... f℥iij.
Potass. chlorat., ... ℥j.
Syr. zingiber., ... f℥ij.
Aquæ, ... q. s. ad f℥iij.—M.

Sig.: f℥j every two hours. STARR.

PHARYNGITIS (Continued).

℞ Zinci sulphat., ... ʒj.
Aquæ, ... fʒj.—M.

Sig.: Use locally. MORRIS.

PHTHISIS.

℞ Codeinæ sulp., ... gr. ⅓.
Acid. hydrocyanic. dil., . . . ♏ij.
Syr. tolu., ... fʒj.—M.

Sig.: Take four times a day. DA COSTA.

℞ Ex. ergotæ fl., ... fʒj.

Sig.: Twenty drops three times a day. (*To relieve diarrhœa and night sweats.*) HODGSON.

℞ Quiniæ sulph., ... gr. j.
Pulv. digitalis, ... gr. ss.
Pulv. opii, ... gr. ¼.
Pulv. ipecac., ... gr. ¼.—M.

Sig.: One pill three or four times a day. (*For fever.*) NIEMEYER.

℞ Tr. benzoin. comp., ... fʒj.
Aq. bullientis, ... Oss.—M.

Sig.: Inhale twice daily. RINGER.

PLEURISY.

℞ Tr. opii deod., ... fʒvj.
Tr. aconiti rad., ... fʒij.—M.

Sig.: Eight drops in water every hour or two. (*In acute stage before effusion.*) BARTHOLOW.

℞ Potass. acetat., ... gr. xv.
Spt. æther. nitro., ... fʒss.
Vini ipecac., ... gtt. iij.
Syr. tolu., ... fʒss.—M.

Sig.: Take four times daily. (*In subacute pleurisy.*) DA COSTA.

PLEURISY (Continued).

℞ Potass. acetat., ... ʒvss.
Spt. æther. nit., ... ʒij.
Aquæ, ... ad fʒviij.—M.

Sig.: fʒss every three or four hours. (*In pleuritic effusion.*)
HARTSHORNE.

℞ Morphiæ sulphat., ... gr. ¼.
Quiniæ sulphat., ... gr. xv.—M.

Et div. in chart. No. i.
Sig.: Take at once. (*To abort an incipient pleurisy.*)
BARTHOLOW.

℞ Tr. iodinii, ... fʒss.
Potass. iodid., ... ʒij.
Aquæ, ... fʒij.—M.

Sig.: Apply on the affected side of chest. NIEMEYER.

℞ Morphiæ acetat., ... gr. ss.
Potass. acetat., ... ʒss.
Tr. veratri viridis, ... ♏ xxiv.
Syr. tolu., ... fʒss.
Liq. potass. citrat., ... fʒiiss.—M.

Sig.: fʒij every three hours. (*In dry pleurisy.*) DA COSTA.

PNEUMONIA.

℞ Potass. iodi., ... ʒj.
Ammon. chlor., ... ʒiss.
Mist. glyc. co., ... fʒvj.—M.

Sig.: fʒss four times a day, to promote absorption.
DA COSTA.

℞ Tr. veratri viridis, ... ♏ xl.
Spt. æther. nitros., ... fʒvj.
Liq. potass. citrat., ... fʒivss.
Syr. zingiber., ... ad fʒvj.—M.

Sig.: Tablespoonful every three hours. (*In the early stage.*)
DA COSTA.

PNEUMONIA (Continued).

℞ Pulv. digitalis, ... gr. vj.
Quiniæ sulphat., ... gr. xij.
Ex. opii,
Ex. ipecac., ... āā gr. iij.—M.

Et ft. pil. No. xii.

Sig.: One pill three times a day with the preceding mixture.
DA COSTA.

℞ Thallin sulphat., ... gr. xxxij.
Aq. aurant. flor., ... f℥j.—M.

Sig.: f℥j every three hours till the fever declines. OSLER.

℞ Tr. aconiti rad., ... f℥ij.
Tr. opii, ... f℥iij.—M.

Sig.: Thirteen drops at once, followed by five drops every hour or two. (*In stage of congestion.*) BARTHOLOW.

PURPURA.

℞ Syr. ferri superphosphitis,
Liq. hydrogen. perox. (10 vol.)
Glycerinæ, ... āā f℥iss.
Aquæ, ... q. s. ad f℥vj.—M.

Sig.: Tablespoonful three times a day. GUITÉRAS.

℞ Ol. terebinth., ... f℥iij.
Ex. digitalis fl., ... f℥j.
Mucil. acaciæ, ... f℥ss.
Aq. menthæ pip., ... f℥j.—M.

Ft. emuls.

Sig.: Teaspoonful every three hours. BARTHOLOW.

℞ Acid. gallici, ... ℥ss.
Acid. sulphuric. dil., ... f℥j.
Tr. opii deod., ... f℥j.
Infus. rosæ co., ... f℥iv.—M.

Sig.: f℥ss every four hours. BARTHOLOW.

QUINSY. (See TONSILLITIS.)

RENAL CALCULI.

℞ Atropiæ sulphat., ... gr. $\frac{1}{80}$.
Morph. sulphat., ... gr. $\frac{1}{4}$.—M.

Sig.: Give hypodermically.

℞ Liq. potassæ, ... f℥ss.
Tr. humuli, ... f℥iss.
Infus. calumbæ, ... f℥iv.
Syr. aurant. cort., ... f℥ij.—M.

Sig.: f℥ss three times a day. GREEN.

RHEUMATISM (ACUTE).

℞ Sodii salicylat., ... ℥ss.
Tr. lavandulæ comp.. . . . . f℥iv.
Glycerinæ, ... f℥ss.
Aquæ, ... q. s. ad f℥viij.—M.

Sig.: Tablespoonful every hour or two until pain and fever abate. MINOT (Mass. Gen. Hos.).

℞ Sodii bicarb., ... ℥iv.

Div. in chart. No. xii.

Sig: Powder in half-tumblerful of water every four hours until the urine is alkaline. LOOMIS.

℞ Liq. opii sed., ... f℥j.
Potass. bicarbonat., ... ℥iv.
Glycerinæ, ... f℥ij.
Aq. bullientis, ... f℥ix.—M.

Sig.: Soak a piece of flannel in the above hot solution and wrap around painful joint. OSLER.

RHEUMATISM (CHRONIC).

℞ Pulv. resinæ guaiaci,
Potass. iodid., ... āā ℥j.
Tr. colchici sem., ... f℥iij.
Aq. cinnam.,
Syr. sim., . . . q. s. ad ft. f℥vj.—M.

Sig.: Dessertspoonful three times a day. PEPPER.

RHEUMATISM, CHRONIC (Continued).

℞ Liq. potass. arsenitis, ... fʒij.
Potass. iodid., ... ʒij.
Syr. simp., ... fʒiij.—M.

Sig.: Teaspoonful three times a day after meals.

DA COSTA.

℞ Tr. aconiti,
Chloroform.,
Aq. ammon., ... āā fʒij.
Lini saponis co., ... ad fʒviij.—M.

Sig.: Use locally. JEFFERSON HOSPITAL, PHILA.

℞ Potass. et sodii tartratis, ... ℥ss.
Potass. nitratis, ... ʒv.
Vini colchici sem., ... fʒij.
Aquæ, ... q. s. ad fʒij.—M.

Sig.: Teaspoonful three times a day.

BELLEVUE HOSPITAL, N. Y.

RUBELLA. (See MEASLES.)

SCIATICA.

℞ Saloli,
Sacch. lact., ... āā ʒiij.

Div. in pulv. No. xii.

Sig.: One powder every four to six hours. ASCHENBACH.

℞ Potass. iodid., ... ℈j.
Decoct. sarsap. co., ... fʒij.—M.

Sig.: To be taken three times a day. (*Chronic cases.*)

WARING.

℞ Pulv. sulphuris sub., ... ℥iv.

Sig.: Dust thickly on the limb and envelop it in soft flannel.

RINGER.

℞ Veratriæ, ... ℈j–ij.
Adipis, ... ℥j.—M.

Sig.: Rub well into painful part. TURNBULL.

SCIATICA (Continued).

℞ Antipyrin, ... ʒij.
Syr. aurant. cort., ... fʒss.
Aq. aurant. flor., ... ad fʒij.—M.

Sig.: A dessertspoonful every hour to four hours, until three to six doses are taken. GERMAIN SÉE.

℞ Morphiæ sulph., ... gr. ss.–$\frac{3}{3}$.
Atropiæ sulph., ... gr. $\frac{1}{25}$.
Aquæ destillat., ... ♏xx.—M.

Sig.: Inject deeply into the muscle over the course of the nerve. BROWN-SÉQUARD.

℞ Tr. aconiti rad.,
Tr. colchici sem.,
Tr. belladonnæ,
Tr. cimicifugæ, ... āā fʒj.—M.

Sig.: Twelve drops every four to eight hours.
J. T. METCALF.

℞ Chloroformi, ... fʒij.

Sig.: Five to fifteen minims hypodermically near the seat of pain. BARTHOLOW.

℞ Tr. colchici sem., ... gtt. xv.
Potass. iodid., ... gr. x.
Tr. zingiber., ... gtt. x.
Syr. simp.,
Aquæ, ... āā q. s. ad fʒij.—M.

Sig.: Apply a strip of blistering plaster over the course of the nerve, and give the above in water three times a day.
DA COSTA.

SCORBUTUS (SCURVY).

℞ Potass. bitartratis, ... ʒj.
Olei limonis, ... ♏xv.
Sacch. alb., ... ʒij.
Aquæ bullientis, ... Oij.

Ft. haustus.

Sig.: Use when cold as a drink. TANNER.

SCORBUTUS (SCURVY) (Continued).

℞ Acid. muriat., ... fʒj.
Mellis,
Aquæ rosæ, ... āā fʒj.—M.

Sig.: Apply three or four times daily to the gums.

BRANDA.

SMALLPOX.

℞ Acid. boric, ... ʒiss.
Glycerinæ, ... fʒj.
Listerine, ... fʒij.
Aquæ, ... q. s. ad fʒvj.—M.

Sig.: Mouth wash.

℞ Antifebrin, ... ʒj–ij.
Spt. vini gal., ... fʒss.
Syr. simp., ... q. s. ad fʒij.—M.

Sig.: Teaspoonful every four hours to reduce temperature.

HEINZELMANN.

℞ Mercurial ointment, ... 24 parts.
Yellow wax, ... 10 parts.
Black pitch, ... 6 parts.—M.

Sig.: Apply on mask night and morning.

CHILDREN'S HOSPITAL OF PARIS.

SPINAL SCLEROSIS.

℞ Argent. nit.,
Ex. belladonnæ, ... āā gr. vj.
Ex. gentian., ... q. s.—M.

Et ft. pil. No. xxiv.

Sig.: One after each meal. A. McL. HAMILTON.

℞ Potass. iodid., ... ʒvj.
Ferri et ammon. citrat., ... ʒij.
Tr. aurant. cort.,
Syr. simp., ... āā fʒiij.
Aq. menth. pip., ... ad fʒiv.—M.

Sig.: fʒj in water an hour after meals. SWERINGEN.

SPLEEN (CHRONIC ENLARGEMENT OF).

℞ Ungt. hydrarg. iodi. rub., . . . ℥j.

Sig.: Rub over tumor before a hot fire. MORRIS.

STOMATITIS.

℞ Potass. chlorat., ... gr. x.
Listerini, ... ℥ij.
Aquæ, ... q. s. ad f℥j.—M.

Sig.: Use as mouth wash.

℞ Potass. chlorat., ... gr. xlviij.
Acid. muriat. dil., ... f℥j.
Syrupi, ... f℥ss.
Aquæ, ... q. s. ad f℥iij.—M.

Sig.: Teaspoonful diluted every two hours for a child of three years. STARR.

℞ Potass. iodid., ... gr. iij.
Glycerinæ, ... f℥ij.
Aq. rosæ, ... q. s. ad f℥j.—M.

Sig.: Use locally.

℞ Potass. chlorat., ... ℥j.
Acid. carbol., ... gr. ij.
Glycerinæ, ... f℥j.
Aquæ, ... q. s. ad f℥viij.—M.

Sig.: Apply to ulcer twice daily, and use as mouth wash. (*Ulcerative.*) STARR.

SYPHILIS.

℞ Hydrarg. chlor. corros., ... gr. iv.
Tr. benzoin., ... f℥ss.
Aq. cologne., ... f℥j.
Aq. rosæ, ... f℥ivss.—M.

Sig.: Apply locally with a sponge to the skin for twenty minutes. (*For squamous syphilides.*) S. W. GROSS.

SYPHILIS (Continued).

℞ Hydrarg. chlor. corros., . . . gr. j.
Potass. iodid., ... ʒij.
Tr. gentian. co., ... fʒiij.—M.

Sig.: fʒj in water after meals. CHARITY HOSPITAL, N. Y.

℞ Potass. iodid., ... ʒij.
Ammon. carb., ... ʒss.
Tr. cinchonæ co., ... fʒiv.
Syr. aurant. cort., ... fʒiss.
Glycerinæ, ... ad fʒj.—M.

Sig.: fʒ, well diluted, after each meal. KEYES.

℞ Hydrarg. chlor. corros.,
Ammon. chlor., ... āā gr. iij.
Tr. cinchonæ comp.,
Aquæ, ... āā fʒiij.—M.

Sig.: Teaspoonful three times a day. BUMSTEAD.

THRUSH.

℞ Sodii salicylat., ... ʒiss.
Aq. destillat., ... fʒj.—M.

Sig.: Apply several times daily. HIRTZ.

℞ Potass. iodid., ... gr. j.–v.
Aquæ, ... fʒj.—M.

Sig.: Apply locally. BARTHOLOW.

℞ Sodii salicylat.,
Sodii borat., ... āā gr. x.
Acid. carbol., ... gr. j.
Glycerinæ, ... fʒij.
Aq. rosæ, ... q. s. ad fʒj.—M.

Sig.: Use locally. STARR.

℞ Glycer. boracis, ... fʒj.—M.

Sig.: Use locally.

THRUSH (Continued).

℞ Potass. chlorat., ... ʒss.
Glycerinæ,
Listerine, ... āā fʒss.
Aquæ, ... q. s. ad fʒij.—M.

Sig.: Use locally.

℞ Mel. boracic., ... ʒj.—M.

Sig.: Apply to patches with a brush. RINGER.

℞ Papain, ... gr. xxx.
Glycerinæ, ... fʒiiss.
Aq. destillat., ... ad fʒv.—M.

Sig.: Apply to patches four or five times daily.
SCHMIDIGER.

TONSILLITIS.

℞ Tr. guaiaci ammoniat., . . . fʒij.

Sig.: Teaspoonful in half a glassful of milk three or four times daily. (*Early stage.*) SAJOUS.

℞ Hydrarg. chlor. mit., ... gr. j.
Sacch. lact., ... ℈j.—M.

Et div. in chart. No. xx.

Sig.: Powder every two hours. BARTHOLOW.

℞ Sodii bicarb., ... ʒss.

Sig.: Apply locally to the tonsil in powder or warm solution.
BAKER.

℞ Sodii salicylat., ... ʒiij.
Syr. acaciæ, ... fʒss.
Aq. cinnam., ... ad fʒiij.—M.

Sig.: ʒij every three hours. EASBY.

TONSILS, HYPERTROPHY OF.

℞ Ammon. iodid., ... gr. x.-ʒss.
Glycerinæ, ... fʒj.—M.

Sig.: Apply with brush at night. WARING.

TONSILS, HYPERTROPHY OF (Continued).

℞ Liq. ferri perchlor., ... f℥ss–f℥j.
Glycerinæ, ... f℥j.—M.

Sig.: Paint over tonsils once or twice daily. MACKENZIE.

ULCER (GASTRIC).

℞ Argenti nitrat., ... gr. v.
Tr. opii, ... f℥iss.
Aq. anisi, ... ad f℥iiss.—M.

Sig.: Teaspoonful three times a day. THOMSON.

℞ Liq. potass. arsenitis, ... f℥ss.—M.

Sig.: One drop repeated as required, to relieve the pain and vomiting. BARTHOLOW.

THE

EXAMINATION OF URINE,

CHEMICAL AND MICROSCOPICAL,

FOR

CLINICAL PURPOSES.

ARRANGED IN THE FORM OF

QUESTIONS AND ANSWERS.

BY

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COLORED PLATE,
AND NUMEROUS ILLUSTRATIONS.

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PREFACE.

The favorable reception accorded to my little book on "The Essentials of Medical Chemistry," together with a wish expressed by many of its readers that it should also contain a special part on urinology and urinalysis, have caused the preparation of this little volume on "The Examination of Urine, Chemical and Microscopical, for Clinical Purposes."

As its scope has a more clinical than purely chemical bearing, it was thought best to present it in separate form rather than as part of a work on medical chemistry. It is intended as an aid for the student in his *studies* on this subject, more so than as a manual for his laboratory practice in chemistry.

The author trusts that as such it will be a welcome friend to the student and help him to master the essentials of this branch of medical science, which is of such great importance in the study of disease.

L. W.

Philadelphia, Pa.,
333 South Twelfth St.,
February, 1890.

LIST OF ILLUSTRATIONS.

	PAGE
Vogel's Scale of Urinary Colors,	*Frontispiece*
Urinometer,	21
Urea and Urea Nitrate,	23
Uric Acid,	29
Acid-Sodium Urate,	30
Triple-phosphate and Ammonium Urate,	30
Hippuric Acid,	31
Pus Corpuscles and Epithelial Cells,	40
Blood Corpuscles,	42
Teichmann's Hæmin Crystals,	43
Esbach's Albuminometer,	46
Renal Epithelial Cells,	48
Epithelial Cells, Urethral and Vaginal,	48
Hyaline and Epithelial Casts,	49
Waxy Casts,	50
Granular Casts,	50
Crystals of Cystin,	52
Leucin and Tyrosin,	52
Calcium Oxalate,	53
Microörganisms,	54
Johnson's Picro-saccharometer,	60

TABLE OF CONTENTS.

	PAGE
Color of Urine,	18
Specific Gravity,	21
Normal Constituents,	22
Normal Organic Constituents,	22
Urea,	23
Uric Acid,	28
Hippuric Acid,	31
Normal Inorganic Constituents,	32
Chlorides, Phosphates and Sulphates,	34
Abnormal Constituents,	37
Biliary Pigments and Acids,	38
Pyuria,	39
Chyluria,	40
Hæmaturia and Hæmoglobinuria,	41
Albuminuria,	44
Urinary Sediments,	47
Tube Casts,	49
Glycosuria,	54
Lead and Mercury in Urine,	61
Dietrich's Table,	63

THE EXAMINATION OF THE URINE.

Which are the objective points in the examination of urine for clinical purposes?

The quantity, appearance, color, odor, reaction, specific gravity, increase or decrease of normal ingredients and presence or absence of abnormal or adventitious substances.

When should the specimen be obtained for examination?

For qualitative purposes the specimen may be taken at any time of the day, the morning urine in preference. For quantitative examination a specimen of the total urine in 24 hours must be used.

How is the total urine of the 24 hours to be collected?

The urine passed at a certain hour is to be thrown away, all subsequently passed up to the same hour of the next day is to be collected in a clean glass jar or bottle and the amount measured.

How soon thereafter should the urine be examined, and why?

Shortly after the specimen is obtained, as putrescence will rapidly set in, which will change the character of some of the ingredients.

What is the average daily quantity voided under normal conditions?

1200 to 1500 c.c., or about 40–50 fluidounces.

Under which normal conditions is this increased or diminished?

Copious drinking increases the quantity; also, a lower temperature or great humidity of the atmosphere; free sweating, purgation or emesis will diminish it.

Which pathological conditions increase the quantity and which diminish it?

Diabetes insipidus and diabetes mellitus largely increase the quan-

tity, acute febrile affections diminish it. It is also increased in certain nervous affections and diminished in hydropic conditions and some renal diseases.

In which way is the appearance of the urine to be noted?

If clear, turbid, or containing a sediment.

Which are the principal causes of turbidity or sedimentation in the urine?

The presence of mucus, precipitation of the earthy phosphates from alkaline reaction, separation of urates by lower temperature, and pathologically the presence of pus or fat in minute subdivision, the latter causing a layer to rise to the surface.

What is the normal color of urine, and how are the variations expressed?

The normal color of urine is yellow, the variations being expressed by Vogel's scale, which contains three yellow, three red and three brown tints, termed respectively pale yellow, light yellow, yellow, reddish-yellow, yellowish-red, red, brownish-red, reddish-brown, brownish-black. (See Frontispiece.)

Which are the normal urinary coloring bodies?

Indican and urobilin; others frequently described are modifications of the latter.

How is urobilin derived?

From the blood; the hæmoglobin changing to hæmatin, this to bilirubin, which by taking up hydrogen is changed to hydro-bilirubin, identical with urobilin.

What is indican, and how derived?

This is sometimes called uroxanthin, and is chemically potassium indoxyl-sulphate, a normal component of the urine, varying in quantities, and derived from disturbances of intestinal digestion and consequent absorption of the indol of the fæces.

How is the presence of indican in the urine demonstrated, and how determined?

Indican can be demonstrated by mixing urine with about ¼ quantity of HCl, when, upon standing 24 hours, a red, purple, or blue color will appear, which if shaken out with chloroform and the sepa-

rated solution compared with a standard solution of indigo in chloroform diluted to the same tint, may be expressed in the quantity of indigo it represents.

What is the color of urine in icterus, and by what produced?

The urine in this condition is of a yellow or greenish, or even greenish-black color, caused by the presence of biliary coloring matter.

How may the presence of blood in the urine affect its color?

It will cause a change of color from light red to brown and almost black.

Do medicinal agents change the color of the urine?

Many of them do: Thus, it is turned brown or black after ingestion of carbolic acid and gallic acid, yellow after santonin, rhubarb (changed to red by addition of ammonia), also, after senna, logwood, etc.

What pathological condition gives rise to a dark brown or blackish color of urine, and why?

Melanotic tumors, owing to the elimination by the kidneys of uromelanin, a black coloring body corresponding to the choroidal pigment.

Describe the odor of fresh normal urine.

Fresh normal urine has a specific, not disagreeable, aromatic odor, due to the organic acids of the aromatic series.

How does this change on standing?

The urine turns alkaline in reaction, and then ammoniacal decomposition takes place, giving rise to a disagreeable ammoniacal odor. This may take place already within the bladder in cystitis.

In what conditions and by which medicines or food is the odor of the urine modified?

In diabetes the urine has often a fruity odor, due to acetone. Asparagus gives it a disagreeable odor. Spirits of turpentine an odor not unlike violets. Copaiba, cubebs, balsam of tolu and oil of sandalwood give it an aromatic odor.

What is the effect of mineral acids, and what of fixed alkalies on the normal odor of the urine?

Mineral acids interfere with the normal odor, fixed alkalies make it aromatic.

Has the urine containing blood a special odor?

It has a slightly putrid odor, resembling that of high game.

What is the reaction of normal urine? What due to?

It is normally slightly acid, due to the presence of acid sodium phosphate. The acid reaction is greatest in the urine of the night, less in that voided after meals.

How is the reaction of the urine ascertained?

If blue litmus paper is touched with a drop of acid urine it will be turned to a red color; if the urine is alkaline, it will turn red litmus paper blue. If, upon exposure in the latter case until dry the red color is restored, this alkalinity is due to ammonia.

In which way is the degree of acidity of urine determined?

By acidimetry, *i. e.*, titration with a decinormal solution of potassium hydrate, expressing the result in the corresponding amount of oxalic acid.

What is the relation of the acidity of urine to disease?

Many diseases show a direct relation with it. Thus, in typhoid fever the acidity is in direct ratio with the fever, in rheumatism with the pain, while in pneumonia, pleurisy, emphysema, etc., the urine is very acid.

Which systemic conditions may cause an alkaline reaction of the urine?

Fear, nervous affections, etc., may bring about alkalinity. Irrespective of food, it is associated with anæmia, debility, etc. This alkalinity is due to fixed alkalies.

Under what local conditions may the urine become alkaline?

In cystitis the urea is decomposed into ammonium carbonate, which renders it alkaline, with ammoniacal odor.

What effect has the alkaline reaction on the urine?

The alkalinity from fixed alkalies causes the precipitation of the earthy phosphates, rendering it of white color. Ammoniacal alkalescence brings about the formation of triple-phosphate.

How do medicinal agents influence the reaction of the urine?

Mineral acids do not directly influence its reaction; alkaline hydrates and carbonates render it less acid or alkaline; the salts of the

vegetable acids, being eliminated as alkaline carbonates, produce alkalinity; benzoic acid or alkaline benzoates are converted into hippuric acid and increase the acidity of the urine.

What does the specific gravity of the urine represent?

The amount of solids contained in solution therein.

State the average specific gravity of normal urine and its variations under various conditions.

The average specific gravity of the normal urine is between 1015 and 1025. When great quantities of liquids are ingested it may fall to 1002, and when great amounts of fluids are withdrawn it may reach as much as 1040.

FIG. 1.

Urinometer.

In which diseases is the urine characteristically high, and in which relatively low?

In diabetes mellitus it is always high, and may reach 1050; in the various forms of Bright's disease, as well as in amyloid degeneration of the kidneys, it is low, reaching 1005 to 1004.

How is the specific gravity of urine ascertained?

With the urinometer (Fig. 1); fill the cylinder for that purpose about three-quarters full, then carefully float the urinometer in it and add enough urine to fill the cylinder to the top, reading off the degree of immersion over the top of the liquid. For very accurate determinations, the specific gravity should be taken with the specific-gravity bottle.

For what purpose does the knowledge of the specific gravity of urine serve?

For the approximate determination of the quantity of solids. Thus, if by Trapp's formula the last two figures of the specific gravity are multiplied by 2, it gives the amount of solids contained in 1000 parts; (the factor 2.33 is sometimes used as being more accurate, but 2 suffices for clinical purposes).

The Normal Constituents of Urine.

What constitutes the normal solid components of urine?

The products of retrograde metamorphosis of nitrogenous bodies, together with the inorganic matter eliminated as waste material by the kidneys from the circulation.

How are they classed according to their chemical character, and what are they respectively?

They may be classed as organic, which form the greater part, and inorganic. The principal ones of the former class are urea, uric acid, hippuric acid, kreatinin, xanthin, sarkin, oxalic acid, oxaluric acid, aromatic ethyl-sulphuric acids, sulphocyanic and succinic acids, sugar, lactic acid, pigments, and extractives. The inorganic are chlorides, phosphates and sulphates of potassium, sodium, ammonium, calcium and magnesium, iron, silicic acid, nitrites and nitrates, also hydrogen peroxide.

Are these constant in proportion, and what influences their presence in the urine?

They are subject to continuous change: thus, a more liberal animal diet increases the amount of urea; age, sex and great exertions influence their amount as well as pathological conditions.

Normal Organic Constituents of the Urine.

Which is the principal solid ingredient of the urine, and what is it?

Urea, a carbonyl diamide, often termed carbamide (CON_2H_4), the ultimate product of oxidation of the albuminoids introduced into or composing the body.

How much urea is excreted under normal conditions daily?

500 grains, or about 30 grammes, which, however, may vary according to the character of the ingested food.

How may the urea be separated and demonstrated from the urine?

By acidulating the condensed urine either with nitric or oxalic acid, and allowing the nitrate or oxalate of urea to crystallize from this.

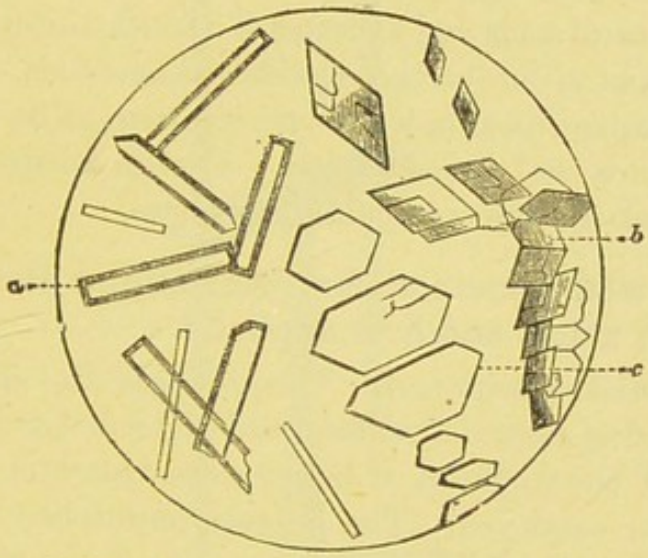
Under which pathological conditions is the elimination of urea increased?

In febrile conditions which do not suppress the renal action. Thus, it is increased in meningitis, typhoid fever, smallpox, erysipelas, intermittent fevers, pneumonia, pleurisy, articular rheumatism with endocarditis, etc.

What pathological conditions diminish the urea in urine?

A diminished nutrition, especially of albuminoids, processes of suboxidation, such as emphysema, valvular disease of the heart and disturbances of the circulation, in which the amount of urea pro-

FIG. 2.



a, urea; *b*, rhombic, and *c*, hexagonal plates of urea nitrate.

duced is less; in other diseases, such as ascites and anasarca, the urea is not secreted, though produced, and is withheld in the circulation; also in cholera and diseases characterized by renal inaction, when with an amelioration the urea reappears. The wasting diseases, such as anæmia, leucocythæmia, phthisis, acute yellow atrophy, etc., are also accompanied by a diminished amount of urea in the urine.

Can urea ever exist as a sediment in urine?

On account of its ready solubility it can never exist as such.

How can urea be recognized under the microscope?

By evaporating a drop of urine cautiously on a slide, when, with a low power, the rhombic prisms of urea may be readily recognized (Fig. 2).

How may the oxalate or nitrate of urea be prepared?

By acidulating condensed urine with either oxalic or nitric acid, when the respective oxalate or nitrate will crystallize out on cooling, in hexagonal plates (Fig. 2).

By what chemical test may urea be recognized?

By the Biuret reaction. This consists of heating a specimen of urea until it ceases to give off ammoniacal vapors; when to the residue a little potassium hydrate is added and a drop of cupric sulphate solution, the color is changed to a reddish violet.

Which are the principal agents to break up urea?

Stronger mineral acids and hydrates of the alkalies cause it to take up water and split up into carbon dioxide and ammonia. Nitrous acid splits it into carbon dioxide water and nitrogen, as do also the alkaline hypochlorites and hypobromites. Certain microörganisms also cause it to break up into ammonium carbonate.

What is Fowler's method for determining the amount of urea in urine, and how applied?

The differential density test. To apply this, the specific gravity of the urine is first accurately determined and noted and then the specific gravity of the specimen of liquor sodæ chloratæ (Labarraque's solution) to be employed. The latter is multiplied by 7 and the product added to the amount of the sp. gr. of the urine; the sum so ascertained is divided by 8, which gives the sp. gr. of a mixture of 1 part of urine and 7 parts of the hypochlorite solution. After this add 1 part urine to 7 parts of Labarraque's solution, and after standing for an hour or two, take the specific gravity of the mixture after the reaction has been completed. This specific gravity subtracted from that first computed for the mixture of the two before reaction, and the difference multiplied by 0.77 gives the percentage of urea in the urine examined.

What is Liebig's method of determining urea by titration?

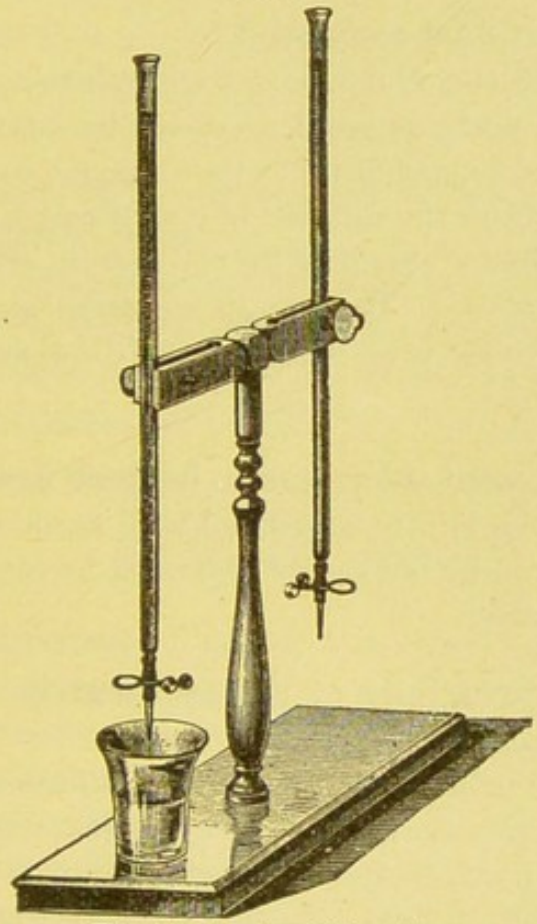
The method depending on the formation of an insoluble compound of mercuric nitrate with urea, and the computation of the amount of urea from a standardized mercuric nitrate solution used for this purpose.

Which are the reagents and apparatus used in this method?

A standardized mercuric nitrate solution, of which each c.c. is equal to 1 centigramme urea. A baryta mixture of 1 part saturated solution of barium nitrate and 2 parts cold saturated baryta water.

A saturated solution of sodium carbonate, a graduated burette, a volume pipette, watch-glasses, glass rod and beaker glass (Fig. 3).

FIG. 3.



Burette-stand and Burettes.

How are the sulphates and phosphates and carbonates first separated in this process?

By mixing 40 c.c. urine with 20 c.c. baryta mixture, after which the liquor is filtered and 15 c.c. is measured into a beaker glass; these 15 c.c. correspond to 10 c.c. urine. At times, if the urine contains an excess of phosphates or alkaline carbonates, it becomes necessary to take more baryta mixture, and then the specimen to be examined must be increased always so as to represent 10 c.c. urine.

In which manner is the titration of the urea performed, and how is the indicator applied?

The mercuric solution is dropped from the burette into the filtered urine, amidst constant stirring, until it ceases to produce a precipitate; then a few drops are taken out into a watch-glass and an equal amount of the soda solution allowed to flow into it. When the resulting reaction begins to show a yellow color, the saturation is complete, if not, more of the mercuric solution is dropped from the burette as above.

How is the result now computed?

As each c.c. of the mercuric nitrate solution is equal to 0.01 gramme urea, as many as were necessary to saturate it were contained in 10 c.c. urine, or ten times that amount constitutes the percentage of urea; from the number of c.c. of mercuric solution 2 c.c. are first to be deducted to allow for the sodium chloride which also enters into this process. Thus if 30 c.c. were used, 28 c.c. would represent 0.28 gramme urea contained in 10 c.c. urine, or 2.8 would be the percentage.

If albumin is present what has to be done first?

The urine must be faintly acidulated with acetic acid, and then the albumin, coagulated by boiling, is separated by filtration before the titration of the urine.

What are the principles of the azotimetric method for the determination of urea in urine?

The urea of the urine is decomposed by a solution of sodium hypobromite (Knop's solution), thus liberating the nitrogen of the urea while the water remains and the carbon dioxide is arrested by the alkaline test solution. The nitrogen is then corrected for temperature, atmospheric pressure and tension of aqueous vapor, and the amount of urea corresponding to each c. c. thereof is equal to 0.0027 gramme.

What comprises the most simple apparatus for this process?

A flask containing 15 c. c. hypobromite solution and also a test tube standing slantingly in it, into which 5 c. c. urine are added. The flask is connected by a rubber tube to an inverted burette or cylinder graduated into c. c.; this latter is contained in a cylinder filled

with water and is immersed therein to the zero mark, so that the water in the graduated tube and the cylinder are on an equal level.

How is the Knop's solution made?

Dissolve 100 grammes sodium hydrate in 250 c.c. water and add 25 c.c. bromine.

How is the process applied?

By allowing the urine to commingle with the hypobromite solution, when the reaction will take place; as the N escapes into the graduated cylinder the latter is raised so as to keep the water inside and outside always on the same level. When the reaction is complete and no more gas given off the number of c.c. are read off.

How is the volume of N so observed corrected and how the urea computed from it?

The volume of nitrogen so observed has to be corrected for temperature, barometric pressure and tension of aqueous vapor. As 1 gramme urea yields 370 c.c. nitrogen at 0°C. and 760 mm. pressure the formula for correction of its observed volume is as follows for 100 c.c. urine:

$$U = \frac{100\ v.\ (b - b')}{760.\ 370.\ x.\ (1 + 0.00366.t)}$$

In this formula

U stands for percentage of urea to be determined.
v the volume of nitrogen read off.
b barometric pressure.
b′ tension of aqueous vapor.
x the measure of urine employed.
t the temperature (C°) at which the process is conducted.

Instead of this correction the corrected weight in milligrammes for each c.c. nitrogen may be taken from Dietrich's table by entering with barometric pressure from above and the temperature from the side, when the corrected weight will be found at the intersection of the two lines. (See Dietrich's table on last page.)

Thus if 10 c.c. N were observed at 15° C. and 740 mm. pressure, each c.c. N would weigh 1.1399 milligramme, or 10 = 11.399, which if multiplied with 2.14 would give the amount of urea in the urine used 24.39388 milligrammes, which if it had been 5 c.c. is multiplied with 20 gives the percentage = 0.49.

Does the method give absolutely correct results?

No, the theoretical amount of N is never obtained; this may be rectified by multiplying the result with 1.044, but even then it is not absolutely correct.

What modifications of the above described apparatus may be used for clinical purposes?

Such apparatus as that of Lyon, which contains on the cylinder, instead of c.c., subdivisions corresponding to the percentage of urea at a temperature of 70° F. Pressure and tension of aqueous vapor may be then neglected if the temperature is at or about 70° F.

What is uric acid?

A bibasic acid of the formula $C_5H_4N_4O_3$ which in the form of salts is a normal ingredient of the urine and next to urea the principal eliminant of nitrogen from the body; the quantity eliminated under normal conditions during 24 hours amounts from about 0.5 to 1 gramme.

Under which normal and abnormal conditions is the amount of uric acid in the urine increased or decreased?

Nitrogenous food increases and carbohydrates diminish the uric acid in the urine. In diseases of the respiratory and circulatory system, as in pneumonia, capillary bronchitis, pleuritic exudations, pericarditis, etc., the amount in the urine is increased, also in most fever processes. Chronic diseases are accompanied by a diminished amount of uric acid in the urine; it is diminished also after profuse hemorrhages and in anæmia, chlorosis, spinal and renal affections, as well as in chronic rheumatic and gouty conditions. During the exacerbations of malarial fevers there is an increase in the elimination of uric acid, as well as in typhoid fever, inflammatory rheumatism, smallpox and in septic fevers.

How is uric acid separated from urine?

By acidulating 500 grammes urine with 10 grammes hydrochloric acid, when, after standing 24 hours, the uric acid will crystallize from it all but a very small amount held in solution.

What are the physical properties and microscopical appearances of uric acid separated from urine?

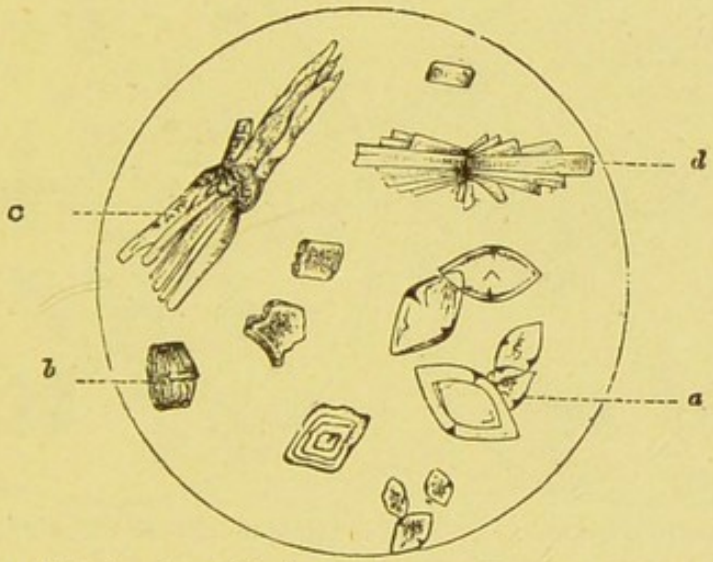
Uric acid is very little soluble in water, about 1 to 18,000, insoluble

in alcohol and ether, readily soluble in the neutral alkaline phosphates and carbonates. The crystals separated from the urine appear to the naked eye as small reddish-brown particles. Microscopically they present a variety of shapes which, however, may be regarded as modifications of rhombic plates. The most frequent of these are the whetstone or lozenge form rounded off at their obtuse angles; other forms resemble barrels, sheaves, rosettes, combs, etc, (Fig. 4).

Is free uric acid ever present in the urine?

It may be present in the urine under abnormal conditions at micturition, when it may give rise to the formation of urinary concretions.

FIG. 4.



a. Rhombic crystals of uric acid, of whetstone or lozenge shape. *b*. Barrel shaped. *c*. Sheaves. *d*. Rosettes of whetstone shaped crystals.

Which are the principal salts of uric acid present in the urine?

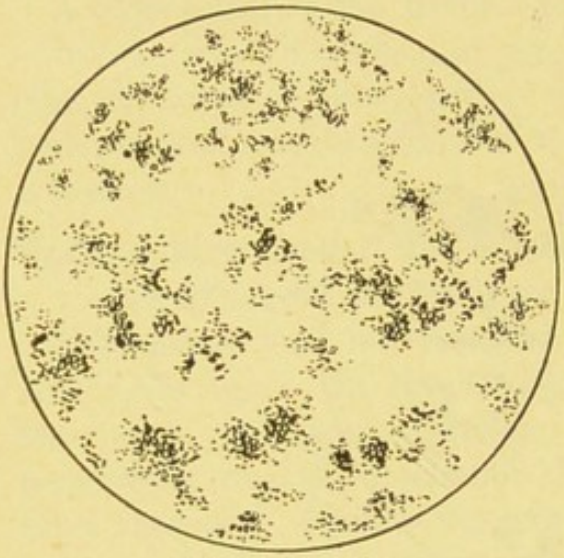
The salts of the alkalies and alkaline earths; these form both neutral and acid salts, the neutral salts being more frequent in normal urine and are more soluble than the acid salts; the acid sodium urate and potassium urate appear frequently as sediments in the acid urine of catarrhal and rheumatic affections and in fevers, and have a reddish color, owing to the presence of uroerythrin (lateritious deposits). They may be recognized by being redissolved in the urine on heating and on addition of alkaline hydrates, also by the microscope as pre-

senting an amorphous granular appearance, as in accompanying cut (Fig. 5). The acid ammonium urate is often found in alkaline urine together with triple phosphate and presents the shape of yellow spheres with one or more hooklets attached, often occurring in attached pairs (Fig. 6). Ammonium urate is present in some vesical concretions and when preformed in the bladder may be the cause thereof.

By which test may the presence of uric acid be shown, and how is it applied?

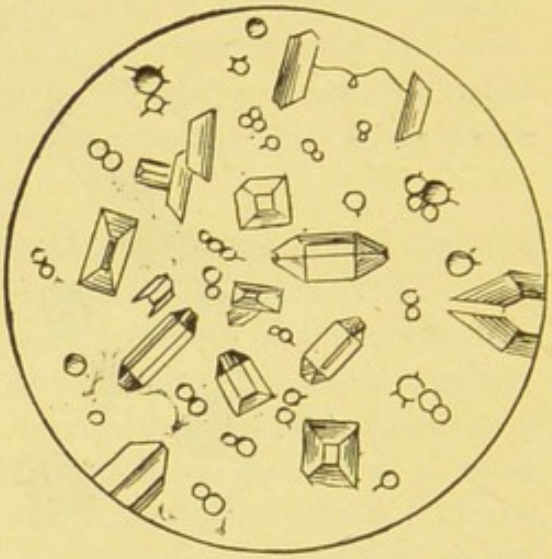
By the murexide test; this consists in dissolving in an evaporating

FIG. 5.



Acid sodium urate.

FIG. 6.



Triple phosphate and ammonium urate.

dish a small quantity of the substance to be examined in a few drops of nitric acid, and evaporating over a moderate heat to dryness. If the dry residue is touched with a drop of ammonia water or exposed to its vapors the bright purple color of murexide will appear if uric acid was present.

How is the amount of uric acid in urine determined?

By acidulating 200 c.c. urine with 10 c.c. HCl and setting it aside for 48 hours. The crystals which have separated are now collected on a weighed filter and well washed with cold water; the filter is then dried until it ceases to lose weight, and for each 100 c.c. fluid employed 0.0038 gramme uric acid is to be added to the increase of

weight over that of the empty filter, which gives approximately the amount of uric acid present.

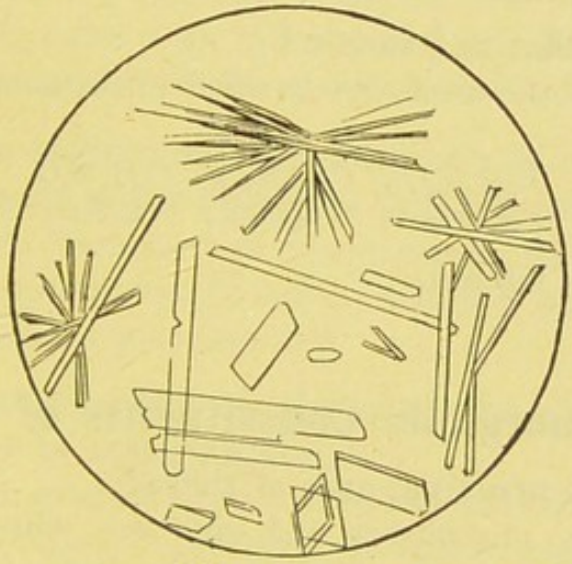
What is hippuric acid?

A normal monobasic acid of the urine, of the formula $C_9H_9NO_3$, which may be regarded a benzoyl glycocin, as it splits up into benzoic acid and glycocin.

In what amounts is it present in the urine and under what conditions may this be increased?

About 0.5 to 2.0 grammes are excreted during 24 hours in the urine, which is increased by vegetable and diminished by animal

FIG. 7.



Hippuric acid.

diet. Benzoic, cinnamic and quinic acids are converted in the body into hippuric acid and excreted as such. It is said to be increased in diabetes mellitus, also in hepatic affections and in jaundice.

State the physical properties and microscopic appearance of hippuric acid.

It is readily soluble in alcohol, less so in ether, and dissolves only in 600 parts of water; it crystallizes in colorless, long, four-sided rhombic prisms, which frequently form stellate bundles (Fig. 7).

What is kreatinin?

A constant component of the urine, of the formula $C_4H_7N_3O$,

supposed to be derived from the kreatin of the muscles. About 1 gramme is daily excreted under normal conditions and mixed diet.

State its properties.

It is a basic body which, when pure, forms colorless, prismatic crystals, soluble in 11 parts of water, and readily soluble in alcohol. In alkaline solutions it changes to kreatin, which, with acids, loses H_2O, and forms again kreatinin.

Under which pathological conditions is it increased, and under which decreased?

In acute diseases, especially typhoid fever, pneumonia, etc., it is increased in quantity; in anæmia, chlorosis, marasmus, tuberculosis, and progressive muscular atrophy it is diminished.

What are xanthin and sarkin?

Extractives of the urine closely allied in chemical composition to uric acid,

$C_5H_4N_4O_3$ (uric acid.), $C_5H_4N_4O_2$ (xanthin.), $C_5H_4N_4O$ (sarkin.)

but of no clinical interest.

Normal Inorganic Constituents of the Urine.

Which are the principal ones of these?

The chlorides, phosphates, and sulphates, which are principally derived directly from the food, but the latter two also result as oxidation products from albuminoids and other bodies which contain phosphorus and sulphur.

Name the most important inorganic salts.

The one which occurs in largest quantities is sodium chloride, next is sodium acid phosphate, also, calcium and magnesium phosphates, the sulphates of sodium and potassium, and traces of iron and silicic acid.

What is the normal amount of chlorides excreted in 24 hours, and what is their importance?

The sodium chloride, which is the principal one of them, is excreted to the amount of about 16.5 grammes in the 24 hours, vary-

ing with the quantity ingested. A considerable amount is always retained in the circulation, serving probably the purpose of cell-nutrition by facilitating the osmotic process. A surplus appears in the urine.

Under which pathological conditions are the chlorides in the urine decreased?

They decrease in all acute febrile diseases, and may, indeed, disappear altogether, to reappear with convalescence. Especially is this the case in diseases accompanied with exudations and transudations, which retain the surplus chlorides until their formation is complete. Thus we find the chlorides diminished in pneumonia, pleurisy, pericarditis, peritonitis, meningitis, also in typhoid fever and rheumatic fever. A decrease of chlorides in the urine is also marked in the nephritis accompanied with albuminuria and dropsy. During the paroxysms of malarial fever the excretion of chlorides is increased.

What are the indications for prognosis of a decrease or reappearance of the chlorides in the urine?

A considerable diminution or disappearance makes the prognosis grave, their reappearance favorable, which is again disturbed by their decrease during convalescence.

How may the chlorides in the urine be shown and their quantity be approximated?

To a specimen of urine in a test tube add a few drops of nitric acid, and then of a solution of silver nitrate sufficient until no more precipitate forms. The precipitate will be dense and curdy if the chlorides are present in normal quantities, milky if diminished, and faint if almost or entirely absent. If the bulk of the precipitate is compared with that of a normal specimen, the relative amount may be approximated.

How are the chlorides in the urine determined by volumetric analysis?

To do so accurately 10 c.c. urine are evaporated to dryness in a porcelain dish and incinerated with 2 grammes pure potassium nitrate until the charred organic matter is burned off. The residue is now dissolved in about 50 c.c. distilled water and acidulated with dilute nitric acid, the excess of which is neutralized by the addition of a

little pure calcium carbonate; a few drops of neutral potassium chromate are then added as indicator.

The chlorides are now titrated with a standardized solution of silver nitrate, each c.c. of which corresponds to 0.01 gramme sodium chloride. The silver nitrate solution is added from a burette to the canary-yellow fluid until a slight change to orange shows the complete precipitation of the chlorides. For each c.c. of the former count then 0.01 NaCl, or ten times that amount, to get the percentage.

Thus, if 10 c.c. urine were used and 15 c.c. $AgNO_3$, the amount in 10 c.c. would be 0.15 grammes, or the percentage 1.5.

May the process of incineration and subsequent acidulation be dispensed with for clinical purposes?

Yes; unless the urine is alkaline, very highly colored or albuminous. If such is not the case the 10 c.c. urine should be diluted with 40 c.c. distilled water, a few drops of neutral potassium chromate added and then titrated and computed as above. As the result is generally too great, 1 c.c. is, however, deducted from the quantity of silver nitrate solution used.

In which compounds is phosphoric acid present in the urine?

As phosphates of the alkalies and alkaline earths, of which there are two-thirds of the former and one-third of the latter. The phosphates of the alkalies are principally present as sodium acid-phosphate, to which the acid reaction of normal urine is due; those of the alkaline earths as phosphates of calcium and magnesium. The total quantity of phosphoric acid normally eliminated in 24 hours is between 2 and 5 grammes.

From what is the phosphoric acid of the urine derived?

From the food and also from the retrograde metamorphosis of tissues containing phosphorus. Thus it is diminished on fasting, and increased by animal diet.

Under which pathological conditions is the phosphoric acid of the urine increased or diminished?

In the beginning of the febrile processes, the P_2O_5 is usually diminished, and decreases still more with fatal termination. With defervescence and convalescence the amount is increased, while in

chronic conditions no constant relations have been established, though the earthy phosphates are no doubt increased in cerebral affections, rheumatism, osteomalacia, rachitis, whereas a decrease has been established in chronic spinal disease, renal affections and dropsy.

How can the earthy and how can the alkaline phosphates of the urine be separated?

If an alkaline hydrate, KOH or NaOH, is added to urine, and the mixture heated to the boiling-point, the earthy phosphates are thrown out and may be filtered off. If to the clear filtrate about one-third of magnesia mixture is added, the alkaline phosphates will be precipitated as ammonium-magnesium phosphate, termed triple-phosphate.

How does triple-phosphate form from ammoniacal urine in cystitis or putrid urine?

From the decomposition of urea into ammonium carbonate; this, with the magnesium phosphate, forms ammonium-magnesium phosphate.

What is the import of triple-phosphate in the urine, and what its appearance under the microscope?

Triple-phosphate, if formed in the bladder, may give rise to the formation of concretions, and as they result from ammoniacal urine, cystitis probably exists. Under the microscope, the crystals of triple-phosphate are prismatic and highly refractive, representing the form of coffin lids, after which they are named. (See Fig. 6, page 30.)

What is the deposit resulting from urine alkaline from fixed alkalies?

The earthy phosphates—*i. e.*, phosphates of calcium and magnesium—appearing under the microscope as granular masses. They have no tendency to form concretions.

How is the phosphoric acid of the urine quantitatively approximated?

By Teissier's method: Into a cylinder graduated in c.c., add 50 c.c. urine and 15 c.c. magnesia mixture; mix well and allow to settle for twenty-four hours. The total P_2O_5 will be precipitated as triple-

phosphate; each c.c., by volume, represents about 0.03 per cent. of phosphoric acid, or about double that amount of phosphates.

Describe the method for the volumetric determination of phosphoric acid in urine.

This is effected with a standardized solution of uranium acetate, each c.c. of which indicates 0.005 grm. P_2O_5. To 50 c.c. filtered urine, contained in a porcelain capsule or a flask, are added 5 c.c. acidulated solution of sodium acetate (sodium acetate 10, acid acetic dil. 10, water to 100). These are heated to the boiling-point, and, while boiling, the uranium acetate solution is gradually added from a burette. When the precipitate has formed, the fluid is tested from time to time by letting a few drops run into a solution of potassium ferrocyanide. As long as no change of color occurs, the process is not finished, and more uranium solution is added. When, on testing, a reddish-brown color begins to appear, all the phosphoric acid has been precipitated. To compute the result, multiply the number of c.c. uranium solution with 0.005, which gives the P_2O_5 in 50 c.c. urine, and double that amount the percentage.

What is the quantity of sulphuric acid eliminated in 24 hours, in which form, and where derived from?

About 2 grms. are eliminated in 24 hours, partly as sulphates of the alkalies, and a small portion as organic sulpho-acids. The sulphates are derived directly by ingestion and also by elaboration in the body of sulphur into its acid.

Under which conditions are the sulphates of the urine increased, and under which diminished?

Animal diet and exertion increase the sulphates in the urine; this is also the case in acute diseases, while in chronic affections of the kidneys, they are, as a rule, diminished. Ingestion of sulphur or sulphur compounds increases the amount of sulphates in the urine.

How are the sulphates in the urine shown?

By treating urine acidulated with a few drops of nitric acid with solution of barium chloride, which will give a precipitate of barium sulphate, insoluble in water or acids.

Describe the method for determining the sulphuric acid of the urine.

This is accomplished by volumetric analysis with a standard solution of barium chloride, each c.c. of which is equal to 0.01 grm. SO_3, in the following manner :—

100 c.c. urine are acidulated with 20 drops hydrochloric acid and heated to the boiling point, when the standard barium solution is gradually dropped in from a burette until fresh additions show but a slight precipitate. This is allowed to settle, and into the clear supernatant fluid one drop added. If no further precipitate results, it must be tested for an excess of the barium chloride with one drop of a sodium sulphate solution. If this shows an excess of barium, the determination has to be made with another 100 c.c. of the same urine, using less barium solution, and thus until neither the latter nor the sodium sulphate show an excess. The standard solution contains 30.5 grms. $BaCl_2$ in 1000 c.c., and each c.c. is equal to 0.01 SO_3. Thus if 13 c.c. $BaCl_2$ was used, the percentage of SO_3 was 0.13.

Abnormal Constituents of the Urine.

What are abnormal constituents of the urine?

Bodies which are not found in normal urine and owe their presence therein to abnormal, *i. e.*, pathological conditions.

Which are the principal abnormal constituents?

Biliary pigments, biliary acids, pus, blood corpuscles, hæmoglobin, albumin, glucose, oxalic acid as calcium oxalate, cystin, leucin and tyrosin, together with certain epithelial cells and casts.

Biliary Pigments and Acids in the Urine.

How is the appearance of the urine affected by the presence of biliary coloring matter?

The color is changed to a deep yellow, yellowish green, or even brownish green and brown; it froths freely on being shaken; the froth persists longer than usual, and is of a yellowish-green, or brownish color.

From which pathological conditions results the presence of biliary matters in the urine?

From icteroid conditions, which may be caused by either hepatogenous or hæmatogenous icterus. In the former, resulting from obstruction of the bile ducts, biliary coloring matter as well as biliary acids are present in the urine; in the latter, consequent upon the formation of bilirubin in the blood itself by the destruction therein of some of the red corpuscles, biliary coloring matter is found present but never biliary acids.

Which is the principal test for biliary coloring matter in the urine, and how applied?

Gmelin's test; on the addition of yellow nitric acid to some urine contained in a test tube, in a manner to cause the two to form different layers, there will be a play of colors from green, blue, violet, red, to yellow, if biliary coloring matter is present; as non-biliary urine sometimes gives off colors with this reaction, it should be closely noted that the colors appear in regular order, and that green should always form first.

What other convenient method or modification may be employed in its stead?

To mix a little urine in a test tube with an equal quantity of a saturated solution of sodium nitrate. Hold this slantingly and allow some concentrated sulphuric acid to run through the mixture and to the bottom of it, when even the smallest traces of biliary coloring matter may be detected, as above.

How may the biliary coloring matter be separated and shown?

By shaking the suspected urine with chloroform, separating the yellowish chloroform and applying the nitric acid test, as in Gmelin's method.

What other bile ingredients are at times found in icteroid urine?

Biliary acids are found in the urine of hepatogenous icterus, but never in that of the hæmatogenous variety. They are not always readily shown directly from the urine, and will then need a separation therefrom.

Which is the principal test for biliary acids, and how applied?

Pettenkofer's test. This consists in adding to some urine a small quantity of cane sugar and overlaying with this some sulphuric acid, contained in a test tube, when, if biliary acids are present, there will appear a purple zone at the junction of the two liquids. A better way is to dip a piece of filtering paper into the saccharated urine, allow this to dry and then touch it with sulphuric acid, when biliary acids will give a purple color. This test may also be made by evaporating some of the urine, with a minute quantity of cane sugar, to dryness in a porcelain dish. If a drop of sulphuric acid added thereto gives rise to purple coloration, biliary acids are present.

How is Oliver's test applied for biliary acids, and what is the composition of the reagent?

The reagent consists of 30 grains powdered meat peptone, 4 grains salicylic acid, 30 minims strong acetic acid, and water sufficient to make f ℥viij. If this is added to urine containing biliary acids a turbidity will arise in proportion to the amount of acids present. This may also be shown by overlaying the urine with the reagent, when the acids give rise to a turbid zone at the junction of the two liquids.

Pyuria.

What is pyuria and its import?

Pyuria is the condition characterized by the presence of pus in the urine. It points toward the existence of an acute or chronic inflammation in the urinary tract, or the communication therewith of abscesses. The sudden appearance of large quantities of pus in the urine would point to the latter condition. Inflammatory conditions of the bladder and renal pelvis are accompanied by more or less pus in the urine. When considerable pus is present in the bladder and the urine strongly alkaline, it will form a viscid jelly, which cannot be readily evacuated.

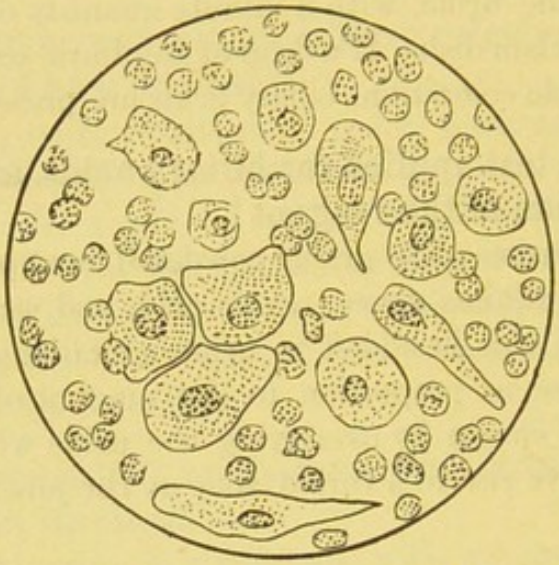
How may pus in the urine be differentiated from mucus?

Pus is turned gelatinous and ropy by caustic alkalies, while mucus is liquefied by them with white flakes.

Which is the most definite method for detecting pus in the urine?

In the acid urine pus appears as a heavy deposit, which, if inspected under the microscope, shows the pus cells as round, opaque, granular spheres, larger than the red corpuscles; on the addition of a drop of acetic acid to the slide, the granular contents and the cell membranes disappear, and the nuclei are readily seen (Fig. 8).

FIG. 8.



Pus Corpuscles and Epithelial Cells.

By what chemical test can pus in the urine be detected?

By Donné's test. This consists in separating the settled deposit by decantation and adding to the sediment a small piece of potassium hydrate, when, upon stirring, the pus will turn a clear and tough gelatinous mass.

Chyluria.

What constitutes chyluria, and what are its causes?

The presence of fat in the urine, which gives it more or less a milky or opalescent appearance; on standing and separation the fat particles rise to the surface. It may be of parasitic origin, as in tropical countries, owing to the presence of filaria sanguinis hominis or distoma hæmatobium in the blood, the lymph and the urine. The

non-parasitic form is met with occasionally in moderate climates, and its origin is attributed to degenerative changes of the kidneys.

How is the presence of fat in the urine demonstrated?

By shaking it with ether and allowing the separated ether to evaporate, when fat will be found if there has been any present. The shaking with ether will not clear up the urine entirely, as the ether will precipitate some of the albumins present in chylous urine. To thoroughly exhaust the fat in the urine, some potassium hydrate solution should be added beforehand.

Hæmaturia and Hæmoglobinuria.

What constitutes hæmaturia?

The presence of blood in the urine, as evident from the existence of intact red corpuscles therein.

In which morbid affections may blood be present in the urine?

If large quantities are present, it will most probably come from the urinary tract otherwise than the renal parenchyma. Thus inflammatory or hyperæmic conditions of the renal pelvis, ureters, also ulcerations, cancer of, or stone in the bladder, may give rise to a considerable amount of blood in the urine. If small quantities only are found present, it is more likely of renal origin, and if in addition to the blood corpuscles renal casts are found in the urine, its origin from the kidneys and the existence of parenchymatous nephritis is almost certain.

How does the blood in the urine differ in appearance according to the part of the urinary tract it is derived from?

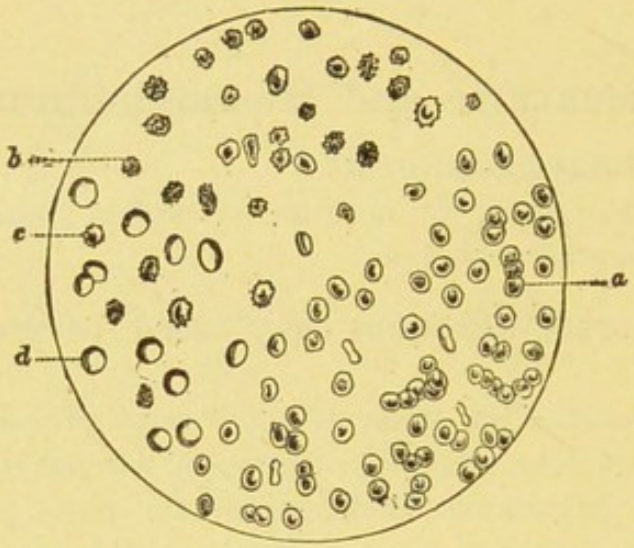
That from the renal parenchyma is well mixed with the urine and gives it a smoky appearance; if from the ureters, it is present in long semicircular clots and strings; the blood from the bladder and urethra is generally more in quantity, bright red, and settles in the urine as clots.

By which means is the existence of hæmaturia confirmed?

By the detection of the red blood corpuscles under the microscope.

To this end a small quantity of the sediment, after subsidence, is spread upon a microscopic slide with a drop of the urine, when, on inspection, the corpuscles will be brought to view in their characteristic biconcave spherical form, either single or grouped in rouleau form, or they may be crenated, as in dense urine; if they have imbibed much water, as in urine of low specific gravity, they may be swelled up and have lost their biconcavity, and they may have partly lost their contour and be partly destroyed, if the urine is ammoniacal (Fig. 9).

FIG. 9.



Blood Corpuscles.—*a*, with biconcave depressions; *b* and *c*, contracted and crenated; *d*, swollen.

What constitutes hæmoglobinuria?

The presence of hæmoglobin in the urine in a diffluent condition, and not in its corpuscular state.

How is hæmoglobinuria caused?

By a solution of the stroma of the red corpuscles in the blood leaving the diffusible hæmoglobin in solution therein which is secreted by the kidneys. The solution of the corpuscular element is noted in certain diseases, as in scurvy, typhus, pernicious malaria; also as the effect of certain poisons, such as hydrogen arsenide, phosphorus, carbolic acid, and by pressure; a periodical form is observed, the cause of which has as yet not been ascertained.

By which simple test may the presence of hæmoglobin in the urine be demonstrated?

By slightly acidulating some of the urine in a test tube with acetic acid and raising it to the boiling point. As hæmoglobin contains coagulable albumin, this will coagulate, and will, on subsiding, be found as a reddish sediment at the bottom, the soluble hæmoglobin having changed to insoluble hæmatin.

How can the hæmoglobin of the urine be demonstrated by the spectroscope?

By placing it in the light entering the prism of a spectroscope, it

FIG. 10.

Teichmann's Hæmin Crystals.

will give rise to two dark absorption bands in D and E of the spectrum, *i.e.*, in the yellow and in the green, the former being narrower, the latter broader.

Which is the chemical test for hæmoglobin?

Almen's test: Add a few drops fresh tincture of guaiac to the specimen of urine contained in a test tube; after agitation add a few drops of old spirit of turpentine or ozonic ether (ethereal solution of hydrogen peroxide); if hæmoglobin is present, the color will change to a distinct blue.

In which manner may the presence of hæmoglobin be most definitely and positively established?

By the production of Teichmann's crystals of hæmatin hydrochloride, often termed hæmin. To this end the slightly acidulated urine is boiled, and the coagulum filtered off; a small portion of this is dried with a gentle heat on a microscopic slide; to the dry residue a trace of common salt (NaCl) is added and well mixed with it, and, after adding one or two drops of glacial acetic acid on the mixture, this is covered with a cover glass, and heated over a lamp to the boiling point of the acid; after cooling, and on examination under the microscope, there will be found present numerous flat, rhombic prisms or tables, of a brown or blue color, which are hæmin, or better termed hæmatin hydrochloride (Fig. 10).

Albuminuria.

Which are the albumins found in the urine of albuminuria?

Principally serum-albumin, but also, and rarer, paraglobulin.

Under which conditions may albumin appear in the urine?

When the blood is surcharged with albumin, as after excessive ingestion of albuminoids; if the blood is much diluted, when œdematous exudations will take place; when the blood pressure in the kidneys is abnormally increased; also, if the chlorides of the blood are wanting, as well as if blood or pus is admixed with the urine.

Which pathological conditions are accompanied by temporary albuminuria?

The acute febrile affections, such as typhoid fever, diphtheria, pneumonia, etc., also the exanthematous diseases during their efflorescence.

When is the presence of albumin in the urine more persistent?

In the various inflammatory affections of the kidneys, variously termed Bright's disease of the kidneys, also in heart and respiratory diseases.

What are the general appearances of albuminous urine?

As a rule it is of pale color, low specific gravity, and when shaken maintains its froth for some time.

How is urine tested for albumin by Heller's test?

To a small quantity of urine in a test tube some nitric acid is added by allowing it to flow down the sides of the inclined test tube, so that the two fluids form separate layers; if albumin is present there will appear at their line of contact a white zone of coagulated albumin. This may be also the result of the presence of urates, but in this case the white zone is not as distinct and more toward the surface of the urine. If warmed, the urate cloud will disappear, but not the albumin.

In which way is the boiling test performed?

In this test the urine should be clear, and has to be filtered if turbid; if it is neutral or alkaline it has to be made slightly acid by the addition of a drop or more of acetic acid. If the upper part of the urine in the tube is now heated to the boiling point, it will be rendered turbid if albumin is present, and may readily be contrasted with the clear layer at the bottom. If the total volume is boiled, the entire albumin therein will be coagulated, and the flocculent coagulum may be separated by filtration. Should too much acetic acid have been added, the coagulation may have been prevented by the formation of acid-albumin. This can be demonstrated and corrected by the addition of a few drops of potassium ferrocyanide solution, when the coagulum will form at once.

How can the boiling test be utilized to approximate or comparatively estimate the quantity of albumin for clinical purposes?

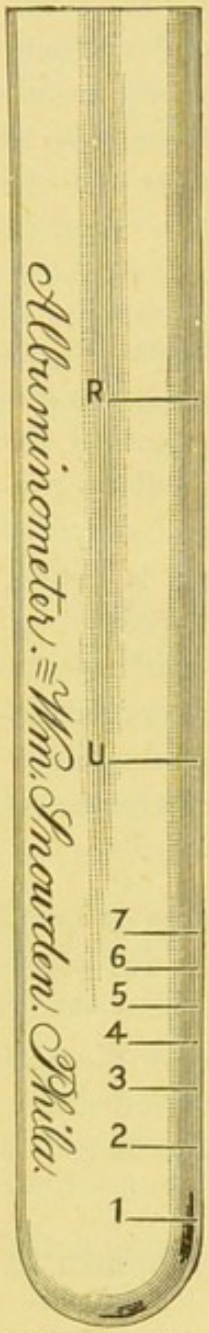
By allowing the coagulated albumin of the total urine in the test tube to subside for 24 hours and expressing the volume of the coagulum in comparison with the total urine boiled, as, for instance, ¼ or ½ albuminous layer.

How can picric acid be utilized for detecting albumin in urine?

If a concentrated solution of picric acid is added to urine there will be a coagulum formed if albumin is present. As this may, however, be caused also by alkaloids or peptones, this test can be used for the exclusion of albumin only, but if found present, it should be confirmed by either of the preceding tests.

In which way may the picric acid test be used for the quantitative estimation of albumin in the urine?

FIG. 11.



Esbach's Albuminometer.

By the use of Esbach's albuminometer (Fig. 11). This consists of a test tube of strong glass, marked near its middle and upper end respectively U and R, and near the bottom with small graduations marked respectively 1, 2, 3, 4, 5, 6, 7. It is used by filling up the tube with urine to the letter U and adding an acidulated picric acid solution to R. After 24 hours the coagulated albumin which has settled is read off in grammes of dry albumin per litre according to the small graduations to which it has settled. To obtain the percentage of dry albumin the respective figure is divided by 10. When the albumin is so abundant that the sediment is above 4, a more accurate result is obtained by first diluting the urine with one or two volumes of water and then multiplying the resulting figures by 2 or 3, as the case may be.

How is the acidulated solution of picric acid for this process prepared?

Dissolve 10 grammes picric acid and 20 grammes citric acid in 800 or 900 cubic centimeters of boiling water, which, on cooling, bring up with water to one litre (1000 c.c.).

Describe the volumetric estimation of albumin in the urine.

This consists of adding to 10 c.c. of urine 2 c.c. acetic acid, diluting with a little water, and then allowing Tanret's solution to drop in, drop by drop, from a suitable pipette, counting the number of drops so used; when the precipitate thus formed grows less, a drop of the urine is taken out and brought in contact with a few drops of 1 per cent. corrosive sublimate solution on a porcelain plate; if on mixing the two a red precipitate occurs, the reaction is complete, and for each drop so used, less 3 drops allowed for excess, 0.5 gramme of dry albumin per litre are present.

Give the composition of Tanret's reagent?

3.32 grammes potassium iodide, 1.35 grammes mercuric chloride, dissolved in 100 c.c. distilled water.

Is the presence of albumin in the urine alone sufficient for the diagnosis of Bright's disease?

No: as various other causes, already enumerated, may cause temporary or pseudo-albuminuria. If the amount of albumin, however, is large and persistently present, if the urine also contains casts and renal epithelium, the evidence points to the existence of renal disease.

Epithelial Cells in Urinary Sediments.

Are epithelial cells normally present in the urine, and, if present in larger quantities, what do they indicate?

Epithelial cells are always present in the urine, but according to their form, and the greater quantity present, they indicate pathological conditions of certain parts of the urinary tract.

What form have the epithelial cells of the uriniferous tubules?

They have a spherical, granular form, with faint outlines, but clearly defined nuclei. They may be present either singly or agglutinated as epithelial casts. The loops of Henle are lined with tessellated epithelium, and the straight tubules with the columnar variety (Fig. 12).

Which epithelial cells are derived from the renal pelvis?

Tessellated epithelium consisting of biconvex and caudate cells. The biconvex are generally as long again as they are broad. The caudate cells have an ovoid or club-shaped body ending in a fine point; their nuclei are well defined (Fig. 12).

Which epithelial cells belong to ureters and bladder?

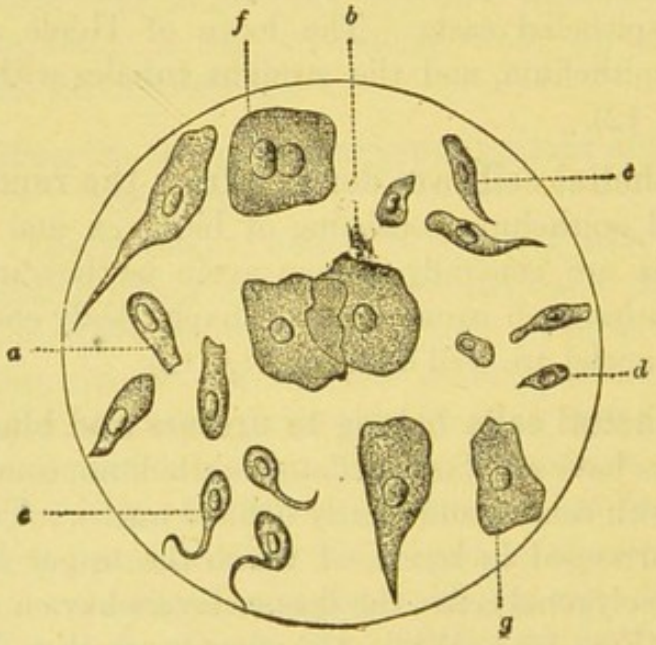
The ureters have regular tessellated epithelium, composed of polygonal cells, with central and clearly defined nuclei. The bladder has epithelium arranged in layers, of which the upper layer is formed by flattened polygonal cells; the deeper layers have a more spherical appearance (Fig. 13). While the female urethra has the same epithelium, that of the male urethra resembles more the renal

epithelium. Renal epithelium is subject to pathological changes, such as fatty or amyloid degenerations, recognized by the microscope and the amyloid reactions.

FIG. 12.

Renal epithelial cells and epithelial cells from renal pelvis.

FIG. 13.



Epithelial cells. *a*, from male urethra; *b*, from vagina; *d*, from Cowper's glands; *e*, from Littré's glands; *f*, from female urethra; *g*, from bladder.

Tube Casts in the Urine.

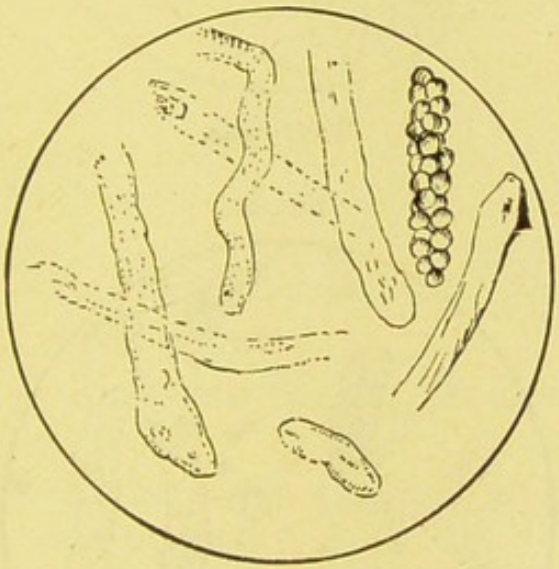
Under what conditions are tube casts found in the urine, and how recognized?

In acute and chronic renal affections, tube casts may be recognized in the urinary sediment by means of the microscope. The sediment should be allowed to settle, and a specimen removed with a pipette for microscopic inspection. To make them more distinct a drop of Lugol's solution or aniline red may be added.

Which are the principal forms of tube casts?

The epithelial casts, composed of coherent epithelial cells of the

FIG. 14.



Hyaline casts, also one epithelial cast.

tubes of Bellini; they are generally pale and transparent, and around them small round cells and nuclei may be recognized (Fig. 14).

The hyaline casts appear as pale, transparent cylinders of various sizes and configurations, and of very delicate outlines; they are also termed mucous casts (Fig. 14).

A modification of these with distinct outlines, slightly yellow color and waxy lustre are termed waxy casts (Fig. 15).

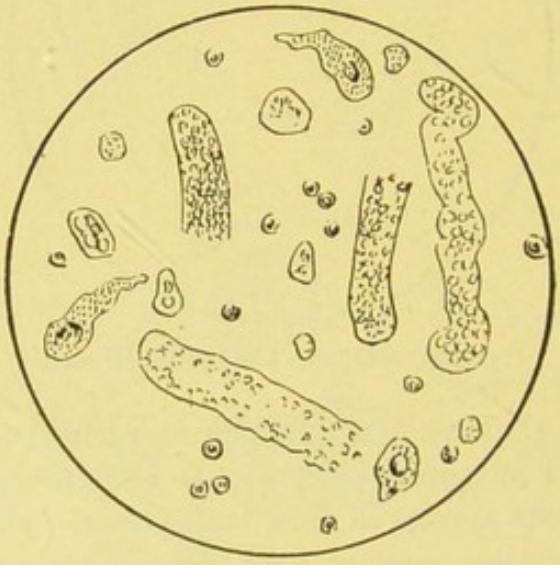
Granular casts (fibrinous casts), resemble the hyaline casts but have granular contents consisting of cells which have suffered granular change, giving them a darker appearance than the former. They

may contain, also, oxalates, blood and pus corpuscles, fat globules and epithelial cells (Fig. 16).

FIG. 15.

Waxy casts.

FIG. 16.



Granular casts with fatty globules; also, blood and pus corpuscles, and epithelial cells.

Have the different tube casts always positive value for differentiating the various renal affections?

Not always, as in acute and chronic nephritis, as well as in amyloid degeneration, all the varieties may be present at one time.

When will they be of such diagnostic value?

When one variety only appears. Thus, if epithelial casts alone persist for several days, they point to the existence of a desquamative nephritis with favorable prognosis, while the simultaneous presence of pus corpuscles renders the prognosis less favorable.

In which cases of nephritis are hyaline and granular casts found?

In the severer cases with a disposition to chronicity, as indicated by their number and persistence. If they contain numerous fat granules and fat globules the diagnosis of fatty degeneration is justifiable, especially if accompanied by fatty degenerated renal epithelium both in the casts and separately.

What would indicate the existence of the contracted kidney?

If the tube casts grow thinner and the epithelial elements appear contracted.

How may amyloid degeneration be indicated?

The tube casts may here appear the same as in the other forms of nephritis, but there will be found besides the fatty degenerated epithelial cells, also such as have undergone amyloid degeneration, recognized by being colored red by methyl-violet, the others turning blue.

What would blood casts show?

That there is renal hemorrhage, the casts being coagulated blood with imbedded corpuscles.

Which are some of the other abnormal sediments of the urine of clinical interest?

Cystin, leucin and tyrosin, calcium oxalate and micro-organisms.

When and in what form is cystin found in the urine?

Cystin is found occasionally, but very seldom, as urinary concretion, also in urinary deposits; it is insoluble in water, not dissolved by heating, but soluble in alkaline hydrates, also mineral and oxalic acids. Under the microscope it appears in the form of colorless, shining, six-sided plates or prisms (Fig. 17).

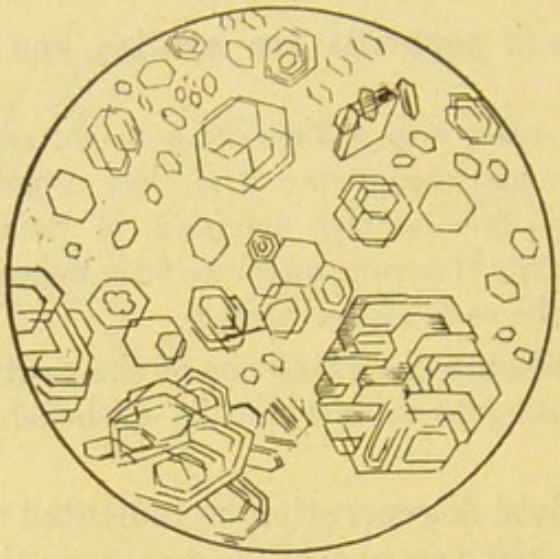
When and how do leucin and tyrosin occur in urinary sediments?

They occur frequently in the urinary sediments in acute yellow atrophy of the liver, also in phosphorus poisoning, and point to an incomplete oxidation of the albuminoids.

What is the microscopical appearance of leucin?

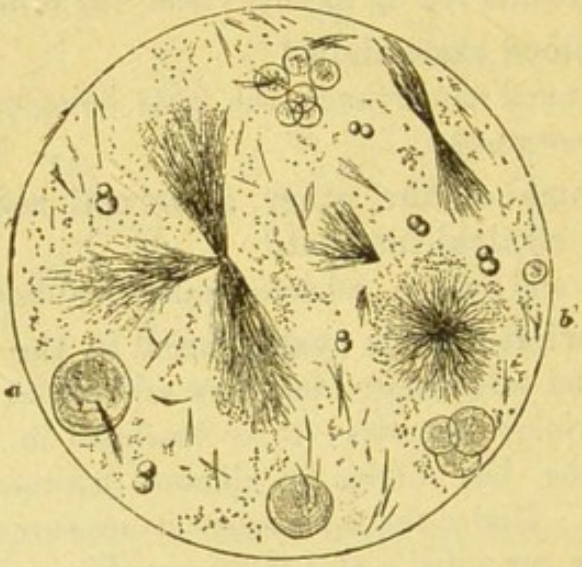
Yellow-colored spheres, at times concentrically striated with protruding delicate points or spines (Fig. 18).

FIG. 17.



Crystals of cystin (*after Ultzmann*).

FIG. 18.



(*a*) Leucin, spheres; (*b*) Tyrosin, needles and sheaves.

How does tyrosin appear under the microscope?

Tyrosin, which appears generally associated with leucin in urinary sediments, has the form of silky, white, microscopic needles which are arranged in sheaves or stellate form (Fig. 18).

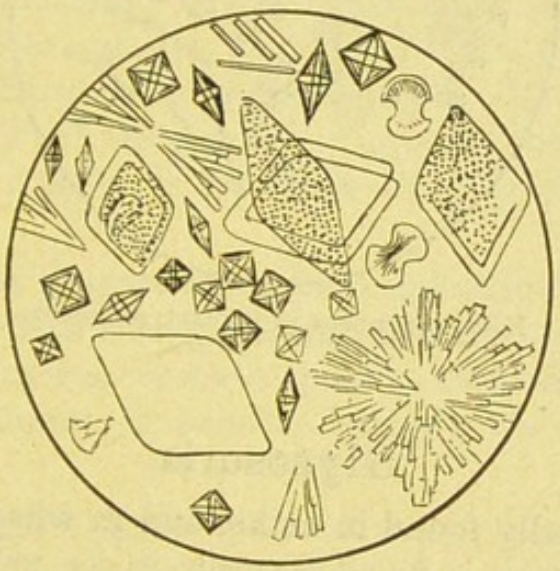
In what compound is oxalic acid occasionally present in the urine?

As calcium oxalate, which is frequently found in the urine, but becomes of importance only when present in larger quantities and persistently, when it gives rise to oxaluria, signifying a retarded metabolism or suboxidation.

Describe the form of crystals of calcium oxalate in urinary sediments.

It forms minute, transparent, brilliant octahedra presenting somewhat the shape of a square envelope. At times the crystals assume

FIG. 19.



Calcium oxalate in octahedral (envelope) crystals; also in dumb-bells; some larger crystals of uric acid.

the form of dumb-bells; to detect them a high power objective should be employed (Fig. 19).

Under which conditions are micro-organisms found in the urine?

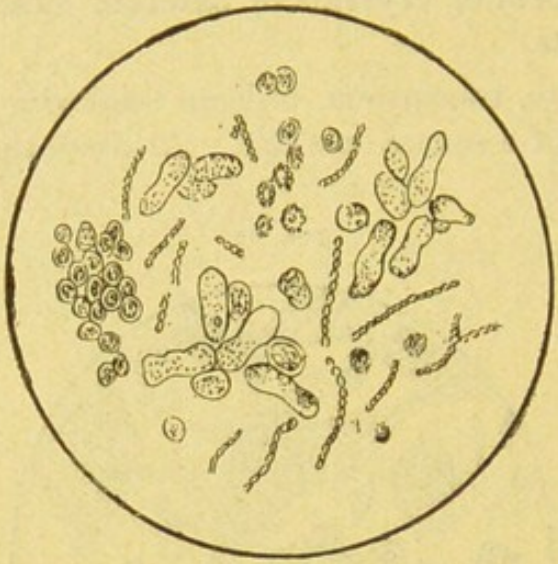
As a rule, micro-organisms are found in the urine only after it has been exposed to the air for some time, but they may be carried into the bladder by catheters or sounds, and there set up ammoniacal decomposition of the urea, giving rise to cystitis. Of greater importance are those which are derived from the blood, as in

infectious diseases, such as scarlatina, typhoid and malarial fevers and renal diseases.

Which are the principal micro-organisms found in the urine?

Mould (penicillium), yeast cells, sarcinæ (in the urine they are smaller than of the stomach), vibriones, bacteria, bacilli, cocci, etc. (Fig. 20).

FIG. 20.



Micro-organisms of urinary sediment.

Glycosuria.

Is sugar normally found in urine, and in what quantities?

Sugar as glucose is found normally in the urine in very small quantities, not more than 0.1 gramme in 24 hours.

If found in larger quantities and persistently, what pathological condition is present?

Glycosuria or diabetes mellitus; it may, however, be found in the urine in larger quantities in cerebral and nervous affections; also temporarily after anæsthesia from chloroform, ether, etc., as well as after copious ingestion of sugar.

What is the appearance and physical condition of the urine of glycosuria?

It is much increased in quantity, and may reach one or two gallons in the 24 hours; it is of pale or pale yellow color, often

slightly greenish, clear, as a rule, but may contain sediments of urates or oxalates; it has a high specific gravity, generally between 1030 to 1040 and even more; the urea eliminated is abnormally increased.

Which foods increase and which diminish the amount of glucose in the urine?

Starchy and saccharine food increases, and animal diet decreases the amount.

In testing urine containing albumin for glucose, what has to be done first?

The albumin has first to be separated by boiling and filtration.

Describe Moore's test for the detection of glucose in urine.

In a long test tube mix about one part of urine with half its volume of liquor potassæ. Heat the mixed liquids in its upper half until active ebullition ensues, when, if glucose is present, the upper part will turn dark yellow to reddish brown. If the whole volume is thus treated, it will change color in a similar manner, and if then some nitric acid is added, it is decolorized and an odor of burnt sugar is given off.

What effect has glucose on certain metallic oxides if heated together in a strong alkaline fluid?

The metallic oxides, especially those of bismuth and copper, are reduced; the former to metallic bismuth, the cupric oxide to cuprous oxide.

Describe Bœttger's test.

Bœttger's, or the alkaline bismuth test, is performed by mixing equal parts of urine and liquor potassæ and adding a small quantity of bismuth subnitrate; boil for a minute or two, and if glucose is present, the bismuth will turn gray, brown or black, owing to the reduction to its metallic state.

What are the advantages and disadvantages of this test?

It has the advantage that uric acid, urates and kreatinin do not affect it, but albumin or sulphides present in the urine, produce similar effects as glucose.

What is Nylander's modification of the alkaline bismuth test?

This consists in the use of a single alkaline bismuth solution

composed of bismuth subnitrate 2 grammes, Rochelle salt 4 grammes, solution of sodium hydrate (8 per cent.) 100 grammes. To 10 c.c. urine 1 c.c. of this solution is added and the two boiled together; if glucose is present, it turns brown or black.

Upon what reactions do the alkaline cupric tests depend?

First, that on adding a few drops of cupric sulphate solution to liquor potassæ a greenish-blue precipitate of cupric hydrate is formed.

$$CuSO_4 + 2KHO = Cu(OH)_2 + K_2SO_4.$$

Second, the cupric hydrate on boiling splits up into cupric oxide and water, the former appearing as a black precipitate.

$$Cu(OH)_2 = CuO + H_2O.$$

Third, the black precipitate does not form in the presence of certain organic matter and excess of alkaline hydrate, but remains in solution, having a deep blue color. When this alkaline cupric oxide solution is boiled in the presence of glucose, the latter takes away oxygen from the cupric oxide, leaving yellow insoluble cuprous oxide.

$$2CuO - O = Cu_2O.$$

Trommer's test, what is it, and how performed?

To some urine in a test tube add one-half or one-third volume of liquor potassæ and a few drops of a 10 per cent. solution of cupric sulphate. If this is heated in its upper half to the boiling point there will be a reddish-yellow turbidity, caused by the separation of cuprous oxide, if glucose is present. The two strata will give a distinct difference in appearance.

How can this test be improved by the addition of glycerin?

The addition of a few drops of glycerin will prevent the precipitation of the black cupric oxide and admit of the test solution being first tested by boiling. If to some liquor potassæ in a test tube a few drops of cupric sulphate solution are added, and also a few drops of glycerin, a clear, deep blue liquid will result. This is brought to the boiling point, and after removal from the flame a little of the urine is added; if glucose is present, the characteristic reddish-yellow cuprous oxide will form within a minute or two. If this does not take place after the first addition of the urine it should be

brought to the boiling point again and a little more urine added as before.

Which is the principal alkaline cupric test, and how applied?

The Fehling's test, an alkaline solution of cupric oxide, which is held in solution by Rochelle salt (sodium-potassium tartrate), (see formula under quantitative tests for sugar). This solution is diluted with 3 to 4 volumes of water, heated to the boiling point, when a little urine is added. If sugar is present, a yellowish-red precipitate of cuprous oxide will form; if indistinct at first, boil again and add more urine. The boiling before adding the urine is necessary to establish the quality of the solution and to show if it does not decompose spontaneously at the boiling point.

What is the most positive evidence of glucose in urine, and how shown?

The alcoholic fermentation on addition of yeast. This may be shown by adding some yeast to a bottle filled with the suspected urine. Invert the bottle in a vessel containing the same urine; if inside of 24 hours part of the urine is displaced by carbon dioxide, glucose was present. This may be further verified by testing the urine for alcohol by adding to the fermented urine a few drops of potassium dichromate solution and sulphuric acid; if, on warming, a green color appears, alcohol was formed and glucose positively present in the urine.

How may the presence of glucose in urine be shown by phenyl-hydrazine?

To equal parts of urine and liquor potassæ add a few drops of phenyl-hydrazine, and heat to boiling. In the presence of glucose an intense yellow or orange color develops; on addition of an excess of acetic acid, yellow crystals are precipitated.

Which are some other very delicate tests for glucose in urine?

The tests with menthol, thymol or alpha-naphthol. These are applied by using their alcoholic solutions (1 to 7 alcohol), and mixing a few drops thereof with the urine. To the mixture, in a test tube, add some sulphuric acid in a manner that they do not mix, when, if glucose is present, there will be a red color at the line of contact with

thymol or menthol, or violet with greenish borders if alpha-naphthol was employed.

How may picric acid be used for the detection of glucose in urine?

By its conversion, on boiling with an alkaline hydrate, into dark, reddish-brown picramic acid if glucose is present. Normal urine produces a similar reaction, but not of the deep reddish-brown color as when glucose is present.

By what ready method may the fermentation test for glucose in urine be used quantitatively?

By the differential density method of Roberts. This is conducted by accurately taking the specific gravity of the urine to be examined, the temperature of the urine being noted. With 4 ozs. of this urine in a 12 oz. flask or bottle, mix a small piece (about ¼ cake) of compressed yeast; after setting aside for 24 hours in a warm place, fermentation is completed. The urine is then cooled to the temperature of the former specimen and its specific gravity also accurately ascertained. The number of degrees of specific gravity lost by fermentation corresponds to the number of grains of glucose in the ounce of urine. The percentage may be obtained by multiplying the number of degrees lost by 0.22.

How is the quantity of glucose in urine ascertained by polarization?

By filling the container of a polarization apparatus with the filtered urine, free from albumin. The urine must be almost colorless, and care must be had that no air bubbles are in the tube. The analyzer is then moved to accurately correct the difference in color in the two halves of the visual field, and the angle through which it was moved is read off by means of the scale and vernier. The amount of glucose is computed by the following formula: $p = \frac{a}{+ 56\, l}$, in which p stands for the quantity of glucose in grammes in 1 c.c. of the urine, a the angle read off, + 56 the specific rotation for glucose, and l the length of the containing tube expressed in decimeters. Thus, if the angle read off were 4.5°, the tube 1 decimeter long, it would be $\frac{4.5^{\circ}}{56 \times 1} = 0.080$ glucose in 1 c.c., or 8.0 in 100 c.c. Various instru-

ments for this purpose are in the market, some admitting the reading off directly of the percentage of glucose.

Give the formula for making Fehling's solution.

As Fehling's solution does not keep, it should not be kept on hand for any length of time, but should be made as two separate solutions, of which equal amounts by measure are mixed together at the time when wanted to form the complete test solution.

No. 1.	℞.	Cupric sulphate (pure, not effloresced, and free from water of crystallization),	34.64 grammes
		Water, q. s. to	500 c.c.
No. 2.	℞.	Rochelle salt (crystallized),	173 grammes
		Solution of sodium hydrate, sp. gr. 1.34,	100 c.c.
		Water, q. s.	500 c.c.

For use, mix equal volumes of No. 1 and No. 2 as needed.

Describe the method for the quantitative determination of glucose in urine by Fehling's volumetric process.

Place into a capsule, beaker or flask 10 c.c. of Fehling's solution diluted with 40 c.c. of water. Heat to the boiling point, and let gradually run into it from a burette a mixture of one part of urine and nine of water, stirring the mixture, and continue thus until the blue color of the test solution has entirely disappeared. The diluted urine is to be added in small quantities only, and the test solution must be raised to the boiling point after each addition, when it is left to subside for a few seconds to be able to view the supernatant fluid with transmitted light. Toward the last only a drop or two at the time should be added, as the clear shade should have a rather yellowish tint. This process should be repeated several times until the amount of diluted urine proves the smallest quantity effecting complete reduction.

How is the quantity of glucose computed from this?

As 10 c.c. Fehling's solution are reduced by 0.05 gramme glucose, that amount of urine which is present in the dilution which has reduced the 10 c.c. of the Fehling's solution contained 0.05 gramme glucose. Thus, if 16 c.c. diluted urine (1 in 10) were used, 1.6 urine contained 0.05 gramme glucose. To obtain the percentage the fol-

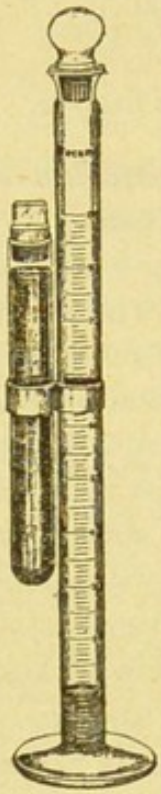
lowing proportion will answer: 1.6 : 0.05 = 100 : x; x = 3. 1 per cent.

What is Johnson's picric-acid test for the quantitative determination of glucose in urine?

It consists of converting picric acid in the presence of potassium hydrate and glucose into reddish-brown picramic acid; the intensity of the color of the latter being proportionate to the amount of glucose present; the color of the picramic acid formed is then compared with that of a standardized solution of ferric acetate, and the amount of glucose ascertained by the dilution required.

How is this test performed?

FIG. 21.



Take of urine f℥j, liquor potassæ f℥ss, solution of picric acid (gr. 5.3 to f℥j) ♏xl, water q. s. ad f℥iv into a test tube and boil for sixty seconds; cool the mixture and bring it up to the original volume (f℥iv). Of this pour 10 c.c. into a 100 c.c. graduated cylinder, which has attached to it a test tube of equal diameter and containing the standard fluid (Fig. 21); dilute the boiled mixture with distilled water to equal in color the standard liquid, and for each 10 c.c. it has been distilled up to, count 1 grain of glucose in the fluidounce of urine tested.

How may the measurement of the ingredients be simplified?

By taking 5 c.c. of each, urine, potassium hydrate solution (sp. gr. 1.036), solution of picric acid (gr. 3.5 to f℥j), and water.

Give the formula for the standard ferric acetate solution.

℞. Liquor. ferri chloridi, U. S. P., f℥j
Ammonii carb., ℥j
Acidi acetici, f℥v
Aquæ destillatæ, q. s. ad f℥ iijss. M.

Is the above formula for the standard fluid reliable, and what should be done to make it so?

It is not reliable, and to make it so it should be standardized by

comparing it with urine, to each fluidounce of which 1 grain of crystallized glucose has been added. To this the KOH, picric acid and water, is added, as above, and boiled for sixty seconds, when, after boiling, the standard iron solution is either diluted or made denser in color by addition of some liquor ferri chloridi to closely correspond in color. By doing so, not only is the error of variable strength of the ferric chloride solution corrected, but the error created by the normal presence of kreatinin is also overcome. When thus executed, this method is as reliable as any other for clinical purposes.

Lead and Mercury in the Urine.

Is lead readily detected in urine after lead-poisoning, and is a failure to find it proof of its absence from the system?

Lead is not always found in the urine after lead-poisoning, and is not always readily detected. Before testing for lead in the urine, iodide of potassium should be administered in full doses for a few days.

Give a method for detecting the presence of lead in the urine.

About 30 to 50 ounces of urine are brought to the boiling point in a porcelain evaporating dish, and while boiling, nitric acid (free from lead) is added in small quantities until on addition no further effervescence results; evaporation is then continued to dryness, the residue is carbonized in a porcelain crucible with the addition of nitric acid. The residuary mass, after combustion, is boiled out with nitric acid; after boiling it is diluted with water, the mixture filtered and the filtrate evaporated to dryness. The dry residue is dissolved in water slightly acidulated with HNO_3, and a current of hydrogen sulphide is allowed to pass through it for some time. If a brownish-black precipitate results, the presence of lead should be confirmed by testing another portion of the clear fluid with potassium iodide or neutral potassium chromate; with either of these it must give a yellow precipitate if lead is present. As the latter are not so sensitive as H_2S, it may be necessary to separate the precipitated lead sulphide, redissolve by gradual addition of HNO_3, evaporate excess of latter, and test after dilution and filtration as before. The

lead iodide so derived will be seen under the microscope as six-sided plates. If the lead sulphide precipitated is sufficient, it may be reduced by the blow-pipe flame to a malleable particle of lead.

When may the detection of mercury in the urine be of diagnostic value?

In cases of protracted mercurialization, as in syphilis, etc.

Describe a ready method for detecting mercury in urine.

300–500 c.c. of urine are acidulated with hydrochloric acid and evaporated to about one-fourth of its volume, allowed to cool and then filtered. In the filtrate boil for a little while a slip of pure, bright copper foil; after boiling sufficiently, take it out, wash off in distilled water and dry it between bibulous paper; then roll up and put into an open glass tube; heat at the place where the copper is to redness, when the mercury will be driven to a cooler portion of the tube, to be recognized by the shape and brilliancy of its globules under the microscope; also, by converting it into red mercuric iodide, when into the hot portion of the tube a minimal fragment of iodine is introduced and its vapors are allowed to flow over the sublimed mercury.

What other very delicate method may be employed for detecting mercury in the urine?

The method of Ludwig is the one best adapted for detecting mercury in the urine. It is conducted by acidulating 200–500 c.c. urine with hydrochloric acid, warming the mixture to 50°–60° C., and adding 5 grammes of pure zinc dust (to be had of dealers in chemicals). The mixture is stirred for some time while warm, and then the zinc is allowed to subside, when the supernatant fluid is separated by decantation; the metallic sediment is well washed with distilled water and dried in a vapor bath after filtration. The mercury, having united with the zinc dust to form an amalgam, can now be driven off; this is done best in an open tube in which the zinc dust is secured by two loose asbestos plugs. The part of the tube containing the zinc is now heated and the mercury driven up to the upper and cooler portion of the tube. That portion of the tube is broken off, and the sublimed mercury is recognized by converting it into red mercuric iodide by passing a vapor of iodine over it.

DIETRICH'S TABLE (*Weight of one C. C. Nitrogen in Milligrams*).

Temp. C.	Barometric Pressure, in Millimeters.												
	720	722	724	726	728	730	732	734	736	738	740	742	744
10°	1.1338	1.1370	1.1402	1.1434	1.1466	1.1498	1.1529	1.1561	1.1593	1.1625	1.1657	1.1689	1.1721
11°	1.1288	1.1320	1.1352	1.1384	1.1415	1.1447	1.1479	1.1511	1.1542	1.1574	1.1606	1.1638	1.1670
12°	1.1237	1 1269	1.1301	1 1333	1.1364	1.1396	1.1428	1.1459	1.1491	1 1523	1.1554	1.1586	1.1618
13°	1.1187	1.1219	1.1251	1.1282	1.1314	1.1345	1.1377	1.1409	1.1440	1.1472	1.1503	1.1535	1.1566
14°	1.1136	1.1168	1.1200	1.1231	1.1263	1.1294	1.1326	1.1357	1.1389	1.1420	1.1452	1.1483	1.1515
15°	1.1085	1.1117	1.1149	1.1180	1.1211	1.1243	1.1274	1.1305	1.1337	1.1368	1.1399	1.1431	1.1462
16°	1.1034	1.1066	1.1097	1.1128	1.1160	1.1191	1.1122	1.1253	1.1285	1.1316	1.1347	1.1378	1.1409
17°	1.0983	1.1014	1.1045	1.1076	1.1107	1.1138	1.1170	1.1201	1.1232	1.1263	1.1294	1.1325	1.1356
18°	1.0930	1.0961	1.0992	1.1023	1.1054	1.1085	1.1117	1.1148	1.1179	1.1209	1.1241	1.1272	1.1303
19°	1.0877	1 0908	1.0939	1.0970	1.1001	1.1032	1.1063	1.1094	1.1125	1.1156	1.1187	1.1218	1.1248
20°	1.0825	1.0855	1.0886	1 0917	1.0948	1.0979	1.1009	1.1040	1.1071	1.1102	1.1133	1.1164	1.1194
21°	1.0771	1.0802	1.0832	1.0863	1.0894	1.0924	1.0955	1.0986	1.1017	1.1047	1.1078	1.1109	1.1139
22°	1.0717	1.0747	1.0778	1.0808	1.0839	1.0870	1.0900	1.0931	1.0961	1.0992	1.1023	1.1053	1.1084
23°	1.0662	1.0692	1.0723	1.0753	1.0784	1.0814	1.0845	1.0875	1.0906	1.0936	1.0967	1.0997	1.1028
24°	1.0606	1.0636	1.0667	1.0697	1.0728	1.0758	1.0789	1.0819	1.0849	1.0880	1.0910	1.0940	1.0971
25°	1.0550	1.0580	1.0610	1.0641	1.0671	1.0701	1.0732	1.0762	1.0792	1.0823	1.0853	1.0883	1.0913

	746	748	750	752	754	756	758	760	762	764	766	768	770
10°	1.1753	1.1785	1.1817	1.1848	1.1880	1.1912	1.1944	1.1976	1.2008	1.2040	1.2072	1.2104	1.2136
11°	1.1701	1.1733	1.1765	1.1717	1.1829	1.1860	1.1892	1.1924	1.1956	1.1988	1.2019	1.2051	1.2083
12°	1.1649	1.1681	1.1713	1.1744	1.1776	1.1808	1.1839	1.1871	1.1903	1.1934	1.1966	1.1998	1.2029
13°	1.1598	1.1630	1.1661	1.1693	1.1724	1 1756	1.1787	1.1819	1.1851	1.1882	1.1914	1.1945	1.1977
14°	1.1546	1.1577	1 1609	1.1640	1.1672	1.1703	1.1735	1.1766	1.1798	1.1829	1.1861	1.1892	1.1923
15°	1.1493	1.1525	1.1556	1.1587	1.1619	1.1650	1.1681	1.1713	1.1744	1.1775	1.1807	1.1838	1.1869
16°	1.1441	1 1472	1.1503	1.1534	1.1566	1.1597	1.1628	1.1659	1.1691	1.1722	1.1753	1.1784	1.1816
17°	1 1397	1.1419	1.1450	1.1481	1.1512	1.1543	1.1574	1.1605	1.1636	1.1667	1.1699	1.1730	1.1761
18°	1.1334	1.1365	1.1396	1.1427	1.1458	1 1489	1.1520	1.1551	1.1582	1.1613	1.1644	1.1675	1.1706
19°	1.1279	1.1310	1.1341	1.1372	1.1403	1.1434	1.1465	1.1496	1.1527	1.1558	1.1589	1.1620	1.1650
20°	1.1225	1.1256	1.1287	1.1318	1.1348	1.1379	1.1410	1.1441	1.1472	1.1502	1.1533	1.1564	1.1595
21°	1.1170	1.1201	1.1231	1.1262	1.1293	1.1324	1.1354	1.1385	1.1416	1.1446	1.1477	1.1508	1.1539
22°	1.1115	1.1145	1.1176	1.1206	1.1237	1.1268	1.1298	1.1329	1.1359	1 1390	1.1421	1.1451	1.1482
23°	1.1058	1.1089	1.1119	1.1150	1.1180	1.1211	1.1241	1.1272	1.1302	1.1333	1.1363	1.1394	1.1424
24°	1.1001	1.1032	1.1062	1.1092	1.1123	1.1153	1 1184	1.1214	1.1244	1.1275	1.1305	1.1336	1.1366
25°	1.0944	1.0974	1.1004	1.1035	1.1065	1.1095	1.1126	1.1156	1.1186	1.1216	1.1247	1.1277	1.1307

INDEX.

ABNORMAL constituents, 37
Albumin, approximation of, 45
boiling test for, 45
volumetric estimation of, 46
Albuminous urine, appearance of, 44
Albuminuria, 44
causes of, 44
Alkaline cupric tests, 56
Alpha-naphthol test for sugar, 57
Ammonium urate, 30
Appearance of urine, 18
Azotimetric determination of urea, 26

BILIARY matter, how resulting, 38
pigments and acids, 37
Biuret reaction, 24
Blood corpuscles, 42
Bœttger's test, 55
Bright's disease, diagnosis of, 47

CALCIUM oxalate, 53
Casts, value for diagnosis, 50
Chlorides, 32
demonstration and determination of, 33
pathological import, 33
Chyluria, 40
Collection of specimen, 17
Color, 18
Computation for urea by azotimetric method, 27
Cystin, 51

DIETRICH'S table, 63

EARTHY phosphates, 35
Epithelial casts, 49
cells from renal pelvis, 47
in urinary sediments, 47
of ureters and bladder, 47
of uriniferous tubules, 47
Esbach's method for determination of albumin, 46

FAT in urine, how demonstrated, 44
Fehling's solution, formula, 59
Fehling's test, 57
Fehling's test for quantitative determination of sugar, 59
Fermentation test for determination of sugar, 58
Fermentation test for sugar, 57
Fowler's method for determination of urea, 24

GLYCERIN-cupric test for sugar, 56
Glycosuria, 54
Gmelin's test, 38
Granular casts, 49

HÆMATURIA, 41
cause of, 41
Hæmoglobin, chemical test for, 43
spectroscopic test for, 43
test for, 43
Hæmoglobinuria, 41
cause of, 42
Heller's test, 45
Hippuric acid, 31
Hyaline casts, 49

INDICAN, 18

JOHNSON'S picric acid test, quantitative, 60

KNOP'S solution, 27
Kreatinin, 31

LEAD, detection of, in urine, 61
Leucin, 51
Liebig's method for determination of urea, 24
Lyon's apparatus, 28

MENTHOL test for sugar, 57
Mercury, detection of, by Ludwig's method, 62
in urine, 62
Microörganisms, 53
Moore's test for sugar, 55.
Mucous casts, 49
Murexide test, 30

NORMAL constituents, 22
inorganic constituents, 32
organic constituents, 22
Nylander's test, 55

ODOR, 19
Oliver's test, 39
Oxalic acid, 53

PETTENKOFER'S test, 39
Phenyl-hydrazine test for sugar, 57
Phosphoric acid, 34
quantitative determination of, 35
Picric acid, acidulated solution of, 46
test for albumin, 45
sugar, 58
Polarization for determination of sugar, 58
Pus, 39
detection of, 40
Pyuria, 39

QUANTITY voided, 17

REACTION, 20

SARKIN, 32
Specific gravity, 21
Sulphuric acid, 36
determination of, 37

TANRET'S reagent, 47
Teichmann's crystals, 44
Thymol test for sugar, 57
Triple-phosphate, 35
Trommer's test, 56
Tube casts in urine, 49
Tyrosin, 51

UREA, 22
oxalate and nitrate, 23
Uric acid, 28
determination of, 30
microscopic appearance of, 28
salts of, 29
Urobilin, 18

WAXY casts, 49

XANTHIN, 32

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