Diseases of the chest, throat, and nasal cavities / by E. Fletcher Ingals.

Contributors

Ingals, E. Fletcher 1848-1918.
Augustus Long Health Sciences Library

Publication/Creation

New York : W. Wood & company, 1894.

Persistent URL

https://wellcomecollection.org/works/mertxub4

License and attribution

This material has been provided by This material has been provided by the Augustus C. Long Health Sciences Library at Columbia University and Columbia University Libraries/Information Services, through the Medical Heritage Library. The original may be consulted at the the Augustus C. Long Health Sciences Library at Columbia University and Columbia University. where the originals may be consulted.
This work has been identified as being free of known restrictions under copyright law, including all related and neighbouring rights and is being made available under the Creative Commons, Public Domain Mark.

You can copy, modify, distribute and perform the work, even for commercial purposes, without asking permission.

wellcome collection

Wellcome Collection
183 Euston Road
London NW1 2BE UK
T +44 (0)20 7611 8722
E library@wellcomecollection.org
https://wellcomecollection.org

Unable to display this page

RC 941 In4
1894

Columbia University
in the City of New York
College of Physicians and Surgeons
Library

Digitized by the Internet Archive
in 2010 with funding from
Open Knowledge Commons

http://www.archive.org/details/diseasesofchestt00inga

DISEASES

OF THE

CHEST, THROAT

AND

NASAL CAVITIES

INCLUDING

Physical Diagnosis and Diseases of the Lungs, Heart, and Aorta, Laryngology and Diseases of the Pharynx, Larynx, Nose, Thyroid Gland, and Œsophagus;

COLUMBIA UNIVERSITY MEDICAL LIBRARY

BY

E. FLETCHER INGALS, A.M., M.D.

PROFESSOR OF LARYNGOLOGY AND PRACTICE OF MEDICINE, RUSH MEDICAL COLLEGE; PROFESSOR OF DISEASES OF THE THROAT AND CHEST, NORTHWESTERN UNIVERSITY WOMAN'S MEDICAL SCHOOL; PROFESSOR OF LARYNGOLOGY AND RHINOLOGY, CHICAGO POLYCLINIC; LARYNGOLOGIST TO THE ST. JOSEPH'S HOSPITAL AND TO THE PRESBYTERIAN HOSPITAL, ETC.; FELLOW OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION AND AMERICAN CLIMATOLOGICAL ASSOCIATION; MEMBER OF THE AMERICAN MEDICAL ASSOCIATION, ILLINOIS STATE MEDICAL SOCIETY, CHICAGO MEDICAL SOCIETY, CHICAGO PATHOLOGICAL SOCIETY, ETC., ETC.

THIRD EDITION, REVISED.

WITH TWO HUNDRED AND FORTY ILLUSTRATIONS.

NEW YORK
WILLIAM WOOD & COMPANY
1894

COLUMBIA
UNIVERSITY
MEDICAL LIBRARY

RC 941
In 4
1894

Copyrighted, 1894,
By WILLIAM WOOD & COMPANY

TO MY PRECEPTOR,

EPHRAIM INGALS, M.D.,

EMERITUS PROFESSOR OF MATERIA MEDICA AND MEDICAL JURISPRUDENCE IN RUSH MEDICAL COLLEGE, TO WHOSE ENCOURAGEMENT AND WISE COUNSEL I AM GREATLY INDEBTED,

This Book is Affectionately Dedicated

BY THE AUTHOR.

PREFACE TO THE THIRD EDITION.

THIS is not meant for an encyclopædic work, but is intended to present in convenient form the known facts relating to diseases of the respiratory tract and circulatory organs, and I have brought their consideration under one cover because the parts are so closely related that when one is diseased it is generally necessary to interrogate the others before a correct diagnosis or proper plan of treatment can be reached.

I have not discussed questionable theories, and have not referred to methods of treatment which do not strongly commend themselves to my judgment.

The favor with which the preceding edition of this work has been received leads me to believe that I have succeeded in my efforts, not only to aid laryngologists in their daily work but also to place these subjects clearly before students and a large class of general practitioners who of necessity must be prepared to meet any emergency.

As it is but little over a year since the second edition was published no great alteration in the text has been necessary, but several minor changes have been made, and a few pages have been added to keep abreast of our advancing knowledge on these subjects. E. F. I.

34–36 WASHINGTON ST., CHICAGO.

PREFACE TO THE SECOND EDITION.

IN the first edition of this work, the consideration of the diseases of the respiratory and circulatory systems was restricted to such a presentation of the diagnosis and treatment as I had formerly made in my lectures to classes of students. With the purpose of completing the work and increasing the value of this edition to both students and practitioners, there have been added the subjects of Etiology, Pathology, Symptomatology, and Prognosis of the diseases to which these organs are liable.

The chapters devoted to physical diagnosis have been but little changed. Those treating of diseases of the lungs and heart have been amplified and modified to correspond with the present advanced line of our knowledge on these subjects, and those relating to diseases of the throat and nasal cavities have been entirely rewritten. I have endeavored to include all diseases of the chest, throat, and nasal passages, as well as the more important affections of the œsophagus and thyroid gland, and to give to each the consideration which its frequency and importance demand. I have carefully consulted the extensive literature of these topics but have made no attempt to collate the various theories and methods suggested by different authors. I have limited the argument to that which personal knowledge of the diseases and of writers, commends to my own judgment; and I have generally confined my recommendations for treatment to those methods which have proved most efficacious in my own practice. The substance of the writings of an individual soon becomes merged in general literature which makes it impossible for me to give personal credit as I would like, to all whose labors have enriched this field, but to all such I gladly acknowledge my

indebtedness. I am indebted to Drs. Ephraim Ingals, Walter S. Haines, J. Edwin Rhodes, and Norman Bridge for aid in proof-reading, and to Dr. Arthur M. Corwin and James H. Blodgett for assistance in proof-reading and revision of copy, as well as to Dr. M. A. Olsen for the index.

E. F. I.

34–36 WASHINGTON ST., CHICAGO,
September, 1892.

PREFACE TO FIRST EDITION.

THESE lectures are designed to present a complete exposition of the subject of Physical Diagnosis so far as it relates to diseases of the Chest, Throat, and Nasal Passages; to give the essential symptoms of each disease; to point out the symptoms and signs which are of most value in a differential diagnosis; and to outline briefly the proper treatment for the various affections. The anatomical characteristics and the causes of these diseases have been pointed out wherever they are of special value in enabling the reader to understand the physical signs, or to properly apply remedial measures. When these lectures were delivered, nothing was said about treatment, but in order to enhance the value of this work to both physician and student, I have appended to the consideration of the diagnosis of each disease an outline of the treatment which I have found most satisfactory. In so doing, I have not even mentioned many methods of treatment of more or less value which have been recommended by other physicians.

In the preparation of these lectures I have availed myself of every source of information at my command, and I hope that little has been overlooked which would be of value to the student or practitioner. The study of this subject for several years, in connection with my lectures, and a large personal experience with these affections have enabled me to discriminate as to the relative importance of different signs and to detect numerous exceptions to the general rules. These exceptions, some of which are extremely rare, are of little importance to the general practitioner, and the study of them is a positive injury to the student unless their true significance is understood. Matter relating to them has, therefore, been set in small type, so that it may be omitted until the student has become thoroughly familiar with the facts that are essential.

The nature of these lectures, which contain information gathered from many different sources by study and by personal observation, and

the fact that much of which they treat has long since become public property, renders it impossible for me in every instance to give the credit to individual authors which I desire, but I freely acknowledge my indebtedness to all who have preceded me in this field. I am indebted to the courtesy of Doctors J. Solis Cohen, of Philadelphia, and Lennox Browne and Morell Mackenzie, of London, for permission to use some of the cuts which illustrate their works. I take special pleasure in expressing my obligation to my clinical assistants, Doctors Philip Leach, W. H. Taylor, and J. T. Eggers, for valuable aid in the revision of my notes.

Messrs. Sharp & Smith, of this city, have kindly furnished electrotypes for the illustrations of instruments.

E. F. I.

CONTENTS.

PAGE

Preface, ... vii
List of illustrations, ... xxiii

DISEASES OF THE CHEST.

CHAPTER I.

Physical diagnosis, ... 3
Divisions of the chest, ... 3
Methods of examination, ... 9
Inspection, ... 9
Palpation, ... 14
Mensuration, ... 16
Succussion, ... 20

CHAPTER II.

Physical diagnosis, continued, ... 21
Percussion, ... 21
In health, ... 21
In disease, ... 28
The Plessigraph, ... 31
Auscultatory percussion, ... 32

CHAPTER III.

Physical diagnosis, continued, ... 34
Auscultation, ... 34
In health, ... 39
In disease, ... 41

CHAPTER IV.

Physical diagnosis, continued, ... 48
Adventitious sounds, ... 48
Vocal sounds, ... 54

CHAPTER V.

Pulmonary diseases, ... 60
Pleurisy, ... 60
Acute pleurisy, ... 61
Subacute pleurisy, ... 72

CHAPTER VI.

PAGE
Pulmonary diseases, continued, ... 76
Chronic pleurisy or empyema, ... 76
Peculiar local forms of pleurisy, ... 82
Hydrothorax, ... 84
Pneumothorax, ... 84
Pneumo-hydrothorax, ... 85

CHAPTER VII.

Pulmonary diseases, continued, ... 89
Bronchitis, ... 89
Acute and subacute bronchitis, ... 89
Chronic bronchitis, ... 90
Capillary bronchitis, ... 95
Plastic bronchitis, ... 99
Dilatation of the bronchial tubes, ... 100
Asthma, ... 102
Pulmonary emphysema, ... 107

CHAPTER VIII.

Pulmonary diseases, continued, ... 113
Pneumonia, ... 113
Lobar pneumonia, ... 113
Lobular pneumonia, ... 123
Peculiar forms of pneumonia, ... 128
Abscess of the lungs, ... 129

CHAPTER IX.

Pulmonary diseases, continued, ... 132
Pulmonary hyperæmia, ... 132
Brown induration, ... 134
Pulmonary hemorrhage, ... 134
Pulmonary apoplexy, ... 137
Pulmonary thrombosis and embolism, ... 138
Pulmonary collapse, ... 139
Pulmonary œdema, ... 142
Pulmonary gangrene, ... 144
Pulmonary cancer, ... 146
Pulmonary tumors, ... 148
Hydatid cysts of the lungs, ... 148
Distoma pulmonale, ... 150
Syphilitic diseases of the lungs, ... 151
Enlarged bronchial glands, ... 152
Pertussis or whooping-cough, ... 153

CHAPTER X.

Pulmonary diseases, continued, ... 156
Pulmonary phthisis, ... 156
Pulmonary tuberculosis, ... 156
Acute miliary tuberculosis, ... 165
Fibroid phthisis, ... 167

CHAPTER XI.

PAGE
The heart, ... 177
Anatomy and physiology of the heart, ... 177
Physiological action of the heart, ... 180
Physical examination of the heart, ... 183
Cause of the heart sounds, ... 190
Modification of the heart sounds by disease, ... 191

CHAPTER XII.

The heart, continued, ... 195
Abnormal heart sounds, cardiac murmurs, ... 195
Anomalous heart sounds, ... 205
Subclavian murmurs, ... 206
Venous signs, ... 206
The sphygmograph, ... 208

CHAPTER XIII.

Cardiac diseases, ... 212
Pericarditis, ... 212
Pneumo-hydropericardium, ... 218
Hydropericardium, ... 218
Endocarditis, ... 219
Acute endocarditis, ... 219
Ulcerative endocarditis, ... 222
Chronic endocarditis, valvular disease of the heart, . . . 223
Myocarditis, ... 231

CHAPTER XIV.

Cardiac diseases, continued, ... 234
Simple cardiac hypertrophy, ... 234
Hypertrophy and dilatation of the heart, ... 236
Dilatation of the heart, ... 239
Atrophy of the heart, ... 242
Fatty heart, ... 242
Aneurism of the heart, ... 245
Rupture of the heart, ... 245
Syphilitic disease of the heart, ... 245
Tumors of the heart, ... 246
Morbus cæruleus, ... 246
Neurotic or functional disease of the heart, ... 247
Tachycardia, ... 249
Bradycardia, ... 250
Angina pectoris, ... 250

CHAPTER XV.

Diseases of the thoracic arteries, ... 254
Aortitis, ... 254
Atheroma of the aorta, ... 254

PAGE
Aortic or thoracic aneurism, ... 256
Aneurism of the sinuses of Valsalva, ... 257
Aneurism of the arch of the aorta, ... 257
Aneurism of the descending aorta, ... 257
Coarctation of the aorta, ... 266
Solid mediastinal tumors, ... 267

DISEASES OF THE THROAT.

CHAPTER XVI.

The throat, ... 271
Examination of the fauces, ... 271
Laryngoscopy, ... 272
Obstacles to laryngoscopy, ... 289
Infra-glottic laryngoscopy, ... 292

CHAPTER XVII.

The throat, continued, ... 293
The larynx and rhinoscopy, ... 293
Examination of the trachea, ... 300
Rhinoscopy, ... 301
Anterior rhinoscopy, ... 301
Posterior rhinoscopy, ... 302
Obstacles to posterior rhinoscopy, ... 304
Vault of the pharynx and posterior nasal cavities, ... 307

CHAPTER XVIII.

Diseases of the fauces, ... 311
Acute sore throat, ... 311
Erysipelatous sore throat, ... 314
Rheumatic sore throat, ... 316
Acute rheumatic sore throat, ... 316
Chronic rheumatic sore throat, ... 318
Sore throat of small-pox, ... 321
Sore throat of measles, ... 322
Sore throat of scarlet fever, ... 323
Simple membranous sore throat, ... 324

CHAPTER XIX.

Diseases of the fauces, continued, ... 328
Diphtheria, ... 328

CHAPTER XX.

Diseases of the fauces, continued, ... 339
Acute follicular pharyngitis, ... 339
Chronic follicular pharyngitis, ... 340
Acute follicular glossitis, ... 347

PAGE
Chronic follicular glossitis, ... 347
Scrofulous sore throat, ... 348
Acute tubercuar sore throat, ... 350
Syphilitic sore throat, ... 353
Syphilitic sore throat in infants, ... 356

CHAPTER XXI.

Diseases of the fauces, continued, ... 358
Diseases of the uvula, ... 358
Acute inflammation and œdema of the uvula, ... 358
Chronic inflammation and elongation of the uvula 358
Malformation and new growths of the uvula, ... 359
Leucoplakia buccalis, ... 360
Acute tonsillitis, ... 362
Phlegmonous tonsillitis, ... 368
Hypertrophy of the tonsils, ... 370
Concretions in the tonsils, ... 375
Mycosis of the throat, ... 376
Tubercular ulceration of the tonsils, ... 378
Cancer of the tonsil, ... 380

CHAPTER XXII.

Diseases of the pharynx, ... 382
Foreign bodies in the pharynx, ... 382
Retro-pharyngeal abscess, ... 383
Tumors of the pharynx, ... 386
Cancer of the pharynx, ... 386
Neuroses of the pharynx, ... 388
Anæsthesia of the pharynx, ... 388
Hyperæsthesia of the pharynx, ... 388
Paræsthesia of the pharynx, ... 389
Spasm of the pharynx, ... 390
Paralysis of the pharynx, ... 391
Scalds and burns of the pharynx, ... 392
Swallowing the tongue, ... 392
Diseases of the valeculæ and pyriform sinuses, ... 393

CHAPTER XXIII.

Diseases of the larynx, ... 394
Acute laryngitis, ... 394
Subacute laryngitis, ... 397
Traumatic laryngitis, ... 398
Chronic laryngitis, ... 398
Trachoma of the vocal cords, ... 408
Phlebectasis laryngea, ... 409

CHAPTER XXIV.

Diseases of the larynx, continued, ... 411
Membranous croup, ... 411

CHAPTER XXV.

PAGE
Diseases of the larynx, continued, ... 427
Phlegmonous laryngitis, ... 427
Erysipelatous laryngitis, ... 428
Abscess of the larynx, ... 429
Œdema of the larynx, ... 430
Chondritis and perichondritis of the laryngeal cartilages, . . . 433
Tubercular laryngitis, ... 434
Syphilitic laryngitis, ... 443
Syphilitic laryngitis in infants, ... 449

CHAPTER XXVI.

Diseases of the larynx, continued, ... 451
Lupus of the larynx, ... 451
Lepra of the larynx, ... 454
Hypertrophy of the larynx, ... 455
Laryngitis of small-pox, ... 455
Laryngitis of measles, ... 455
Laryngitis of scarlet fever, ... 455
Chronic stenosis of the larynx, ... 456
Stenosis of the trachea, ... 460
Tracheitis, ... 460

CHAPTER XXVII.

Diseases of the larynx, continued, ... 463
Morbid growths in the larynx, ... 463
Benign tumors of the larynx, ... 465
Malignant tumors of the larynx, ... 476
Eversion of the ventricle of Morgagni, ... 483
Tracheal tumors, ... 483
Post-tracheotomy vegetations, ... 485
Involution of the trachea, ... 485
Tracheocele, ... 486
Syphilis of the trachea, ... 487

CHAPTER XXVIII.

Diseases of the larynx, continued, ... 489
Fracture of the larynx, ... 489
Dislocation of the larynx, ... 490
Foreign bodies in the larynx, ... 490
Foreign bodies in the trachea, ... 492
Spasm of the glottis, ... 496
Spasms of the larynx in adults, ... 497
Irritative cough, ... 498
Nervous cough, ... 498
Anæsthesia of the larynx, ... 499
Hyperæsthesia, paræsthesia, and neuralgia of the larynx, . . . 500
Chorea laryngis, ... 501

PAGE
Spasm of the vocal cords, ... 502
Falsetto voice, ... 503
Laryngeal vertigo, ... 504

CHAPTER XXIX.

Diseases of the larynx, continued, ... 505
Paralysis of the thyro-epiglottic and ary-epiglottic muscles, . . . 505
Paralysis of the crico-thyroid muscles, ... 506
Paralysis of the thyro-arytenoid muscles, ... 507
Bilateral paralysis of the lateral crico-arytenoid muscles, . . . 508
Unilateral paralysis of the lateral crico-arytenoid muscles, . . . 510
Paralysis of the arytenoid muscle, ... 511
Bilateral paralysis of the posterior crico-arytenoid muscles, . . 511
Unilateral paralysis of the posterior crico-arytenoid muscles, . . . 514
Anchylosis of the arytenoid cartilages, ... 514
Atrophy of the vocal cords, ... 515

DISEASES OF THE NOSE.

CHAPTER XXX.

Diseases of the nasal cavities, ... 519
Influenza, ... 519
Rhinitis, ... 522
Simple acute rhinitis, ... 522
Traumatic rhinitis, ... 526
Chronic rhinitis, ... 527
Simple chronic rhinitis, ... 528

CHAPTER XXXI.

Diseases of the nasal cavities, continued, ... 531
Rhinitis, continued, ... 531
Chronic rhinitis, continued, ... 531
Intumescent rhinitis, ... 531
Hypertrophic rhinitis, ... 540
Submucous infiltration at the sides of the vomer, . . . 547
Atrophic rhinitis, ... 547

CHAPTER XXXII.

Diseases of the nasal cavities, continued, ... 553
Hay fever, ... 553
Furunculosis of the nose, ... 558
Epistaxis, ... 559

CHAPTER XXXIII.

Diseases of the nasal cavities, continued, ... 564
Nasal mucous polypi, ... 564
Nasal fibrous polypi, ... 569

PAGE
Nasal papillary tumors, ... 569
Nasal vascular tumors, ... 570
Nasal osseous cysts, ... 570
Nasal cartilaginous tumors, ... 571
Nasal bony tumors, ... 571
Nasal malignant tumors, ... 572

CHAPTER XXXIV.

Diseases of the nasal cavities, continued, ... 574
Syphilis of the nose, ... 574
Congenital syphilis of the nose, ... 577
Tuberculosis of the nares, ... 578
Empyema of the antrum, ... 579
Empyema of the sphenoidal sinuses, ... 583
Inflammation of the frontal sinus 584
Chronic suppurative ethmoiditis 585
Lupus of the nares, ... 587
Rhinoscleroma, ... 588
Glanders, ... 589
Nasal affections in acute diseases, ... 591
Perverted sense of smell, ... 591
Parosmia, ... 591
Anosmia, ... 591

CHAPTER XXXV.

Diseases of the nasal cavities, continued, ... 593
Congenital deformity of the nose, ... 593
Fractures of the nose, ... 593
Dislocation of the nasal bones, ... 594
Deflection of the nasal septum, ... 594
Ecchondroma and exostosis of the nasal septum, ... 597
Perforation of the nasal septum, ... 601
Hæmatoma of the nasal septum, ... 602
Abscesses of the nasal septum, ... 603
Foreign bodies in the nose, ... 603
Rhinoliths, ... 604
Myasis narium or maggots in the nose, ... 605

CHAPTER XXXVI.

Diseases of the naso-pharynx, ... 607
Rhino-pharyngitis, ... 607
Throat deafness, ... 610
Hypertrophy of the pharyngeal tonsil, ... 613
Retronasal fibrous tumors, ... 620
Retronasal fibro-mucous tumors, ... 624
Retronasal cartilaginous tumors, ... 625
Malignant tumors of the naso-pharynx, ... 625
Cystic tumors of the naso-pharynx, ... 626

DISEASES OF THE THYROID GLAND AND THE ŒSOPHAGUS.

CHAPTER XXXVII.

PAGE
Goitre, ... 629
Exophthalmic goitre, ... 632
Œsophagitis, ... 632
Acute œsophagitis, ... 632
Chronic œsophagitis, ... 633
Stricture of the œsophagus, ... 634
Compression of the œsophagus, ... 637
Spasm of the œsophagus, ... 637
Paralysis of the œsophagus, ... 638
Foreign bodies in the œsophagus, ... 640
Paræsthesia of the œsophagus, ... 642

APPENDIX.

Formulæ for prescriptions, ... 645
Gargles, ... 647
Sedatives, ... 647
Astringents, ... 647
Stimulants, ... 647
Antiseptics, ... 647
Trochisci or lozenges, ... 647
Sedatives, ... 647
Demulcents, ... 648
Astringents, ... 648
Stimulants, ... 648
Antiseptics, ... 649
Vapor inhalations, ... 649
Sedatives, ... 650
Antispasmodics, ... 650
Mild stimulants, ... 650
Strong stimulants, ... 651
Spray inhalations, ... 651
Sedatives, ... 651
Astringents and stimulants, ... 652
Hæmostatics, ... 653
Antiseptics, ... 653
Dry inhalations, ... 654
Sedatives, ... 654
Stimulants, ... 654
Fuming inhalations, ... 654
Sedatives, ... 655
Stimulants, ... 655
Pigments, ... 655
Local anæsthetics, ... 655

PAGE

Astringents, ... 655
Stimulants and caustics, ... 656
Antiseptics, ... 656
Insufflations, ... 656
Sedatives, ... 656
Antiseptics and stimulants, ... 657
Astringents and stimulants, ... 657
Nasal douches, ... 658

LIST OF ILLUSTRATIONS.

FIG.		PAGE
1.	Regions of the chest,	4
2.	Regions of the chest,	5
3.	Outline of the chest,	10
4.	Quain's stethometer,	17
5.	Carroll's stethometer,	17
6.	Flint's cyrtometer,	18
7.	Spirometer,	18
8.	Allison's stethogoniometer,	18
9.	Hammond's hæmadynamometer,	19
10.	Flint's hammer and pleximeter,	21
11.	Camman's stethoscope,	32
12.	Ingals' emballometer,	33
13.	Solid wooden stethoscope,	36
14.	Knight's stethoscope,	36
15.	Allison's differential stethoscope,	37
16.	Phthisis,	47
17.	Bronchial râles,	49
18.	Acute pleurisy,	53
19.	Curved line of flatness in pleurisy, posterior view,	64
20.	Curved line of flatness in pleurisy, anterior view,	65
21.	Subacute pleurisy,	73
22.	Cabot's drainage tubes,	79
23.	Strong's drainage tubes,	79
24.	Ingals' flat trocar,	79
25.	Ingals' drainage tubes,	81
26.	Pneumo-hydrothorax,	86
27.	Pneumonia,	117
28.	Tubercle,	157
29.	Tubercle bacilli, colored plate,	168
30.	Globe nebulizer,	174
31.	Physiological action of the heart,	181
32.	Rhythm of the heart,	183
33.	Areas of endo-cardial murmurs,	198
34.	Auricular systole,	201
35.	Ventricular systole,	202
36.	Marey's sphygmograph,	208
37.	Normal radial pulse, tracings,	208
38.	Normal radial pulse, tracings,	208
39.	Aortic obstruction,	209
40.	Aortic obstruction,	209
41.	Mitral regurgitation,	209

FIG. PAGE
42. Aneurism, ... 209
43. Aortic regurgitation, ... 209
44. Aortic regurgitation and obstruction, ... 209
45. Cardiac hypertrophy in Bright's disease, ... 210
46. Tracing of the senile pulse, ... 210
47. Mitral constriction, tracing, ... 210
48. Mitral constriction and aortic regurgitation, tracing, ... 211
49. Mitral hypertrophy and dilatation, ... 211
50. Türck's tongue depressor, ... 271
51. Pocket tongue depressor, ... 271
52. Bosworth's tongue depressor, ... 271
53. Throat mirrors for laryngoscopy, ... 273
54. Schrötter's head band with nasal rest, ... 278
55. Krishaber's illuminator, ... 278
56. Modified Mackenzie's rack-movement bull's-eye condenser, . . . 278
57. Modification of Mackenzie's illuminator, ... 279
58. Laryngoscopic reflector, ... 283
59. Position of the head giving the best view of the larynx, . . . 284
60. Position of the head giving a poor view of the larynx, ... 285
61. Laryngoscopic mirror in position, ... 286
62. Brun's pincette, ... 291
63. Infra-glottic laryngoscopy, ... 291
64. Relative relations of the larynx and its image, ... 293
65. Normmal larynx in respiration, ... 293
66. Pitcher-shaped inter-arytenoid fold, ... 295
67. Lapping of arytenoid cartilages in phonation, ... 295
68. Cushion of epiglottis, ... 295
69. Pointed epiglottis, ... 295
70. Jews'-harp epiglottis, ... 295
71. Larynx of a woman in respiration, ... 295
72. View of left side of larynx, ... 297
73. Normal larynx of woman in formation of head tones, ... 298
74. View of posterior wall of trachea, ... 300
75. View of anterior wall of trachea, ... 300
76. Ingals' nasal speculum, ... 301
77. Jarvis' nasal speculum, ... 301
78. Sajous' nasal speculum, ... 301
79. Cross section of head showing ethmoid cells and nasal cavities, . . 302
80. Fraenkel's rhinoscope, ... 303
81. Position for rhinoscopy, ... 304
82. Rubber palate retractor, ... 306
83. Porcher's self-retaining uvula and palate retractor, ... 306
84. Palate retractor, ... 306
85. Rhinoscope with uvula holder, ... 306
86. Rhinoscopic image, ... 307
87. Adenoid tissue at vault of the pharynx, ... 309
88. Pharyngeal bursa, ... 309
89. Chronic follicular pharyngitis, ... 343
90. Modification of Shurly's battery, ... 345
91. Ingals' cautery electrodes, ... 346
92. Perforation of the palate, syphilitic, ... 354

FIG. PAGE
93. Scissors for amputating the uvula, ... 359
94. Mathieu's tonsillitome, ... 372
95. Mathieu's tonsillitome, oblique fenestra, ... 372
96. Ingals' tonsil forceps, ... 373
97. Fibroma of pharynx, ... 386
98. Superficial ulcers of the vocal cords, ... 395
99. Superficial ulceration of the epiglottis, ... 395
100. Mackenzie's laryngeal lancet, ... 397
101. Catarrhal ulcer of the vocal cord, ... 399
102. Chronic catarrhal laryngitis, with deformity, ... 399
103. Chronic catarrhal laryngitis, ... 401
104. Catarrhal laryngitis, with deformity, ... 401
105. Subglottic œdema, ... 401
106. Davidson's atomizers, set No. 66, ... 405
107. Ingals' laryngeal applicator, ... 405
108. Davidson's atomizer, No. 59 old style, ... 406
109. Trachoma of vocal cords, ... 408
110. Ingals' chromic acid applicator and handle, ... 409
111. Ingals' galvano-cautery handle, ... 409
112. O'Dwyer's intubation instruments, ... 418
113. Henrotin's gag, ... 419
114. Waxham's gag, ... 419
115. Allingham's gag, ... 419
116. O'Dwyer's extractor, ... 420
117. Abscess of the larynx, ... 429
118. Infra-glottic abscess of the larynx, ... 430
119. Infra-glottic abscess of the larynx, twelve hours after opening, . . 430
120. Œdema of the larynx, ... 432
121. Tubercular laryngitis, ... 435
122. Tubercular laryngitis, pyriform swelling of the arytenoids, . . 435
123. Tubercular laryngitis, pyriform swelling of the arytenoids, . . 435
124. Tubercular laryngitis, ... 435
125. Incipient tubercular laryngitis, ... 436
126. Tubercular laryngitis, ... 436
127. Tubercular ulceration of the vocal cords, ... 437
128. Tubercular ulceration of the vocal cords, ... 437
129. Tubercular ulceration of the ventricular bands, ... 438
130. Tubercular ulceration of the ventricular bands and vocal cords, . . 438
131. Tubercular laryngitis, sluggish action of the vocal cords, . . . 438
132. Tubercular ulceration of the larynx, ... 440
133. Tubercular laryngitis, with syphilis, ... 440
134. Condyloma of the epiglottis, ... 444
135. Gumma of the larynx, ... 444
136. Multiple gumma of the larynx, ... 444
137. Syphilitic laryngitis, ... 444
138. Syphilitic laryngitis, ... 446
139. Syphilitic ulceration of the epiglottis, ... 446
140. Syphilitic ulceration, ... 446
141. Lupus of the larynx (Ziemssen), ... 451
142. Lupus of the larynx (Türck), ... 452
143. Lepra of the larynx, ... 454

FIG. PAGE
144. Syphilitic laryngitis, ... 456
145. Syphilitic stenosis of larynx, ... 456
146. Mackenzie's laryngeal dilator, ... 458
147. Whistler's cutting dilator, ... 458
148. Tube for laryngo-tracheal stenosis, ... 459
149. Mount Bleyer's tongue depressor, ... 464
150. Papilloma of right vocal cord, ... 465
151. Papilloma of the larynx, ... 465
152. Papilloma of vocal cords, ... 466
153. Papilloma of vocal cords, ... 466
154. Papilloma of the larynx, ... 466
155. Fibroma of left vocal cord, ... 466
156. Fibro-cellular tumor of the larynx, ... 467
157. Cystic tumor of the larynx, ... 467
158. Cystic tumor of the larynx, ... 467
159. Cyst of the epiglottis, ... 467
160. Adenoid tumor of the larynx, ... 467
161. Adenoid tumor of the larynx, ... 467
162. Cartilaginous tumor of the larynx, ... 468
163. Vascular tumor of the larynx, ... 468
164. Vascular tumor of the larynx, ... 468
165. Laryngeal forceps, ... 471
166. Mackenzie's tube forceps, ... 472
167. Stoerk's laryngeal instruments, ... 473
168. Tobold's laryngeal knives, ... 474
169. Cancer of the larynx, ... 477
170. Cancer of the larynx, ... 477
171. Cancer of the larynx, ... 477
172. Cancer of the larynx, ... 477
173. Cancer of the larynx, ... 478
174. Cancer of the larynx, ... 478
175. Mixed sarcoma of larynx, ... 478
176. Cancer of the larynx, ... 478
177. Tumor in the trachea, ... 484
178. Ingals' punch forceps, ... 485
179. Syphilitic laryngitis, ... 487
180. Seiler's tube forceps, ... 495
181. Bilateral paralysis of the crico-thyroid muscles, ... 507
182. Acute laryngitis, ... 507
183. Paralysis of the thyro-arytenoid muscles, ... 508
184. Paralysis of the lateral crico-arytenoid muscles, ... 508
185. Mackenzie's laryngeal electrodes, ... 509
186. Unilateral paralysis of the lateral crico-arytenoid muscles, respiration, 510
187. Unilateral paralysis of the lateral crico-arytenoid muscles, phonation, . 510
188. Unilateral paralysis of the crico-arytenoid muscles, ... 510
189. Ziemssen's laryngeal electrodes, ... 511
190. Bilateral paralysis of the posterior crico-arytenoid muscles, inspiration, ... 512
191. Bilateral paralysis of the posterior crico-arytenoid muscles, expiration, 512
192. Unilateral paralysis of the posterior crico-arytenoid muscles, inspiration, ... 514

FIG. PAGE
193. Unilateral paralysis of the posterior crico-arytenoid muscles, phonation, ... 514
194. Anchylosis of the arytenoid cartilages, ... 514
195. Powder blower, ... 536
196. Davidson's oil atomizer, No. 50, ... 536
197. Flat nasal probe and applicator, ... 537
198. Hypertrophy of the inferior turbinated body, ... 541
199. Hypertrophy of the posterior ends of the inferior turbinated bodies, . 542
200. Ingals' nasal scissors, ... 545
201. Nasal burrs, ... 546
202. Nasal trephines, ... 546
203. Submucous infiltration at sides of the vomer, ... 547
204. Ingals' nasal syringe, ... 550
205. Nasal douche, ... 551
206. Nasal douche, traveller's, ... 551
207. Galvano-cautery handle with écraseur, ... 567
208. Ingals' snare, ... 567
209. Cotton applicator, ... 568
210. Hypodermic syringe, long silver nozzle, ... 568
211. Ingals' nasal dressing forceps, ... 576
212. Cross section of head looking from behind forward, ... 579
213. Ingals' electric lamp, ... 581
214. Brainard's bone drill, ... 582
215. Ingals' drainage tubes for antrum, ... 583
216. Cross section of head, ... 584
217. Curtis' ethmoid-cell wash-bottle, ... 586
218. Ingals' septum forceps, ... 596
219. Ingals' septum knife, ... 596
220. Ingals' right-angle cutting forceps, ... 597
221. Exostosis from the septum, ... 598
222. Sajous' knife, ... 599
223. Nasal spud, ... 599
224. Ingals' nasal saw, ... 599
225. Ingals' flat nasal saw, ... 599
226 and 227. Sajous' saws, ... 599
228. Ingals' heavy bone scissors, ... 600
229. Ingals' nasal bone forceps, ... 600
230. Ingals' nasal spatula, ... 600
231. Gross' instruments for removing foreign bodies, ... 604
232. Post-nasal syringe, ... 609
233. Curtis' Eustachian tube vaporizer, ... 612
234. Rhinoscopic view of post-nasal vegetations, ... 614
235. Mackenzie's, John N., post-nasal forceps, ... 617
236. Ingals' post-nasal snare applicator, ... 623
237. Retro-nasal fibro-mucous polypus, ... 624
238. Sand's œsophagotome, ... 636
239. Flexible œsophageal forceps, ... 641
240. Bristle extractor, ... 642

Diseases of the Chest

CHAPTER I.

PHYSICAL DIAGNOSIS.

In this work I shall first describe the methods for detecting disease which are based upon the pathological changes in the organs affected; next point out the characteristics and significance of the various signs; and finally consider the individual diseases.

The term physical diagnosis is used to designate the methods referred to, whether used in the examination of the chest or in the examination of any other part of the body; but as it is in the exploration of the chest that such methods have yielded the most brilliant results, it is now customary to apply the term physical diagnosis simply to the examination of the thorax.

It is in this limited sense that we shall generally use it, though it will also be applied to the examination of the upper air passages.

DIVISIONS OF THE CHEST.

To simplify the study, and to enable us to fix accurately in mind the position of the intra-thoracic organs, the chest has been divided into a number of regions which are purely arbitrary, and their boundaries vary with different authors.

J. M. Da Costa divides the chest into the anterior, the posterior, and two lateral regions, and subdivides these into upper and lower regions. He locates signs present in these regions by certain fixed marks which may be found on the surface of the chest. For instance, anteriorly, a sign may be located in a certain intercostal space, or beneath a rib or the clavicle, at a given distance from the sternum. Posteriorly, a sign may be located in a similar manner with reference to the spinous processes, or to the angles and the borders of the scapulæ. Such a division is well enough for the record of cases, but it does not aid us in remembering the location of the intra-thoracic organs.

The division here adopted is similar to one quite commonly taught, with only such changes as make it plainer and more easily remembered.

While learning these boundaries, one should fix in mind the exact position of the intra-thoracic organs.

We divide the chest primarily into anterior, posterior, and lateral regions, and subdivide as follows.

Upon the anterior surface on either side, from above downward, we have the supra-clavicular, clavicular, infra-clavicular, mammary, and infra-mammary regions; between these two lateral groups we find the supra-sternal above the line of the clavicles, and the sternal region subdivided into the superior-sternal and inferior-sternal.

The posterior portion of the chest, on each side, is subdivided into the supra-scapular and the scapular regions, between these the inter-scapular region, and below the scapulæ the infra-scapular regions (Fig. 2).

Laterally we have the axillary and the infra-axillary regions.

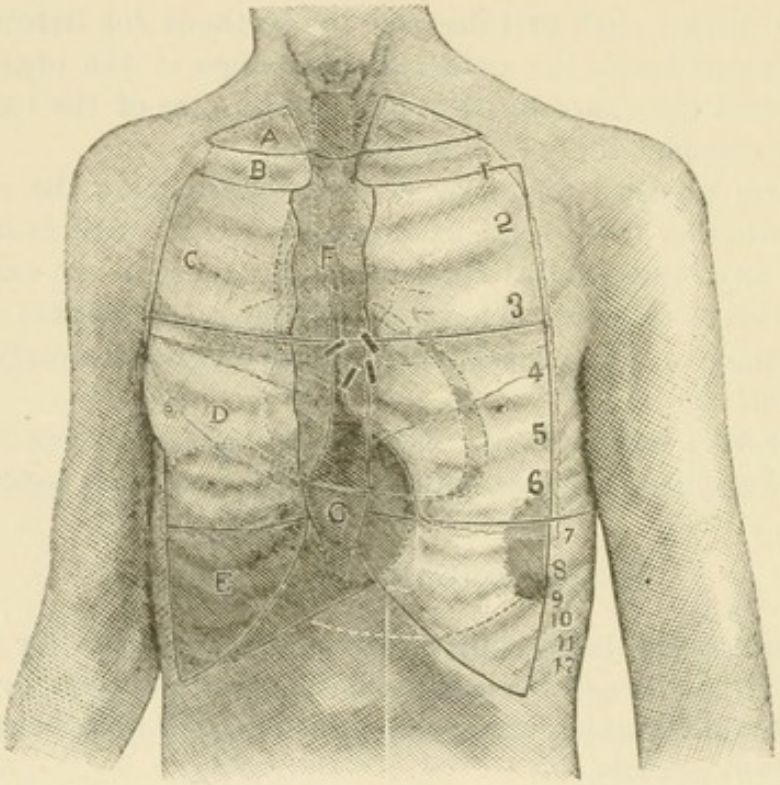

FIG. 1.—A, Supra-clavicular region; B, clavicular region; C, infra-clavicular region; D, mammary region; E, infra-mammary region; F, superior-sternal region; G, inferior-sternal region. The wavy lines represent the borders of the lungs and the pulmonary fissures. The dotted lines correspond to the outlines of the various organs, viz., trachea, aorta, bronchial tubes, heart, liver, spleen, and stomach. The very dark shading over the solid viscera shows the normal areas of flatness, and the shading next lighter over the upper part of the liver shows the hepatic dulness. The black rectangular spots near the third rib correspond to the position of the valves of the heart.

THE SUPRA-CLAVICULAR REGION corresponds to that portion of the pleural cavity which extends above the clavicles. It is triangular in form, with its base internal, its apex external. It is bounded above by a line drawn from the upper ring of the trachea outward to the junction of the middle with the external third of the clavicle. The inferior boundary of this region corresponds to the upper margin of the inner two-thirds of the clavicle. The internal boundary corresponds to the sterno-cleido-mastoid muscle. This region contains, on either side, the apex of the lung and portions of the subclavian artery and vein.

THE CLAVICULAR REGION corresponds to the inner two-thirds of

the clavicle and is bounded above and below by the borders of the bone. It contains lung tissue. Upon the right side, externally we find the subclavian artery, and at the inner extremity the arteria innominata and the recurrent laryngeal nerve as it passes up to supply the muscles of the larynx. Aneurisms in this locality, by pressing upon this nerve, give rise to serious symptoms due to paralysis or spasm of the glottis. Upon the left side, at the inner end of this region we find the carotid and the subclavian arteries, deeply seated and running almost at right angles with the clavicle.

THE INFRA-CLAVICULAR REGION is bounded above by the clavicle, internally by the margin of the sternum, and externally by a straight

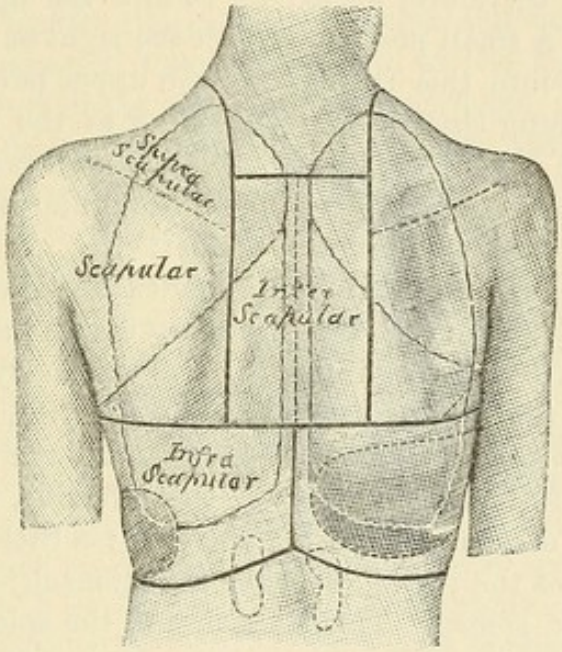

FIG. 2.—The wavy lines correspond to the borders and fissures of the lungs. The dotted line across the scapular region indicates the position of the spine of the scapula. The dotted lines and shaded areas in the infra-scapular regions indicate the position of the liver and spleen.

line let fall from the outer extremity of the clavicular region, and passing about an inch externally from the nipple. It is bounded below by the lower margin of the third rib. This region contains lung tissue on either side. On the right, close to the border of the sternum, we find portions of the ascending aorta and of the descending vena cava. Just beneath the second costal cartilage, we find the right bronchus as it passes into the right lung. Upon the left, in the second intercostal space, close to the margin of the sternum, the pulmonary artery is located. In the same space is found the left bronchus, which inclines more downward, and is located lower than the main bronchus on the opposite side. A portion of the base of the heart occupies the internal inferior angle of this region.

THE MAMMARY REGION, which lies immediately below the preceding, is bounded internally by the margin of the sternum, externally by

a continuation of the line which bounds the infra-clavicular region, and inferiorly by the lower margin of the sixth rib. We may easily remember the boundaries of the infra-clavicular and the mammary regions, by recollecting that we have three ribs in each. The inferior border of the third rib forms the lower boundary of the upper region and the lower margin of the sixth rib bounds the lower region inferiorly. This region contains lung tissue on both sides. On the right, the thin margin of the lung, which overlaps the liver, reaches to the sixth interspace, and extends even lower in full inspiration. Deeper seated we find the upper convex surface of the liver, carrying the diaphragm above it, as high as the fourth intercostal space. The nipple is usually located in the fourth intercostal space; therefore, we expect to find the upper border of the liver beneath it. A small portion of both the right auricle and the right ventricle extends into this region. In the upper part of the *left* mammary region, the lung tissue is in front as low as the fourth rib. Here the border of the lung passes outward and downward to the fifth rib, leaving between it and the median line a triangular space in which the heart and its investing membrane are superficial.

THE INFRA-MAMMARY REGION is bounded externally by a continuation of the outer boundary of the mammary region; above by the lower margin of the sixth rib, and internally and inferiorly by the margin of the sternum and the lower borders of the false ribs. This region contains, on the right side, the liver, and occasionally the inferior margin of the lung during full inspiration. On the left side, near the sternum, we find a portion of the left lobe of the liver; a little farther outward, near the middle of the region, we have the stomach; in the outer third is a portion of the spleen. The stomach and the spleen usually extend as high as the sixth rib.

The mammillary or nipple line is a vertical line drawn through the nipple, and, according to some authors, it forms the external boundary of the infra-clavicular, mammary, and infra-mammary regions.

The regions between the lateral portions of the anterior surface of the chest are three in number.

THE SUPRA-STERNAL REGION, the first counting from above, is bounded inferiorly by the upper end of the sternum, or inter-clavicular notch; laterally by the sterno-cleido-mastoid muscles; and above by the first ring of the trachea. The most important organs in this region are the trachea and the thyroid gland, the lobes of which lie on each side of the trachea and are connected by the isthmus in the upper part of this region. Here are also found certain small veins and arteries which are of interest to the surgeon. In the lower right angle of this region the innominate artery is found, and in the inter-clavicular notch we can frequently feel the arch of the aorta.

THE SUPERIOR-STERNAL REGION, next in order, is bounded below by a line connecting the lower margins of the third ribs, and lat-

erally by the borders of the bone. This region contains lung tissue. Superficially, the inner or anterior margin of each lung reaches the median line. Deeper, we find the descending vena cava, the ascending, the transverse, and a part of the descending portion of the arch of the aorta, and at the left a portion of the pulmonary artery. At a point opposite the second costo-sternal junction is the bifurcation of the trachea.

THE INFERIOR-STERNAL REGION, known also as the sternal region, has for its boundaries the borders of all that portion of the sternum lying below the third rib. In it the anterior margin of the right lung corresponds to the median line, and is superficially situated. But the corresponding margin of the left lung recedes from the median line at the level of the fourth rib, passing outward and downward, leaving a triangular space between it and the margin of the right lung. In this space the right ventricle of the heart is superficial. In the upper part of this region we find a large portion of the right auricle and the origin of both the aorta and the pulmonary artery. The portions of the left side of the heart which present anteriorly lie to the left of this region.

In this region we find portions of the four sets of valves which guard the orifices of the heart (Fig. 1). At the left edge of the sternum, under the third rib, are the pulmonary valves; a trifle lower, beneath the centre of the sternum, are located the aortic valves; lower yet, at its left border in the third intercostal space, we find the mitral valves. We locate the tricuspid valves beneath the middle of the sternum on a line with the fourth costo-sternal articulation. These valves lie so closely that a circle scarcely more than an inch in diameter will include all of them, and a circle of half that diameter will embrace a portion of each.

At the lower part of this region we have a portion of the liver and of the attachment of the pericardium to the diaphragm.

The mesosternal line is an imaginary line passing down the centre of the sternum.

The sternal lines of the right and left sides correspond to the borders of the sternum.

Posteriorly are the supra-scapular and the scapular regions on each side, extending from the second to the seventh rib and corresponding very nearly to the outlines of the scapula when the patient's arms are hanging loosely by his sides (Fig. 2).

THE SUPRA-SCAPULAR REGION corresponds to the supra-spinous fossa. It is occupied by lung tissue.

THE SCAPULAR REGION corresponds to the infra-spinous fossa. It is occupied by lung tissue.

THE INTER-SCAPULAR REGION lies between the borders of the scapulæ divided by the spinous processes of the vertebræ, and extends from the level of the second dorsal vertebra to the level of the seventh. It contains lung substance, the main bronchi, and the bronchial glands. The

descending aorta runs along the left of the spinal column, beside the œsophagus. The trachea bifurcates opposite the third dorsal vertebra.

In the three preceding regions the chest walls are very thick.

THE INFRA-SCAPULAR REGION on either side is bounded internally by the spinous processes of the vertebræ; externally by a vertical line let fall from the inferior angle of the scapula; above by the lower margin of the scapular and inter-scapular regions, which corresponds to the seventh rib; and below by the inferior margin of the false ribs. This region contains lung tissue on either side, extending to the tenth or to the eleventh rib. Below the margin of the lung, on the right side, we have the liver; on the left side, the intestines are superficial near the middle portion of the region, and externally we find the spleen (Fig. 2). The kidneys are located near the spinal column on either side. The left kidney extends an inch higher than the right, and its upper extremity is frequently found in this region.

Laterally we have two regions, the axillary and the infra-axillary.

THE AXILLARY REGION is bounded below by a line drawn from the lower margin of the mammary region backward to the inferior angle of the scapula; above by the axilla; in front by the outer boundaries of the infra-clavicular and the mammary regions; and posteriorly by the axillary border of the scapula. This region contains lung tissue on each side and, deeply seated, the main bronchi.

THE INFRA-AXILLARY REGION is bounded above by the axillary; posteriorly by the outer margin of the infra-scapular region; anteriorly by the external margin of the infra-mammary region; below by the margin of the false ribs. On either side we find the lower border of the lung running from near the upper anterior angle of this region downward and backward. Below this, on the right the liver, and on the left the spleen, and a portion of the stomach, are superficial.

Pulmonary Fissures.—On each side at a point about three inches below the apex of the lung, corresponding very nearly to the inner end of the spine of the scapula, we find the beginning of the pulmonary fissure which separates the upper from the lower lobe. These fissures run obliquely downward and forward to the sixth rib near the mammillary line. On the right side at a point on this fissure, four or five inches from the sternum, we find the commencement of another fissure, which passes inward to the margin of the lung near the fourth costal cartilage. By this fissure a small triangular portion is cut off from the lower part of the upper lobe to form the middle lobe of the right lung. The positions of these fissures necessarily change considerably with inspiration and expiration.

It is a common error with students to suppose that the interlobar fissures run in the opposite direction; that is, downward and backward from the upper part of the anterior margins of the lungs.

METHODS OF PHYSICAL EXAMINATION.

The principal methods of physical examination, six in number, are: Inspection, Palpation, Mensuration, Succussion, Percussion, and Auscultation. Unfortunately the majority of physicians rely for their diagnosis almost exclusively upon auscultation. There are many cases in which it will be necessary to use every method and to scrutinize every symptom before one can arrive at an accurate diagnosis.

The evidences of disease which these methods furnish are known as signs or physical signs.

There is a marked difference between symptoms and signs. Subjective symptoms, which are chiefly derived from the statements of the patient, may be called *presumptive* evidence of disease, while objective signs are considered *positive* evidence.

The value of these signs will depend upon a knowledge of the alterations which produce them.

The early students of physical diagnosis noted the various characteristics of a sign accurately, and located it upon the surface of the chest; then at the autopsy they sought to ascertain its causes. At present we only need to study the sign clinically, for its causes may be learned from text-books; however, it will be of great advantage, when possible, to study at the autopsy, lesions the evidences of which we have discovered by physical diagnosis.

INSPECTION.

By inspection we learn the general appearance of the patient, the color of the integument, the presence or absence of subcutaneous emphysema, œdema, or tumors, and the size, form, and movements of the chest.

Whatever method of physical diagnosis is employed, it is necessary, first, to be familiar with the healthy conditions which it would reveal.

The healthy chest has a generally rounded or convex appearance; the shoulders are level, the clavicles are horizontal, and the two sides are almost perfectly symmetrical; however, in many cases more or less depression will be observed in the supra-clavicular and infra-clavicular regions, and not infrequently the pectoral muscles are better developed on one side than on the other.

In men a deep furrow just below the fifth rib marks the lower border of the pectoralis major muscle. At the borders of the sternum, about an inch below the clavicles, we often notice rounded prominences about an inch in diameter, which mark the position of the second costal cartilages. These are frequently mistaken by students for abnormal swellings. In some patients the ribs and the intercostal spaces are

very distinct, while in others they are hidden by adipose tissue. The obliquity of the inferior ribs varies greatly in different individuals.

In the fifth intercostal space, about two inches to the left of the sternum, we observe the impulse of the chest walls caused by the apex beat of the heart.

Occasionally we find local bulging or depression, independent of disease of the internal organs. The prominent sternum known as pigeon-breast, usually due to violent cough or obstructed respiration, as from catarrh or enlarged tonsils in childhood; the pear-shaped chest, due to rachitis, and the long, narrow, and flat chest, which often results from rapid growth, are all found independent of intra-thoracic disease.

There is often bulging of the præcordial region, especially in children. Deep depressions of the lower sternal region, and of the ribs in

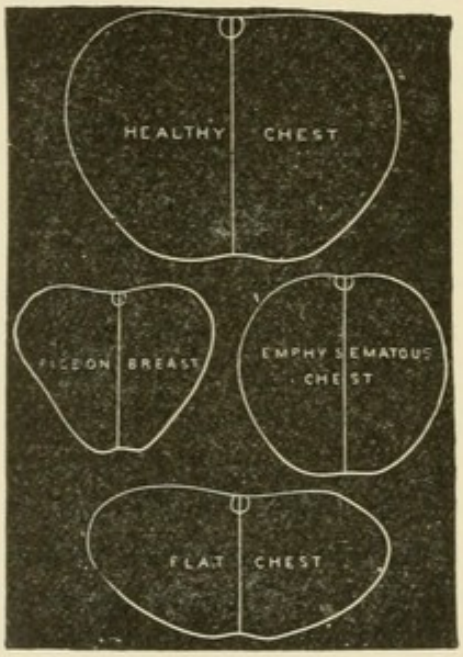

FIG. 3.—TRANSVERSE OUTLINES OF CERTAIN FORMS OF THE CHEST (THOMPSON).

rare instances, occur in healthy individuals. I have a cast taken from life, which shows a depression of the lower sternal region from an inch and a half to two inches in depth; yet the individual from whom it was taken enjoyed perfect health.

Most deviations from symmetry in the two sides are due to slight curvatures of the spinal column. In the examination of a large number of patients, not more than one in seven will be found with a perfectly symmetrical chest.

In health, the respiratory movements are repeated sixteen to twenty times a minute in adults, and from twenty to twenty-five or even thirty times in children.

Considerable difference in the form and in the movements of the chest exists in persons of different ages and sexes. In women the upper

portion is more prominent than in men. The respiratory movements vary accordingly, being more marked at the upper part in women, at the lower part in men. This disparity is most conspicuous in rapid respiration. In children of either sex, the chest walls often hardly move at all; and respiration seems to be performed by the abdominal muscles. The respiratory movements in these three localities are named superior-costal, inferior-costal, and abdominal breathing.

The movements of the chest may be altered considerably, irrespective of pulmonary or cardiac disease. In health, the respiratory movements are readily accelerated by active exercise, and in hysterical patients they are nearly always rapid and superficial, being confined mostly to the upper part of the chest. In persons suffering from some diseases of the brain the respiratory movements become slower and slower until they may not exceed three or four per minute. In hemiplegia the respiratory movements are incomplete or wanting, on the affected side of the chest.

Pregnancy, ascites, or large abdominal tumors cause pressure on the diaphragm, and consequent interference with respiration. The pain of peritonitis compels the patient to restrain the movements of the abdominal muscles, and thus confines the respiratory movements to the chest and renders them deficient and consequently more frequent.

Often among the first signs noticeable on inspecting a patient with disease of the intra-thoracic organs are pallor, cyanosis, icterus, pityriasis, dropsy, and subcutaneous emphysema.

Pallor of the surface and emaciation are seen in chronic pulmonary disease. Pallor also results from fatty degeneration of the heart, and, in some cases, from mitral disease.

Cyanosis, more or less marked, indicates incomplete oxidation of the blood, due to obstruction of the air passages or to diminution of breathing surface; also to affections of the heart, such as congenital malformations or valvular disease. Occasionally this sign results from interference with the descent of the diaphragm by disease of the abdominal organs.

Icterus is found in bilious pneumonia and in the later stages of those cardiac diseases which cause congestion of the portal circulation.

Pityriasis is often found with phthisis pulmonalis, but it also occurs with other diseases, and sometimes even in apparently healthy individuals.

Dropsy due to recent *renal* disease usually shows itself first in the lower eyelids, and subsequently disappears from this locality, to appear in the lower limbs and in the backs of the hands. Dropsy due to *cardiac* disease usually appears first over the instep, and gradually extends upward, involving the limbs, trunk, and serous cavities.

Subcutaneous emphysema may be caused by internal or external injuries of the larynx, the trachea, or the lungs. Air escaping from the

larynx or the trachea causes emphysema in the region of the throat. Rupture of the air cells from over-distention, as in croup, diphtheritis of the larynx, whooping-cough, bronchitis in children, and emphysema in the aged, causes subcutaneous emphysema, which appears first in the areolar tissue of the neck, and subsequently extends to the chest. The air in these cases finds its way into the mediastinum, and thence to the neck. Subcutaneous emphysema from external injury appears first on the chest.

Alterations in the form and in the movements of the chest may be most advantageously studied when grouped together as they occur in different thoracic diseases. First, let us consider the modifications found in pleurisy.

Pleurisy is divided into three stages: first, a dry stage; second, a stage of liquid effusion into the pleural sac; third, the stage of resolution or absorption. In the first stage we find decubitus upon the sound side; respiratory movements rapid, short, and catching.

In the second stage we usually find movements of the affected side diminished, and intercostal depressions less marked than in health; impulse of the heart displaced to the right or to the left, according as the left or the right pleura is distended.

In the third stage, the signs of the second stage gradually subside.

Sub-acute pleurisy manifests the same signs as acute pleurisy, with excessive exudation.

Chronic pleurisy at first manifests signs which do not differ from those of the second stage of acute pleurisy. After absorption or evacuation of the liquid takes place, the affected side becomes retracted and flattened; the shoulder is depressed; the inner border of the scapula projects like a wing and respiratory movements are limited.

In pulmonary emphysema, on first sight of the patient we notice a dusky hue of the countenance, often a sunken condition of the cheeks, marked general emaciation, and more or less turgescence of the superficial veins of the neck and upper extremities. The nostrils dilate on inspiration, and there is a peculiar drawing downward of the corners of the mouth. There is elevation and drawing forward of the shoulders, with anterior curvature of the spine, giving a young patient the stooping appearance of old age.

Inspection generally reveals the peculiar form known as the barrel-shaped chest. In this condition the antero-posterior diameter of the chest is increased (Fig. 3), its surface is rounded, and the upper anterior portion stands out considerably beyond its normal plane. Laterally, the diameter is diminished, and its inferior portion, in the region of the false ribs, is more or less retracted. The elevation and drawing forward of the shoulders cause the neck to appear unusually short. The scaleni and sterno-cleido-mastoid muscles are hypertrophied and prominent so that they stand out like tense cords, resulting from excessive use

of these muscles which elevate and fix the anterior and upper part of the thorax.

Inspiration is short and quick, followed by prolonged and sometimes labored expiration. With inspiration, the anterior and superior portions of the chest are lifted as though composed of a single bone, and there is apparently no anterior or lateral expansion of the chest walls, because the ribs are already rotated as far as their articulation with the spinal column will permit. The ribs have less obliquity, forming with the costal cartilages more obtuse angles than in the normal chest.

The intercostal spaces above are much wider than usual, but at the lower, lateral portion of the chest the ribs are closer together than in the normal condition, sometimes even to the obliteration of interspaces. In well-marked cases there is generally with inspiration retraction of the inferior ribs instead of lateral expansion. This falling in of the thoracic walls is not noticed if the disease is slight.

Sometimes we meet with local emphysema, where a single lung or only one lobe is affected. In such instances we notice local bulging of the chest, with loss of motion.

In extreme emphysema the anterior margin of the left lung overlaps the heart, so that the apex cannot strike the chest wall, hence no impulse can be seen. In milder cases the impulse may be seen closer to the sternum than in health.

In pneumonia, upon first glance we generally notice a dusky flush of the cheek and accelerated respiration. Inspection of the chest shows diminished motion over the diseased organ. This loss of motion may be marked, but is seldom or never complete.

In pulmonary phthisis, the signs obtained by inspection are of considerable value. If the case is advanced the chest wall over the diseased lung will be depressed and its movements restricted, in phthisis more apt to occur at the apex, and contrary to the general belief, quite as commonly upon the right as upon the left side. These phenomena are due to local shrinkage and loss of pulmonary elasticity.

In pneumothorax we observe distention of the chest, proportionate to the tension of the air or gas in the pleural sac, and a corresponding loss of motion.

With great distention there will be no motion of the lower ribs, but prominence of the spaces between them.

Exceptional.—In some rare cases of this disease the upper portion of the affected side seems to move more than the corresponding part of the sound side.

This is due to the extreme efforts on inspiration by which the superior ribs are lifted directly upward as in emphysema, though there is little or no anterior expansion.

Hydrothorax presents a condition, on both sides, similar to that found in pleurisy with effusion upon one side; hence loss of motion and more or less bulging of the infra-axillary regions.

Pericarditis, if the amount of effusion is sufficient, causes considerable bulging of the præcordial region, especially in children; but in older patients, on account of the firmness of the cartilages, this is not so likely to occur. There is also diminution of the respiratory movements on the left side, due to pressure from the distended pericardium.

Cardiac hypertrophy also occasions local bulging, most marked in young patients. The impulse, if visible, will be seen to the left, below its normal position. Its area will also be increased.

Tumors within the thoracic cavity cause bulging when of sufficient size to press upon the parietes. If the tumor be aneurismal or solid and rest upon a large artery, it will usually pulsate synchronously with the contraction of the heart. An enlarged liver or spleen may occasion local bulging.

In cases of pneumothorax and pleurisy with great effusion, we obtain valuable information by examining the impulse caused by the apex of the heart, which will be seen crowded from its normal position toward the unaffected side.

In membranous croup, œdema glottidis, foreign bodies or morbid growths in the larynx or in the trachea, the amount of air entering the lung is considerably less than normal. This has the effect of prolonging inspiration and rendering it laborious, though expiration is not notably affected. Here the respiration is not quickened as in most pulmonary diseases, and it may be even slower than usual. This differs from emphysema in that here there is obstruction to inspiration; in emphysema, the principal interference is with expiration.

When the obstruction in the larynx or trachea is considerable, we observe sinking in of the soft parts of the chest above the clavicle and in the intercostal spaces, especially at the lower part of the chest, during inspiration. This is due to atmospheric pressure from without, as the chest walls expand more rapidly than air can enter through the obstructed passage to fill the lungs.

In chronic bronchitis the signs obtained by inspection are of little value, though we may occasionally observe prolonged expiration, and in some instances irregular expansion of the chest, in different parts, due to plugging of the bronchial tubes by secretions.

PALPATION.

Palpation consists of physical exploration by the sense of touch, either with the tips of the fingers or the palms of the hands.

In practising palpation upon the chest, the palmar surface of the hands should be used, and in many instances it is desirable to cross the hands so that, as one sits in front of the patient, the right hand rests upon his right side and the left upon his left side. If the signs are only slight, we thus appreciate them more clearly.

By the sense of touch we appreciate slight alterations in the movements of the heart and thoracic walls; we sometimes detect the presence of intra-thoracic tumors which cause no bulging of the surface, and determine their nature, whether hard, soft, or pulsating; and we may differentiate between the pain of intercostal neuralgia and that of pleurodynia or pleurisy.

The information regarding size, form, and movements obtainable by this method is essentially the same as that furnished by inspection.

NORMAL VOCAL FREMITUS is a peculiar vibration which will be felt if the hand be gently placed upon the chest of a healthy person while he is speaking. It is produced by the transmission to the chest wall of the vibrations of air in the bronchi, caused by the act of speaking. Modifications of vocal fremitus are among the most important signs which are obtained by palpation.

The normal vocal fremitus varies in different individuals. It is not usually marked in women and children. In males it will be found more or less defined in proportion to the pitch or force of the voice. Voices of low pitch cause a more distinct fremitus than those which are higher. The distinctness of this sign also depends upon the thickness of the chest walls, the diameter of the bronchi, the proximity of the bronchi to the parietes, and the distance of the point examined from the larynx. It is therefore more marked upon the right than upon the left side, and in the infra-clavicular region than in the lower part of the chest.

In women, this sign may be obtained over the upper portion of the chest, but is seldom found over the lower part. In men it is usually perceptible over the whole chest.

Normal vocal fremitus may be increased, diminished, or abolished by disease. As a rule, it is increased by all diseases causing consolidation of lung tissue, as *phthisis*, *pneumonia*, *œdema*, and *apoplexy* of the lungs. It is generally increased by *dilatation of the bronchial tubes*, in which case there is more or less induration of the parenchyma of the lungs.

Exceptional.—In pneumonia, when the bronchial tubes are completely filled by the inflammatory deposit, vocal fremitus cannot be felt.

Owing to the great variation of this sign in different individuals and to its mutations in disease without clearly defined causes, it is not of very much value when taken alone.

Vocal fremitus is diminished or suppressed by any disease causing separation of the lung from the chest wall by the intervention of air, gas, or fluid. In *pneumothorax*, *hydrothorax*, and *pleurisy* with effusion, absence of vocal fremitus over the air or the fluid is a sign of great value.

Exceptional.—Presence of vocal fremitus is not always a certain sign that fluid does not exist, as shown by a few rare cases. If there is but a small collection of air or fluid in the pleural sac, vocal fremitus may be simply diminished; and in multilocular pleurisy it remains over the bands of adhesion.

In emphysema, vocal fremitus is diminished.

Aneurismal or other intra-thoracic tumors cause diminution or absence of vocal fremitus directly over them, providing no lung tissue intervenes between the tumor and the chest wall.

Vocal fremitus is principally of value in differentiating between consolidation of lung tissue and fluid in the lower part of the chest. When lung tissue is consolidated, fremitus is increased, but when there is a collection of fluid, it is absent. Exceptions to this rule are unimportant.

FRICTION FREMITUS, vibration caused by rubbing together of the roughened surfaces of the pericardium or pleura, is indicative of inflammation, with exudation, which causes roughening of the serous surface.

Bronchial or rhoncal fremitus is the term applied in acute or chronic bronchitis, especially in children, when secretion is abundant, and the chest walls are thrown into vibration by air bubbling through fluid within the bronchi. The vibrations communicate to the hand a distinct bubbling sensation, which cannot be mistaken.

Fluctuation of fluid within the pleural cavity may often be felt in the intercostal spaces by the fingers while tapping at a little distance with the fingers of the other hand.

MENSURATION.

Mensuration is rarely used, since inspection and palpation give sufficiently accurate and more quickly obtainable knowledge of the signs furnished. Many instruments have been devised for determining the size, capacity, and degrees of curvature or flatness of the chest. The only measurement of special clinical value is that of the circumference, in inspiration and in expiration, which may be readily taken by means of a simple tape.

A good device for this consists of two tapes joined at their extremities and so padded near the line of junction as to form a sort of saddle, which rests upon the spinous processes and prevents slipping. In using this instrument, adjust the pads to the spine and carry the tapes about the chest on both sides to the median line in front. The exact amount of motion of the two sides may thus be easily ascertained.

In measuring with a single tape, place the thumb nail at a certain point on the tape, the first finger about one-fourth of an inch nearer its end. Then press the tape with the thumb nail against the middle of a spinous process and press the forefinger down beside it. This enables one to hold the tape firmly in position, and, by preventing the skin from sliding in respiration, gives a fixed point from which to measure. It is always desirable to mark the median line in front before commencing this measurement.

The circumference of the chest may be taken above or below the nipples, but best on a level with the sixth costo-sternal articulation. In recording cases, always note the level of the measurement.

The measurement should be taken during both full inspiration and forced expiration, and the two results should be compared to determine the expansion. The two sides must be compared to ascertain whether either is distended or deficient in movement. Quain and Carroll invented very satisfactory instruments for taking these measurements, known as stethometers. Quain's instrument (Fig. 4) consists of a cylindrical box with a dial and an index, moved by a rack to which is attached a cord long enough to compass the chest. Each rotation of the index about the dial indicates one inch of movement. The box is placed upon the centre of the chest in front, and the string is carried horizontally around the chest; as the patient breathes, the index revolves about the dial, registering accurately the expansion of the chest walls. Carroll's stethometer is simple and exact (Fig. 5). Ordinarily a simple tape is sufficient.

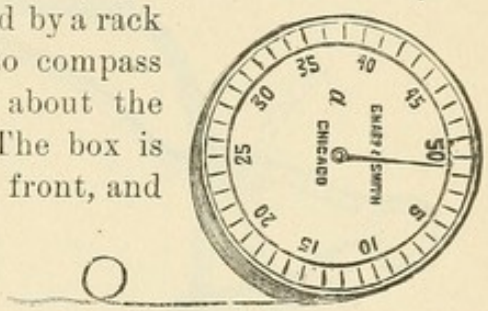

FIG. 4.—QUAIN'S STETHOMETER.

Measurements of the healthy chest, of course, vary in different individuals. The average in men is thirty-two and one half inches. Generally, the right side exceeds the left by half an inch, but in left-handed persons the reverse is true.

In disease, the affected side may be distended or contracted, and its movements may be diminished or increased, conditions usually noticeable on inspection and by palpation, but it is not uncommon to find, upon mensuration, that a side which had the appearance of distention is

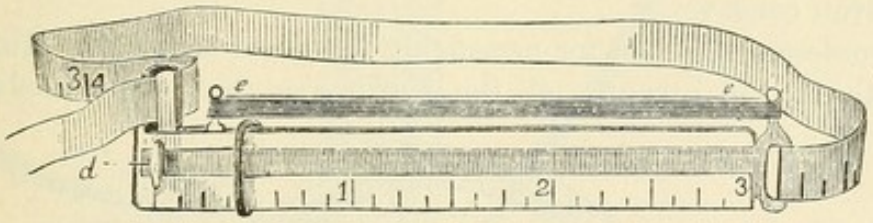

FIG. 5.—CARROLL'S STETHOMETER.

smaller than its fellow; frequently expansion, which has seemed comparatively free, will be found by the tape not to exceed one-eighth of an inch.

The diseases causing expansion or contraction, and loss of movements of the chest wails, were mentioned under inspection.

The transverse diameter of the chest may be obtained by means of a pair of calipers, or by Flint's cyrtometer (Fig. 6).

Gee's cyrtometer, consisting of two pieces of composition gas-pipe joined together by means of a piece of rubber tubing, is the cheapest and perhaps the best instrument for ascertaining the transverse outline of the chest. In using it, the joint is placed upon the spine, and the pieces of pipe are accurately moulded round the chest. The instrument

is then removed and laid on paper, when an exact tracing can be made. In a well-formed chest, the antero-posterior diameter will be to the transverse diameter in men as three to four, in women as four to five (Fig. 3). Scott Allison invented an instrument, known as a stethogoniometer, for measuring the curves or the flatness of the surface of

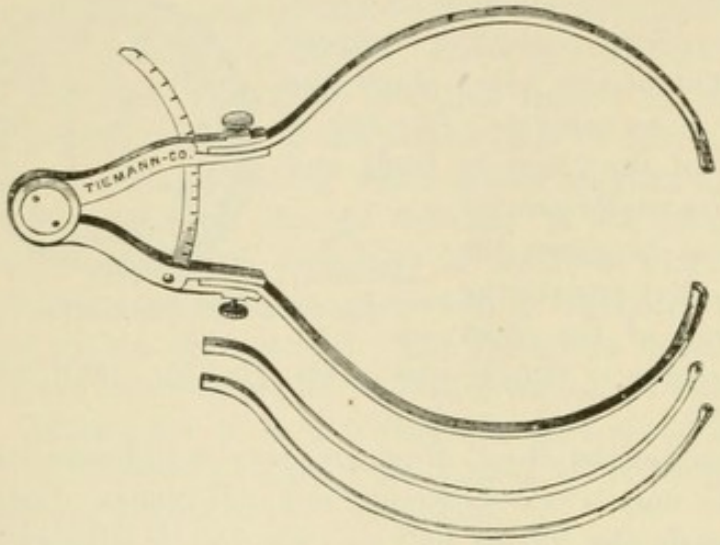

FIG. 6.—FLINT'S CYRTOMETER.

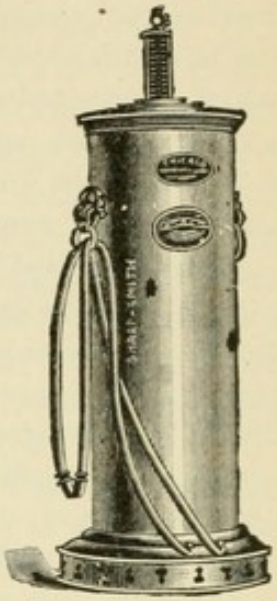

FIG. 7.—SPIROMETER.

the chest (Fig. 8). It has been claimed that the infra-clavicular space should always be convex in health. With this instrument the curvatures could be accurately ascertained, but unfortunately the information is of very little value, because, in healthy individuals, this region is often flat or even concave.

Spirometers are used for measuring the chest capacity. Hutchinson was, I think, the inventor of the spirometer, but many modifications

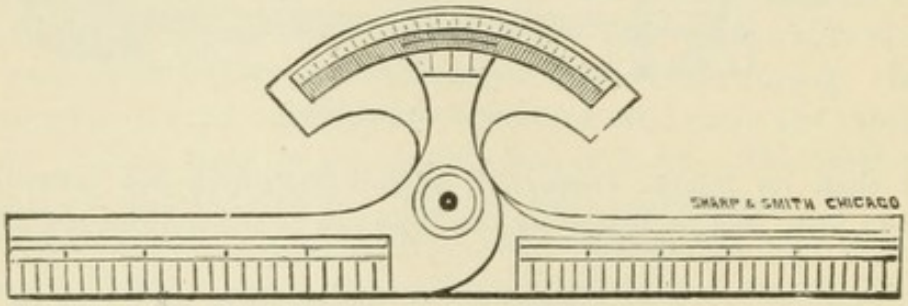

FIG. 8.—ALLISON'S STETHOGONIOMETER.

have been devised. Recently portable instruments about the size of a watch have been made. In one of these, as the patient inspires, or blows into the tube, the index revolves on the dial, registering the number of cubic inches of air inhaled or expired.

Hutchinson found that people five feet in height usually possess a vital capacity of one hundred and seventy-four cubic inches, and for every inch of height above five feet, eight cubic inches should be added

to the healthy standard. There are many obstacles to the use of spirometers rendering them practically useless. For instance, it takes most persons some time to learn how to blow into one of these instruments. A patient may at one time expire only one hundred and fifty cubic

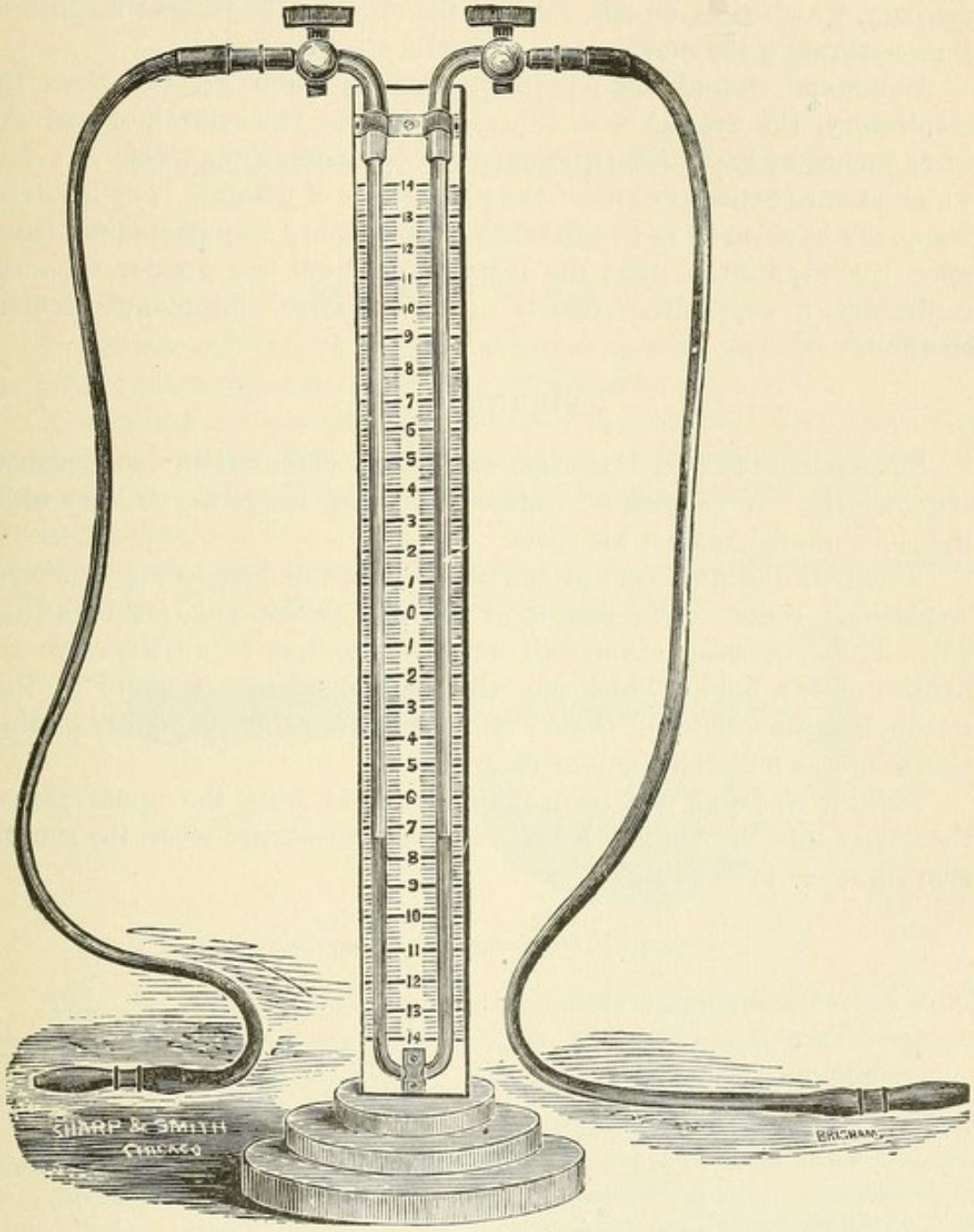

FIG. 9.—HAMMOND'S HÆMADYNAMOMETER.

inches, and at another, without any change of health, two hundred cubic inches. Furthermore, women and men, the young and the old, all have different vital capacities, and it has not yet been possible to arrive at an accurate healthy standard.

Hammond devised the hæmadynamometer, which he used for measuring the force of inspiration and expiration. He found that individ-

uals five feet eight inches in height possess the maximum respiratory power. His instrument (Fig. 9) consists of a bent glass tube fastened to a graduated scale, and joined at each end by a rubber tube, through which the patient is to breathe. The instrument is partially filled with mercury, which rises on one side or the other as the patient inspires or expires through the mouth-piece and falls after he ceases.

Hammond found the expiratory power much greater than the inspiratory, the average man being able to raise the column of mercury three inches by expiration, and only two by inspiration. This is a fact which at once explains some of the phenomena of disease. For instance, Laennec's hypothesis as to the cause of pulmonary emphysema was based upon the supposition that the inspiratory power was greater than the expiratory, a supposition clearly untenable after Hammond's demonstration.

SUCCUSSION.

Succussion, the fourth method of physical exploration, was known to Hippocrates. It consists of suddenly shaking the patient's body while the ear is placed against his chest.

When air and fluid occupy the pleural sac, this proceeding will cause a splashing sound. The sign is of value in pneumo-hydrothorax (Fig. 26). The succussion sound will vary more or less in quality with the density of the fluid. Thick pus will not yield the same sound as thin serum, but the quality of these sounds is not usually sufficiently distinctive to aid us materially in our diagnosis.

Metallic tinkling, due to dropping of fluid from the upper part of the cavity into the effusion below, can usually be heard when the succussion signs are present (Fig. 26).

CHAPTER II.

METHODS OF EXAMINATION.—*Continued.*

PERCUSSION.

PERCUSSION IN HEALTH.

PERCUSSION is the art of eliciting sound by striking with the fingers, or with instruments constructed for the purpose.

As a means of diagnosis, it is generally supposed to have originated during the last century with Avenbrugger, a physician of Vienna, but the works of Hippocrates indicate that he was familiar with it, to a limited extent.

Hippocrates and Avenbrugger recommended *immediate percussion*, in which the blow is struck directly upon the chest wall.

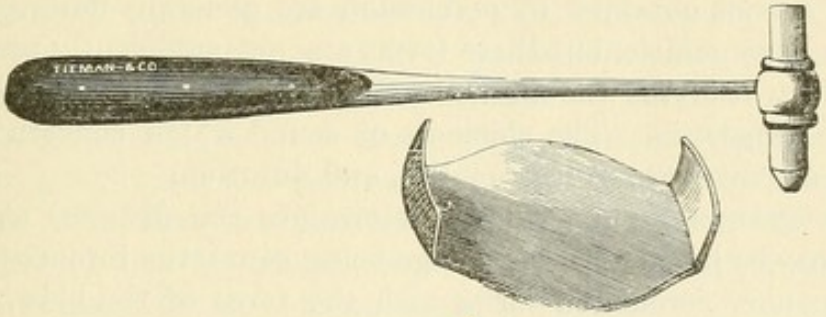

FIG. 10.—FLINT'S HAMMER AND PLEXIMETER.

This form of percussion has been nearly supplanted by one which originated about sixty years ago, with M. Piorry, termed *mediate percussion*, in which the blow is received on some intervening substance.

Before mediate percussion was employed, it was quite essential to intensify the sounds; this was accomplished by placing the patient with his back against a hollow wall. In some women the signs elicited by immediate percussion are quite distinct over the upper part of the chest, but usually this method is very unsatisfactory.

In mediate percussion, a small hammer or plexor and an instrument known as a pleximeter or plessimeter are employed. The hammer in common use consists of a cylindrical rubber head attached to a light handle about eight inches in length. Metallic hammers faced with rubber, as sometimes used, are objectionable on account of their weight, which renders the blow so forcible that it is apt to cause pain.

Pleximeters are made of various materials, as rubber, bone, wood, ivory, or leather. Some of them are graduated in order that they may be used in mensuration. Among the best is one which consists of a narrow oval disc of hard rubber, with large ears at each extremity.

It should be narrow enough to be placed between the ribs, and should have a large projection at each end, that it may be firmly grasped. I have frequently used a small cylinder of soft rubber about two inches long and half an inch in diameter. It has the advantage of being easily adapted to the intercostal spaces, and of emitting no sounds of its own when struck.

For ordinary percussion it is best to use the middle or index finger of one hand in place of the pleximeter, and two or three fingers of the other, with their tips brought into line, as a hammer. The fingers used as a plexor should be brought as nearly as possible to a right angle at the second joint, that the terminal phalanges may strike perpendicularly upon the finger of the opposite hand.

When the fingers are used, there is noticeable a certain sense of resistance which is not obtained with instruments. Often this would enable us to detect internal organic changes even with our ears completely stopped. So valuable is it in intricate cases that, when there is difficulty in making an accurate diagnosis, I always employ the fingers instead of instruments for percussion.

The sounds obtained by percussion are generally described as clear, dull, and tympanitic, but these terms are not sufficiently precise to aid us much in studying the method. I prefer a classification based upon acoustic properties. The elements of sound which concern us in percussion are intensity, pitch, quality, and duration.

THE INTENSITY of a sound determines the distance at which the sound may be heard. Other things being equal, the intensity of a sound in pulmonary percussion varies with the force of the blow, the volume of air in the lung, and the thickness and elasticity of the chest walls. It is diminished by thick layers of fat or muscle, by rigidity of the costal cartilages, and by contraction or consolidation of the lung, and it is increased by the opposite conditions.

THE PITCH of a percussion sound may be high or low. Those familiar with music will understand this, but a common mistake is to confound pitch with intensity. Many students suppose that the higher the pitch, the greater the intensity. The reverse of this is usually true in pulmonary percussion, intense sounds being low pitched, and high-pitched sounds possessing feeble intensity.

This difference between pitch and intensity can be easily recognized by striking two notes at opposite ends of the keyboard of a piano. By striking a high note forcibly, one will obtain a sound loud enough to be heard some distance; then by gently tapping a key at the other end, one will obtain a sound heard at exactly the same distance, but of a much lower pitch.

The pitch of the percussion note over a healthy lung is always low,

but it will vary in different portions of the chest, owing to difference in the volume of air and the position of other intra-thoracic organs.

QUALITY of sound is that element by which we distinguish between the tones of musical instruments, or of voices of different individuals, having, it may be, the same intensity and pitch.

In pulmonary percussion, we obtain a peculiar quality termed vesicular, impossible to describe, but always to be obtained by percussion of the healthy chest. It is soft and low in pitch, and usually seems as though coming from a point a couple of inches beneath the surface. It can be learned only by studying the healthy chest.

DURATION of the healthy percussion note depends upon the same causes as its pitch. If its pitch is high, the duration is short; if the pitch is low, the duration is prolonged. Indeed, a definite relation exists between all these different elements; that is, intense sounds are apt to be low pitched; those which are feeble are generally short and high pitched, and, instead of the vesicular, they possess a solid character.

Percussion seems very simple as practised by an adept, but accuracy is not acquired without much practice.

Certain rules essential to accurate percussion should be early fixed in mind.

The surface of the chest should be bare; but if for any reason this cannot be secured, have the covering soft, thin, and smooth. It is absolutely useless to percuss the chest of a patient who has on one or two shirts and perhaps a chest protector or corset.

The patient should be in a comfortable position, whether sitting, standing, or lying upon the back, and the two sides must be relatively symmetrical. The first two positions are preferable, but very sick patients should not rise for the examination; it will be better to make a less critical examination than to endanger the patient.

Persons suffering from diseases which cause feebleness of the heart should not be asked to sit or stand. Illustrating the importance of this caution, I have seen cases of sudden death from overtaxing of a weak heart, by slight exertion, such as the getting out of bed of a patient convalescing from pneumonia or diphtheria.

Do not allow the patient to twist the body or move the arms during percussion, as such motions change the relations of the muscles, and thus alter the percussion note.

The physician's ear should be squarely in front of the part percussed. If he stand partially to one side, the signs obtained on that side, even though the same as those on the other, will reach the ear with a different tone. His position should be easy and unrestrained, or he will not recognize slight differences in sound.

In percussing any particular region of the chest, aim to have the chest walls as thin and tense as possible. To secure this on the anterior portions of the chest, the arms should hang at the sides and the shoulders

should be thrown backward. In examining the lateral regions, it is well to have the hands rest upon the head. If the arms are held up, the muscles stand out so prominently that they interfere with obtaining the pulmonary resonance. In percussing the posterior regions, the trunk should be bent forward and the arms crossed in front.

In percussing the chest, compare corresponding portions of the two sides. If changes from the normal are slight, they can be detected in no other way. Ordinarily it is sufficient to repeat a series of strokes first on one side, then on the other, or to percuss both sides repeatedly in quick succession. However, the percussion sounds vary slightly at different periods of the act of respiration; therefore, whenever the changes are so slight as to require great care for their discrimination, the sides should be compared during the same stage of the respiratory act. The best period at which to make the comparison is at the close of a forced expiration.

Exceptional.—In health the two sides are not always alike as regards disparity between the note elicited in full inspiration and that elicited in forced expiration.

In applying the finger or the pleximeter, be careful that it presses evenly upon the surface and displaces all the air beneath it. Otherwise, the resonance of the pleximeter is obtained instead of that from the chest, and at the same time the air is suddenly forced out, causing a sound very similar to cracked-pot resonance.

The force of the stroke should be moderate, never great enough to cause the patient pain, and alike on both sides. In percussing superficial portions of the lung, the stroke should be very gentle, but to obtain the resonance from deeper parts it must be more forcible. Beginners commonly strike much too hard.

The stroke should be from the wrist alone, whether made with the hammer or with the finger. When striking from the elbow, we cannot control the force of the blow. Some diagnosticians are accustomed to strike a single blow, first upon one side, then upon the other; but I get better results by making three or four taps in rapid succession.

The direction of the stroke should always be perpendicular to the surface of the chest. If we percuss obliquely, instead of obtaining the resonance from the lung immediately beneath the pleximeter, we get that from a rib or from more distant tissue.

In percussing near the sternum, in the upper portion of the chest, we obtain resonance from the trachea instead of from the lung, unless care be taken to direct the blow toward the central portion of the apex.

The stroke should be a simple tap, the finger or hammer being allowed to rebound instantly, instead of resting a moment on the pleximeter, which has an effect on pulmonary resonance similar to that produced by touching a vibrating tuning-fork. In percussing with the fingers, strike with their tips, instead of with the pulps.

As the signs in a healthy chest vary in its different regions, we must take special pains to familiarize ourselves with all the healthy sounds. There are no two healthy people whose chests are exactly alike, therefore we can take no one person as a standard for comparison; but after percussing many healthy chests, we may form an ideal standard from which no great variation can occur without indicating disease.

Normal vesicular resonance is obtained most perfectly in the left infra-clavicular region; and this, being the sound obtained over the pulmonary air vesicles, is taken as the standard for comparison in pulmonary percussion.

In the right infra-clavicular region the percussion note is nearly the same as in the left, but is slightly harder or more tubular in quality, owing, probably, to the greater size of the bronchial tubes.

In the middle of the supra-clavicular region the resonance is soft or vesicular in quality, but toward the inner part of this region it becomes harder in quality or tubular and higher in pitch. Austin Flint called this an approach to tympanitic resonance. Externally in this region the vesicular quality is diminished. In percussing over the central portion of the clavicular region, the sound is fairly vesicular, but it becomes less and less so toward either end of the clavicle.

In the mammary regions the sounds are altered on one side by the presence of the heart, and on the other side by the presence of the liver (Fig. 1). In the upper part of the right mammary region we obtain vesicular resonance extending down to the line of hepatic dulness in the fourth interspace. Below this, where the lung overlaps the liver, dulness is appreciable on forcible percussion, gradually becoming more and more distinct as the lung decreases in thickness, until we reach the lower border of the lung at the sixth rib, the line of hepatic flatness, below which we lose all pulmonary resonance.

The lines of hepatic *dulness* and of hepatic *flatness*, the first along the upper margin of the liver, the second at the lower margin of the lung, are ordinarily about two inches apart.

Exceptional.—In deep inspiration the lower line may be carried an inch and a half or two inches lower, and in forcible expiration it may be elevated from one to five inches; therefore the area of hepatic dulness, between the two lines, may vary from two to seven or even eight inches. This wide range is not common, but its occasional occurrence shows the necessity for studying the chest in both inspiration and expiration.

In the left mammary region pulmonary resonance exists over the outer part. Near the middle of the region forcible percussion elicits cardiac dulness. Near the sternum the heart is superficial, covered only by the pericardium and by cellular tissue; here there is a small, triangular space yielding flatness. It is about an inch and a half wide at its base, which corresponds to the sixth rib, and extends from the fourth

to the sixth costal cartilage. The apex of this triangle is located at the margin of the sternum on a level with the fourth rib.

The resonance of the mammary region is modified more or less by the thickness of the muscles in men and by the mammary glands in women.

In the infra-mammary region, on the right side usually, there is nothing but the liver to affect the percussion note, hence we have a sound termed flatness, like that obtained by percussing the thigh. If the colon be distended by gas, we obtain tympanitic resonance in the lower part of this region.

In the left infra-mammary region flatness caused by the left lobe of the liver extends a couple of inches to the left of the median line. In the outer portion of this region we obtain a similar sound from the spleen, and between these two organs we elicit tympanitic resonance from the stomach.

In the upper sternal region, as low as the level of the second costal cartilage, the sound is tubular, or, according to Flint, tympanitic. This is due to the presence of the trachea, the sounds of which are modified by the anterior borders of the lungs which are in apposition throughout this region. Below the level of the second ribs, on light percussion, pulmonary resonance may be heard, though modified by the timbre of the bone. But deep percussion gives dulness, resulting from the presence of the great blood-vessels.

Over the lower sternal region, by light percussion, pulmonary resonance is obtained to the right of the median line, while on forcible percussion there is dulness. Left of the median line, the heart is superficial and yields flatness. At the inferior portion of this region, flatness is due to the left lobe of the liver.

Over the scapula, the vesicular sound is indistinct from the thickness of the muscular tissue, but above the spine of the scapula it is much more marked than below, and in the upper part of this region it is quite clear.

In the inter-scapular regions the sounds are hard in quality and high pitched, because the chest walls are thick. There is, however, in all cases some pulmonary resonance. The pitch is a trifle higher on the left side on account of the aorta.

In the infra-scapular regions the vesicular resonance is well defined, though not quite so clear as in the infra-clavicular region. It extends downward to the tenth or eleventh rib. On the right side we find the line of hepatic dulness at the eighth rib and the line of hepatic flatness at the eleventh rib; but these vary from one to two inches during forcible respiration (Fig. 2).

On the left side the resonance is slightly modified near the spine by the nearness of the liver. Below the tenth rib the intestinal canal, if filled with gas, causes a tympanitic sound. In the outer part of this

region, between the ninth and eleventh ribs, dulness is obtained over the spleen, and for a short distance about this dull region resonance is rendered more or less tympanitic by the stomach and intestines. In the lower part of the left infra-scapular region, close to the spinal column, dulness is found over the kidney, and it occurs in a similar position, though a trifle lower, on the right side.

In the axillary regions the resonance is often more marked than in the infra-clavicular.

In the infra-axillary region the resonance is modified on the right side by the liver, and upon the left by the stomach and spleen.

In this region the margin of the lung passes obliquely downward and backward from the anterior boundary near the sixth rib to the posterior near the tenth rib. On the right side, hepatic flatness is found below this line, and hepatic dulness a couple of inches higher. On the left side, below this line, we find tympanitic resonance in front over the stomach, and dulness posteriorly over the spleen. In this locality the pulmonary resonance is often modified by the stomach, as high as the fourth rib.

The size of the spleen varies considerably, even in health. The area of dulness which it causes seldom exceeds two and one-half inches in height by about four inches in width; about half of this dull space is in the infra-scapular and half in the infra-axillary region.

Exceptional.—In rare cases the spleen rises as high as the lower boundary of the axillary region, or the stomach may yield decided tympanitic resonance as high as the fourth rib.

In the infra-scapular region, upon the right side in children, dulness is sometimes very pronounced, due to the disproportionate size of the liver in early life. This is not infrequently mistaken for the consolidation of pneumonia.

The percussion sounds in different regions of the chest are modified by age, sex, and various idiosyncrasies. In old age, the chest walls are less elastic than in middle life, and the lung has undergone some change which renders the sounds harder in quality and higher in pitch. In children, the lungs are very resonant, and the costal cartilages are elastic; consequently we obtain a low-pitched, intense vesicular sound. In men the percussion note over the upper portion of the chest is not usually so resonant as in women, but it is more distinct over the lower portions. It will be seen, from what has already been said, that there is notable dissimilarity of the percussion sounds on the two sides in the mammary regions, as also in the infra-mammary, infra-axillary, and infra-scapular regions. In all other portions of the chest the resonance is nearly identical on the two sides, but the slight normal disparity in the infra-clavicular regions is a point of great importance.

PERCUSSION IN DISEASE.

In disease, the percussion sounds may occur in every gradation from normal to tympanitic resonance or flatness. These varieties have been variously classified. R. E. Thompson classifies them as clear, dull, tympanitic, amphoric, and cracked-pot resonance. Flint arranged them under six heads; and A. L. Loomis under seven, as follows: Exaggerated pulmonary resonance, dulness, flatness, tympanitic resonance, vesiculo-tympanitic resonance, amphoric resonance, and cracked-pot resonance, or the cracked-metal sound.

EXAGGERATED PULMONARY RESONANCE differs from the normal vesicular sound only in its intensity. The pitch and quality are the same as in health, but the intensity is increased. This sound is obtained over lung tissue which is receiving more air than usual, and which might therefore be said to be in the highest degree of health.

The sign is therefore only negative, as it is indicative of no disease whatever in the place where it is obtained, but rather points to deficient action in some other part of the respiratory tract. Exaggerated pulmonary resonance, in adults, is very nearly the same as the normal resonance in children. The sign results from obstruction to the entrance of air into some portion of the respiratory tract, whether from filling up of the air cells by inflammatory exudation as in pneumonia, from narrowing of the bronchial tubes, or from collapse of the air cells. Pneumonia of one lung or of a single lobe of a lung causes exaggerated resonance over healthy portions of the lungs. Compression of the lung from air or fluid in the pleural sac gives rise to exaggerated resonance on the sound side. If one main bronchus is occluded, from causes within it or external to it, resonance is exaggerated on the opposite side. In extreme anæmia exaggerated resonance occurs on both sides, due probably to a diminished amount of blood in the pulmonary circuit. As the chest is practically a cavity with unyielding walls, diminution in its fluid contents must cause a corresponding increase in the amount of air.

DULNESS indicates a small amount of air beneath the part percussed. It can always be obtained in the healthy chest where the lung overlaps the liver. This sign differs from normal vesicular resonance in having high pitch, hard quality, and comparatively short duration. Its intensity is usually less than that of vesicular resonance. Varying degrees of dulness should be carefully studied on the healthy chest. Over the liver, on forcible percussion, slight dulness is found in the fourth intercostal space, becoming more distinct, higher in pitch, harder in quality, and shorter in duration, as examination extends downward, toward the lower margin of the lung.

This sign, when obtained in a position which should yield vesicular resonance, indicates that something has occurred to diminish the normal amount of air in that part of the lung. It is obtained over *consoli-*

dated lung, from simple inflammation or from phthisis, from compression of the lung or from collapse of the air cells; over *collections of fluid* in the bronchi or in the air vesicles; over *moderate exudations* in the pleural sac separating the lung from the chest walls, but effusions of any considerable amount destroy pulmonary resonance entirely, giving flatness. Dulness is also obtained over *intra-thoracic tumors,* whether solid or fluid, provided a small portion of lung tissue containing air intervenes between them and the thoracic wall. It is one of the signs found in *pneumonia, pleuritis, phthisis, atelectasis,* and in *intra-thoracic abscesses, aneurisms,* and *tumors.*

Exceptional.—Dulness results occasionally from pulmonary apoplexy. In such cases it is usually found at the lower angle of the scapula. It may arise from brown induration of the lung, due to a varicose condition of the pulmonary veins. In this disease it is found near the middle of the lungs on both sides. It may arise from enlarged bronchial glands, and in a few instances it is found in bronchitis over the apex of the lungs, or more clearly at the lower posterior part of the chest, due to a collection of secretions within the bronchi.

FLATNESS differs from dulness in complete absence of vesicular resonance. Dulness indicates that there is some air beneath the point at which the stroke is made; flatness, that there is none. Dulness is obtained over that portion of the liver overlapped by lung tissue; flatness over that portion below the sixth rib, which is superficial. Dulness occurs in pleurisy where the exudation has separated the lung a short distance from the chest wall and caused a corresponding diminution in the volume of air. Flatness will be found in the same disease, when an effusion of serum lifts the lung above it, removing all air-containing tissue from beneath the point percussed.

Flatness is found in *pleurisy* with effusion oftener than in any other disease.

Exceptional.—In rare cases of pneumonia the inflammation runs to such a height that not only the air cells, but also the bronchial tubes are filled with the exudation, and in such cases absolute flatness is found over the lung tissue. Again, when the lung becomes completely collapsed from pressure or obstruction of a large bronchus, flatness results.

Tumors or abscesses within the thorax, when they rest against the chest walls, cause flatness.

TYMPANITIC RESONANCE is the name given to the sound which may be normally obtained over the stomach or the intestines when filled with air or gas. It indicates a quantity of air enclosed by walls thin and yielding and not too tense (Da Costa).

Under certain conditions, this sign is met with over the thorax. Tympanitic resonance is usually described as of higher pitch than the vesicular sound. Its duration may be longer or shorter, and its quality is hollow, conveying the idea of more or less tension; it is also somewhat hard, metallic, and ringing. Statements of different authors

conflict concerning the pitch of this sign. Some hold that it is high, others that it is low.

It seems to me that the discrepancy has arisen from mistaking the ringing metallic quality of the sound for a high pitch, when it may really be low. I find the weight of opinion in favor of a high pitch. R. E. Thompson, in his little work on physical examination of the chest, states that the pitch of this sign may be either high or low: high when the tension of the volume of air is great, and low when it is slight.

This variety of resonance is never found in the healthy chest, unless it be transmitted from some of the organs beneath the diaphragm; it is frequently obtained below the fourth rib, on the left side from gaseous distention of the stomach or the intestines and occasionally over the infra-mammary region on the right side when the colon is distended. When obtained over portions of the chest which should yield a vesicular sound, the sign usually indicates a collection of air or gas in the pleural sac, as in pneumothorax. Occasionally it is found over a large cavity in the lung tissue containing air.

Pulmonary cavities are generally produced by phthisis; hence the rule, that there are only two diseases of the chest, pneumothorax and phthisis, in which this sign is found.

Exceptional.—Guttman, Gee, and some others claim that this variety of resonance sometimes results from diminished tension of the pulmonary parenchyma, and may be found in any condition causing partial collapse of the lung.

Perfect tympanitic resonance may be obtained in that very rare condition in which air or gas collects in the pericardium. It is said to be found in some cases of emphysema and of acute tuberculosis. According to Da Costa, it is sometimes found in pulmonary œdema.

Tympanitic resonance from the stomach may be elicited far above its normal seat, when the lung is retracted and the stomach and intestines are correspondingly elevated.

VESICULO-TYMPANITIC RESONANCE is a quality of sound midway between the vesicular and the tympanitic.

This sign occurs in extreme emphysema, where the air cells and the chest walls are distended.

AMPHORIC RESONANCE is a modified tympanitic sound which may be closely imitated by tapping the cheek gently when the mouth is filled with air, but not much distended. The sound is hollow and somewhat metallic. It is obtained in very much the same conditions as cracked-pot resonance—that is, over an empty pulmonary cavity with yielding walls; but to produce this sign the cavity must communicate freely with a large bronchial tube, so that the air can be driven quickly from it by the percussion stroke. It is found also over collections of air in the pleural sac, when this cavity opens through the lung into a large bronchus.

Pulmonary cavities are generally caused by phthisis, but they may

result from abscess. Amphoric resonance is therefore a sign of *pneumothorax, phthisis,* and possibly of *abscess* or *gangrene.*

Bell Sound.—While listening over a large pulmonary cavity, if percussion be made on the opposite side of the chest, with one large coin striking upon another used as a pleximeter, a ringing bell sound will be heard, which is sometimes very loud.

CRACKED-POT RESONANCE (*bruit de pot fêle*) may be imitated by placing the hands loosely together, palm upon palm, and striking upon the knee. It is described as resembling the clinking of coin or the *timbre* of a cracked metallic kettle. Generally the sign seems to be the result of forcing air suddenly from a pulmonary cavity through a *small* opening. It has been considered by some as diagnostic of a pulmonary cavity, but this sign may occasionally be obtained when no cavity exists, and sometimes even in healthy individuals. Something closely resembling this resonance is apt to be heard during percussion if the pleximeter is placed lightly against the surface, so that air remains beneath and is suddenly forced out by the blow.

It is said that occasionally this sound may be elicited in the bronchitis of children, or just above the level of the fluid in pleurisy with effusion.

As a rule, cracked-pot resonance is significant of a cavity, but the majority of cavities do not produce it. When found, it can seldom be heard more than two or three times together, and it requires an interval of rest before it can be reproduced. This is probably due to the small opening into the cavity—the air, having been driven out, returns slowly.

THE PLESSIGRAPH.

In percussion with the ordinary pleximeter, no matter what its material or its form of construction, all the tissue beneath it is thrown into vibration. This renders it next to impossible to define sharply the outlines of dulness when solid tissue is overlapped by the lung, because the pleximeter covers too much space, and the sounds from the tissues containing air and from those which do not are blended. For instance, in attempting to determine the lower border of the lung, overlapping the liver, we commence above and percuss downward to the point of complete flatness, then upward again to a point where the vesicular resonance is clear, and thus back and forth, until two adjacent points are reached where we obtain on the one hand quite perfect pulmonary resonance, and on the other flatness. Then we judge that the border of the lung lies midway between the two.

To avoid throwing too much tissue into vibration, the size of the pleximeter must be abridged ; but as the size is diminished, unless compensated for in some way, the intensity of the sound is correspondingly lessened. These difficulties seem to have been overcome in the construction of a little instrument known as the plessigraph devised by M. Peter, of Paris.

It consists of a small cylinder of wood about four inches in length and five-eighths of an inch in diameter, with a disc at one end upon which percussion is to be made. The other end consists of a truncated cone, the plane surface of which measures nearly an eighth of an inch in diameter. In using the instrument, the

small end is placed on the surface of the chest, and percussion is made upon the other end with the pulp of a single finger. Care must be taken that the instrument is held perpendicular to the surface. On account of the smallness of the surface which rests against the chest, the sound obtained would be very feeble, were it not in a measure intensified by the body of the instrument acting as a sounding-board. Trousseau claimed that it is not necessary to strike upon the disc, but that we may simply tap upon it with the pulp of the finger, and that by means of this instrument even students may rapidly map out the liver or heart, when with ordinary percussion this might be impossible, even for a skilled diagnostician. The instrument as constructed by Peter had upon the side an arrangement holding a crayon which could be pressed down to mark the skin when the border of the organ had been found, so that a dotted line would be left corresponding to the outlines of the solid viscus or tumor. I have found this instrument very satisfactory in determining superficial dulness, so long as it is employed only in the intercostal spaces, but not when applied over the ribs.

AUSCULTATORY PERCUSSION.

Auscultatory percussion was instituted by Camman and Clark in 1840. It consists, as the name implies, of combined auscultation and percussion. In practising it, a stethoscope is needed. For this purpose the originators of the method devised a peculiar instrument, which con-

Fig. 11.—Camman's Stethoscope for Auscultatory Percussion.

sists of a solid cylinder of wood formed at one end into a truncated wedge, and at the other into a disc (Fig. 11). The wedge-shaped extremity is placed in an intercostal space, over the most superficial portion of the organ or tumor to be examined, and the examiner's ear is placed upon the disc. An assistant then percusses from the healthy lung tissue toward the instrument. The moment percussion is made over solid tissue, the changed sound reveals the fact to the listener, and thus enables him to determine the deep outlines of the solid mass much more accurately than by simple percussion. The ordinary binaural stethoscope with the smaller chest-piece may be used for the same purpose. The advantage claimed for this method is that it enables one to determine the outlines of intra-thoracic tumors or organs much more accurately and rapidly than by other means. Outlines of the liver, the spleen, and the kidney may also be ascertained with considerable accuracy, even when ascites is present.

In the practice of this method, a second person has been necessary to make the percussion, and it is often impossible to get a skilled assistant at the time needed. To overcome this difficulty, I have devised an instrument known as the emballometer (Fig. 12). It consists of a hol-

low cylinder about three inches in length by five-eighths of an inch in diameter, within which plays a metallic plunger. To the objective end of the instrument is fitted a soft-rubber chest-piece, against which the plunger strikes. To the other end is attached a rubber tube about eighteen inches in length, connecting it with a rubber bulb. Compression of the rubber bulb drives the plunger against the chest-piece; at the instant the pressure is removed, the bulb expands and the plunger is forced upward by atmospheric pressure. In practising auscultatory percussion by the aid of this instrument, the stethoscope is held with

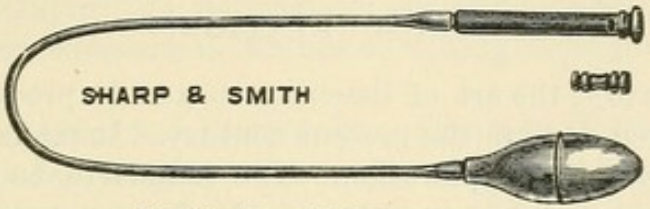

FIG. 12.—INGALS' EMBALLOMETER.

the left hand; the bulb of the emballometer is held in the palm of the right hand by the last three fingers, and the cylinder by the thumb and forefinger. This enables the physician to move the instrument without restraint, to strike any point as rapidly or as slowly as he chooses and with whatever force may be desirable. By means of this little instrument and the binaural stethoscope, auscultatory percussion can be satisfactorily practised without the aid of an assistant. In using the binaural stethoscope for this purpose, the small chest-piece should be employed. Probably one still smaller or flattened, so that it might be applied between the ribs, would give even better results.

CHAPTER III.

METHODS OF EXAMINATION.—*Continued.*

AUSCULTATION.

AUSCULTATION, the art of listening to sounds produced within the chest, originated early in the present century. It ranks first among the methods for physical exploration. The sounds to be studied by this method are produced during either inspiration or expiration, or during both portions of the respiratory act.

Auscultation may be mediate or immediate. In the former, the sounds are conducted to the ear through an instrument known as the stethoscope; in the latter, the ear is placed directly on the surface of the chest, or on the chest but slightly covered.

In this connection, a brief notice of Laennec, the inventor of mediate auscultation, is of peculiar interest. He was born in an obscure province in France, and at the age of nineteen went to Paris to obtain his medical education, where he very soon attracted the attention of the profession by his diligence and attentiveness at the hospitals.

From the time that he entered Paris until his final departure, about five years before his death, his whole life seems to have been given to careful clinical study and verification of the results by autopsy. The fruit of his labor we find in papers written on inflammation, melanosis, encephaloid cancer, and numerous other topics, but especially in the great work of his life, his treatise on auscultation, published in 1816, when the author was about thirty-five years of age. This was the introduction of auscultation to the profession. So thorough were the author's observations, so accurate his conclusions, that subsequent writers have been able to add but little to the information upon this subject gathered by him. Not long after he published this work, close application began to undermine his health, and in a few years the very method which he had introduced disclosed the signs of phthisis in his own chest. Realizing fully their significance, he resigned his work in Paris and retired to his native province, where he died at the age of forty-five, leaving a name which will still be remembered when most of those now prominent have sunk into oblivion.

Since Laennec's death, the method known as immediate auscultation, according to him first practised by Boyle, has received great favor with the profession. Many physicians now consider this the only proper method of auscultation, while a few others rely entirely upon the mediate method. Whatever the advantages of either, we must familiarize ourselves with both to become accurate diagnosticians.

The stethoscope has some disadvantages. The first and main objec-

tion is that it has a peculiar ringing sound always confusing to beginners. Until we become sufficiently familiar with the instrument to ignore this, we shall be unable to appreciate the pulmonary sounds. Many of these instruments are poorly constructed. The stethoscope is of very little value in examining children, because it is likely to frighten them; besides, the respiratory murmur in them is so loud that it can be easily heard with the unaided ear.

In examining the lungs, the ear alone is usually sufficient; but to differentiate between the sounds produced at the various orifices of the heart, we must employ the stethoscope, the small chest-piece of which excludes in a great measure all sounds excepting those produced immediately beneath it.

Mediate auscultation has, however, the advantage of greatly intensifying the intra-thoracic sounds, so that signs which could not be heard by the unaided ear may be readily recognized through the instrument. Some portions of the chest cannot be easily examined by immediate auscultation—for instance, the axillary space and the supra-clavicular region; therefore the instrument becomes necessary; sometimes it may be unpleasant to apply the ear to the chest, and sometimes for the sake of delicacy it is not advisable.

The advantages claimed for immediate auscultation are: It yields no humming sound; it obviates the necessity of carrying an instrument; it does not frighten little children, and the results obtained are usually sufficiently accurate.

If the stethoscope moves slightly upon the chest, it produces a grating sound much more intense than the respiratory murmur. The same thing occurs if the finger moves upon the instrument, if the hand is drawn over the surface of the chest, or if the patient's clothes move upon the chest or upon the instrument. In some cases neither mediate nor immediate auscultation alone yields accurate results, while the two combined enable us to make a proper diagnosis.

There is now a great variety of stethoscopes. They may be classified, however, as solid and flexible, some of which are binaural and others single. The binaural instrument is provided with two tubes which conduct the sound simultaneously to both ears. The single stethoscope is designed only for one ear. The solid stethoscope most in use is a tubular instrument about six inches in length, expanded at one end into a bell-shaped chest-piece about an inch and a fourth in diameter. At the other extremity is a disk or ear-piece about two inches in diameter (Fig. 13). Some of these instruments are so made that the ear-piece may be removed for convenience in carrying, and a soft-rubber ring encircles the disk, so that it may be used as a hammer in percussion. I think physicians generally find more difficulty in examining the chest with this instrument than with the binaural stethoscope. A binaural stethoscope devised by Leared, of London, was made of gutta-percha and

consisted of two tubes, one for each ear. The auricular extremities of these tubes were disk-shaped, and the other ends were fitted into a hollow cylindrical or cup-shaped chest-piece. The elasticity of the tubes kept the disks in firm apposition with the ears. This instrument was exhibited in London in the year 1851, but it attracted little attention. About the same time Camman, of New York, introduced the binaural instrument that bears his name. This consists of two metal tubes so curved

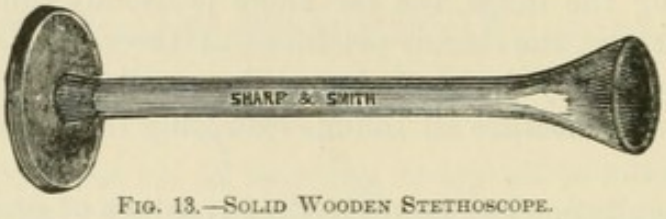

FIG. 13.—SOLID WOODEN STETHOSCOPE.

as to fit into both ears, and connected with each other by a hinge-joint. These, when placed in the ears, are held in position by an elastic passing from one to the other just above the joint, or by springs of various contrivance. The auricular ends of these tubes are tipped with gutta-percha or ivory of sufficient size to close the external meatus and prevent the entrance of external sounds. To the other ends are fitted two flexible tubes which connect them with the body of the instrument to which the chest-piece is attached (Fig. 14). Each instrument has two chest-pieces, one about an inch and a quarter in diameter, for examination

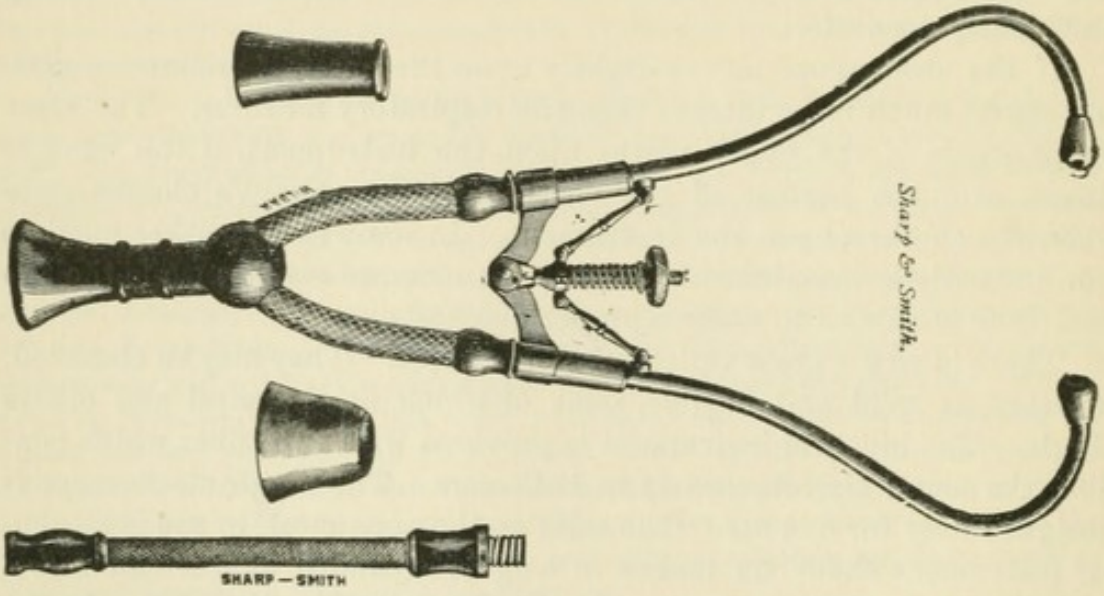

FIG. 14.—KNIGHT'S STETHOSCOPE, WITH EXTENSION. This extension tube renders it easy for the student to examine his own chest and is a great convenience in examining patients in bed.

of the lungs; the other five-eighths of an inch in diameter, for the examination of the heart.

Of the various modifications of Camman's stethoscope, Knight's is the best. It possesses all of the essential points of a good instrument, viz.: the metallic ear-tubes are curved at the proper angle to conduct

the sound directly into the auditory canal; the ear-tips are of proper size to exclude external sounds, and are not so small as to pass into the auditory canal and occasion pain; the tubes which connect the ear-pieces with the chest-piece are very pliable and have a calibre equal to that of other portions of the instrument; the chest-pieces are of proper size, and the whole instrument is thoroughly finished.

With many instruments a soft-rubber attachment is furnished which may be fitted over the end of the smaller chest-piece, and is designed for the examination of emaciated patients. This chest-piece, however, is practically worthless, on account of the creaking which is produced, during the respiratory movements, by friction with the wooden chest-piece on which it is adjusted.

Charles Dennison, of Denver, has an excellent modification of the binaural instrument; the conducting tubes are of large calibre, composed of gutta-percha and unite in a common tube with flaring extremity about an inch across; into this three other chest-pieces may be tightly fitted, two of the same material, one of medium size and one three inches in diameter. The latter is especially valuable when it is desired to hold

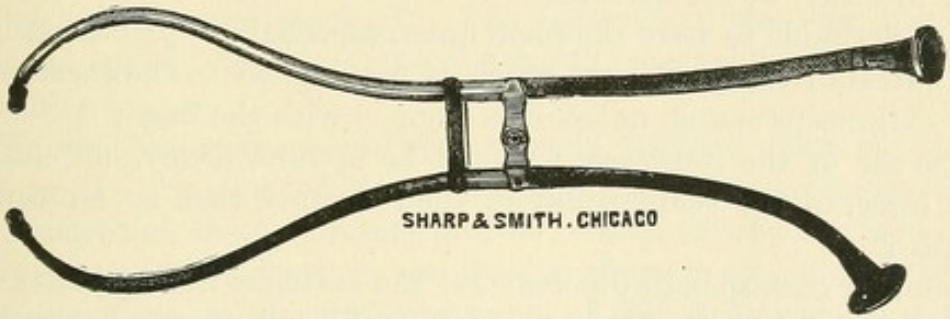

FIG. 15.—ALLISON'S DIFFERENTIAL STETHOSCOPE.

the chest-piece of the stethoscope before the patient's open mouth while percussion is being made on the chest as recommended when the signs of consolidation of the lung are indistinct. The third chest-piece is of soft rubber.

The differential stethoscope invented by Allison is essentially the same as Camman's, except that the flexible tubes are each fitted with a distinct chest-piece, so that sound can be conducted to the two ears simultaneously from different portions of the chest (Fig. 15).

A stethoscope which will fit one person perfectly and allow the sounds to be conducted without obstruction into the auditory canal, with another may rest against the external ear in such a position as nearly to occlude the orifice of the ear-piece; therefore in purchasing, one should see that the tubes are so bent that the instrument fits the ears accurately. The larger chest-piece ought never to exceed one and one-fourth inches in diameter. If larger than this, it cannot be accurately applied to an emaciated patient; consequently air passing beneath it will produce a humming sound, which will drown the pulmonary signs.

The apparatus on Knight's stethoscope for adjusting the pressure of the ear-pieces works perfectly, and is often very useful, though a simple rubber band of proper length would answer the purpose, if only one person were using the instrument. A rubber band, which could be lengthened or shortened by a buckle, would allow the instrument to be easily adjusted to any head, and would be less expensive than the metal attachment.

Considerable practice is required to perform auscultation properly. As guides, a few rules may be laid down:

In mediate auscultation, the chest must be bared; in immediate auscultation, the covering must be as soft, thin, and smooth as possible.

The position of both patient and examiner should be easy and unrestrained. If the patient is in bed, it is preferable to have him sitting, if health will permit. If the examiner is in an uncomfortable position, he cannot properly concentrate his attention upon the sounds.

In examining a child, or a patient in bed, it is a good plan to rest on one knee, so that the head will not be on a plane lower than the body, otherwise gravitation of blood to the brain will cause fulness of the head, dizziness, and impaired sense of hearing.

We must early learn to concentrate the whole attention on the sound to which we are listening.

It is desirable to have the room quiet, especially in practising immediate auscultation, for the ear which is not applied to the chest catches every extraneous sound, unless it is stopped with the finger.

The ear or the stethoscope should be applied firmly, but not with great force, to the surface, and in such manner that no air can pass beneath it.

Compare corresponding portions of the two sides during both natural and deep respirations. If one side is examined during ordinary or forcible respiration, the other must be examined under the same conditions.

The pulmonary sounds are not exactly alike in any two individuals, nor are they the same in different regions of the chest in the same individual; therefore it is necessary to study healthy cases carefully, in order to become familiar with all varieties of healthy sounds. This familiarity must be so perfect that no effort of the mind is required to remember the variations in different localities. This cannot be urged too forcibly, because until we can easily recognize the healthy sounds it is absolutely useless for us to attempt to detect the signs of disease.

When the blood leaves the right side of the heart, surcharged with carbonic acid and other débris of tissue metamorphosis, it makes a peculiar impression upon the respiratory nerves, which is transmitted to the brain as a call for more oxygen. Instantly a message is flashed back over the nerves, to the inspiratory muscles, causing them to contract. By this action the diaphragm is shortened and its convexity lessened; the ribs are lifted, and by rotation on their articulations with the spinal column, they are at the same time carried forward and outward. Thus

the diameters of the chest are increased in every direction, and air rushing in through the open glottis distends the elastic lungs as the chest expands. Immediately the respiratory act ceases, the muscles relax, the elastic tissue of the lung asserts itself, and the air is expelled from the pulmonary vesicles. This latter is a passive movement, in which the expiratory muscles take little part, excepting in forcible expiration.

While inspiration is taking place, we hear a soft, breezy, or rustling sound, known as the inspiratory murmur. As soon as it ceases, a sound soft and breezy, but less intense and much shorter, occurs, which is the expiratory murmur. This is followed by a period of rest, which completes the cycle of respiration.

AUSCULTATION IN HEALTH.

A variety of signs may be obtained in the normal chest owing to the position of surrounding organs, and the difference in the force and volume of the air current producing the sounds.

Auscultatory sounds are possessed of elements similar to those of the percussion sounds, viz., intensity, pitch, quality, duration, and in addition, rhythm. The latter refers to the relation between the different portions of the respiratory act. The *intensity* of the sound varies in different people. The *pitch* and the *quality* are practically the same in all healthy cases.

The *duration* of the sound also varies in different cases, but is about equal to the duration of the respiratory act which produces it. All modifications of the respiratory murmur which may be obtained in different regions of the chest are simply alterations in one or more of these elements. Thus in the different parts of the respiratory tract we obtain the normal vesicular murmur, bronchial respiration, and tracheal and laryngeal respiration, each of which differs from the others more or less in intensity, pitch, quality, duration, and rhythm. The clearest vesicular murmur is obtained in the infra-clavicular and infra-scapular regions. Laryngeal respiration and tracheal respiration are obtained over the larynx and the trachea, and are essentially the same. Bronchial respiration, or more properly broncho-vesicular respiration, may be heard over the bronchial tubes, and for an inch or more about them in every direction upon either the anterior or the posterior surface of the chest.

THE VESICULAR MURMUR, which is the sound obtained over the pulmonary parenchyma, is taken as the standard of comparison for all others. This sound may be best studied in the infra-scapular region, though it is more intense in front, below the clavicle; but in the latter position the heart sounds interfere with its easy recognition. The vesicular murmur, like all other respiratory sounds, is possessed of two parts. The first of these, the inspiratory, begins as a soft and distant blowing sound, and gradually increases in intensity and approaches more nearly to the ear toward the end of the act, when it is breezy or rustling in

character. It varies in intensity in different individuals, but is generally easily heard. Its pitch is low; in duration it corresponds with the inspiratory act. Its quality, called vesicular, cannot be accurately described, though it may be easily learned by practice upon a healthy chest. This sound is followed immediately by a gentle rustling sound, the expiratory murmur, which passes off gradually into a low breath or puff. It is less intense than the preceding, being usually so feeble that one must listen for it very attentively; it is of the same low pitch, and about one-fourth the duration of the inspiratory sound. Though termed vesicular, its quality is neither strictly vesicular nor bronchial, but slightly blowing.

The normal vesicular murmur is modified in different regions of the chest, by the size of the bronchial tubes, and more or less by the thickness of the chest walls and by the position of other organs. It is heard in perfection in the left infra-clavicular region. On the right side the sound is more intense, and the expiratory sound generally slightly prolonged; this disparity being due evidently to the direction and size of the right bronchus as compared with the left. There may be a very slight interval between the inspiratory and expiratory murmurs, and the quality of both is usually slightly tubular.

Over the upper portion of the sternum and the inner third of the infra-clavicular regions, the proximity of the trachea and of the large bronchial tubes renders the normal murmur somewhat tubular or broncho-vesicular in quality.

In the inter-scapular space, owing to the thickness of the chest walls, the vesicular sounds are less distinct; owing to the presence of the main bronchi, they are more tubular in character, so that in this position also we find a sound which might properly be termed the broncho-vesicular murmur, but which is usually called normal bronchial breathing.

In the scapular regions, the thickness of the chest wall renders the vesicular sound indistinct.

In children, the vesicular murmur is much more intense than in adults. Over the upper portion of the chest it is usually much more intense in women than in men. In the aged, it frequently loses something of its soft quality, and becomes slightly more tubular, and is altered in its rhythm, the expiratory sound being occasionally preceded by a short period of silence, and having a duration nearly or quite equal to the inspiratory murmur. This change seems due to partial atrophy of lung tissue and to changes in the elasticity of the chest walls.

In extreme anæmia, the vesicular murmur is intensified over the entire chest.

In listening to the respiration of muscular subjects, a continuous, low-pitched, superficial, rumbling murmur is heard where the muscles are thickest, which is due to the contraction of muscular fibres. In rare cases this is so marked as closely to resemble the vesicular murmur.

LARYNGEAL AND TRACHEAL RESPIRATION.—The respiratory murmur over the larynx and the trachea differs from vesicular respiration in its intensity, pitch, quality, duration, and rhythm. The inspiratory sound is much more intense than in the vesicular murmur, its pitch is higher, its quality tubular, and there is a marked interval between it and the expiratory sound.

The expiratory sound is generally more intense than the inspiratory, and even higher in pitch. It has the same tubular quality and about the same duration. To sum up these points of distinction, laryngeal and tracheal respiration differs from the vesicular in being more intense, higher pitched, and tubular in quality; in having an interval between the two portions of the act, and the expiratory sound is as long as the inspiratory, or even of greater duration.

BRONCHIAL RESPIRATION, or, perhaps more properly, BRONCHO-VESICULAR RESPIRATION, is next in importance to the vesicular. It may always be found in the healthy chest, but is only heard in a limited area, immediately over and surrounding the large bronchial tubes. The latter term seems more appropriate, as this combines both the bronchial and the vesicular varieties. True bronchial breathing is the same as tracheal, excepting that it is usually less intense. It is the sound obtained in pulmonary diseases where the air vesicles are completely filled by inflammatory lymph or other products. Broncho-vesicular respiration holds a place midway between bronchial and vesicular, and is the sound obtained when only a portion of the air vesicles are occluded.

The sound heard over the main bronchial tubes in the healthy chest is more intense than the vesicular murmur, and its pitch is higher; its quality is a combination of the vesicular and tubular, and a slight interval may be noticed between inspiration and expiration. The expiratory sound is of nearly equal duration with the inspiratory.

We shall at once perceive the necessity of being able to recognize these normal sounds and of knowing the localities in which they occur; for some of these, when heard in abnormal positions, are the signs of grave diseases.

AUSCULTATION IN DISEASE.

The auscultatory sounds are altered by disease, principally in their intensity, rhythm, and quality.

The intensity may be increased, giving rise to exaggerated, compensatory, or supplementary respiration. It may be diminished, and is then called feeble respiration; or the sounds may be entirely suppressed. The rhythm of the murmur may be interrupted. It is then termed jerking, wavy, or cog-wheel respiration; and the interval between the two portions of the act may be lengthened, or the expiratory sound may be prolonged.

The quality of the sound may be rude, termed broncho-vesicular, or bronchial, cavernous, or amphoric.

EXAGGERATED RESPIRATION differs from the normal murmur in intensity and duration, both the inspiratory and the expiratory sounds being intensified and somewhat prolonged. It is produced in lung tissue which is performing more than its ordinary function. When obtained over the chest of an adult it closely resembles the natural sound in a child, and hence has been termed puerile respiration. It is also termed supplementary or compensatory respiration. Like exaggerated percussion resonance, it may be said to indicate the highest degree of health in the organs where it is produced; but it also points to disease of some other portion of the respiratory tract, and is therefore a valuable negative sign. It results from any condition which, by interfering with the entrance of air into one portion of the respiratory organs, may cause more activity in the remainder. Thus, *partial consolidation, collapse, or compression of the lung* gives exaggerated respiration well marked in the sound portion of the affected organ, and more or less also on the sound side. So also obstruction of a bronchial tube by secretion or *diminution in its calibre,* by compression from tumors or thickening or contraction of its wall, may give rise to this sign in the portions of the lung not thus obstructed.

Œdema of the lungs may also cause exaggerated respiration over their apices; and in *hemiplegia,* more or less paralysis of the respiratory muscles on one side causes exaggerated respiration on the other.

FEEBLE RESPIRATION differs from the normal vesicular murmur in being less intense and shorter in duration. The inspiratory part of the sound is most affected. The sign may be occasioned by anything which interferes with the perfect transmission of sounds to the surface, as thick chest walls whether due to muscular or to adipose tissue; it is also caused by small quantities of air, fluid, or inflammatory lymph in the pleural sac.

It may result from loss of elasticity of the lung tissue in consequence of dilatation of the air vesicles, as in pulmonary *emphysema,* or from *tubercular or inflammatory consolidation* of the lung; also from deficient action of the respiratory muscles, occurring in *paralysis;* or it may exist in *diseases of the abdominal* or *thoracic organs* which give rise to pain and cause the patient to restrain muscular movement.

Collections of *fluid or gas* in the pleural cavity, *tumors* in the chest or abdomen or a pregnant uterus may interfere with the function of the lung, and prevent the descent of the diaphragm by mechanical pressure, thus causing feeble respiration.

Obstructions of the larynx, trachea, or bronchi also cause feeble respiration resulting from collection of fluids, the presence of foreign bodies, thickening of the walls by inflammation, diphtheritic or croupous deposits, œdema, and neoplasms; from contraction of the walls, as in asthma, spasm of the glottis, or paralysis of its dilators; or through compression from without by inflammatory growths, tumors, and the like.

When this diminished murmur is found in the upper part of one lung, it often indicates phthisis; if found in the lower part of the lung, it is very often an indication of pneumonia; found over the lower portion of both lungs, it is suggestive of œdema.

SUPPRESSED RESPIRATION is due to the same causes which, occurring in a less degree, give rise to feeble respiration. It is often observed over the diseased portion of a lung, the remainder of which yields the exaggerated respiratory murmur.

IN INTERRUPTED RESPIRATION, also known as COG-WHEEL RESPIRATION, either inspiration, expiration, or both may be broken into two or more parts, the sound being suddenly interrupted, to return again, and perhaps again and again, before a single respiration is complete. The interruption takes place most frequently with inspiration. The sign is found under a variety of circumstances, not only in disease, but also in health, so that it is not of much importance, though sometimes helpful in confirming a diagnosis based on other evidence. It is sometimes present over the whole chest, at other times confined to a limited space.

When occurring in health, it is often heard over the whole chest; but when resulting from pulmonary disease, it is more apt to be localized. In the incipiency of phthisis this sign is frequently obtained directly over the diseased lung, especially when the lesions are in the left apex.

It may be produced by any disease which renders respiration painful, as *intercostal neuralgia*, *pleurisy*, and *pleurodynia*. It also occurs in nervous persons when agitated by the examination, and is very apt to be found in *hysterical patients*. When due to nervousness or pain, the sign will be found over the whole of one or both lungs.

As an indication of disease, interrupted respiration is a sign of very little value, excepting in the early stage of phthisis.

In incipient phthisis the immediate cause of this sign seems to be forcible contraction of the heart, whereby an abnormal amount of blood is forced into the pulmonary circuit, thereby causing some narrowing of the calibre of the bronchial tubes.

A PROLONGED INTERVAL between inspiration and expiration may be caused by shortening of the inspiratory murmur, or by a delay in the commencement of the expiratory murmur.

Shortened Inspiration.—The inspiratory sound in this condition ceases before the act is complete and is consequently shortened, in partial consolidation of the lung due to *inflammatory or tubercular* deposits. It is deferred in its commencement after the inspiratory act begins, and thus is shortened where the air vesicles are dilated.

Deferred Expiration.—The expiratory sound is delayed when the air vesicles are distended, as in pulmonary *emphysema*.

PROLONGED EXPIRATION results from a loss of elasticity of the lungs, either by consolidation or by distention.

When due to consolidation, a prolonged expiratory murmur is usually more intense than normal. It is high pitched and more or less tubular in quality, and usually possesses so much of the bronchial element as to be termed broncho-vesicular.

The prolonged expiratory murmur which is sometimes found in healthy chests possesses the same pitch and quality as the normal vesicular sound, which enables us to distinguish it from the prolonged expiration of consolidation, in which the pitch is always high and the quality somewhat tubular. We must not forget that in health the vesicular murmur over the right apex is sometimes more or less tubular and high in pitch, and that the expiratory sound is prolonged, as compared with the left side. Therefore, in this position the sign cannot always be considered as indicative of disease, unless it be taken in connection with other signs.

When obtained on the left side, prolonged expiration is nearly always due to phthisis or to emphysema. The difference in the two is that in consumption the expiratory sound is high pitched and more or less tubular in quality; while in emphysema it is usually even more prolonged—it may be two or three times as long as the inspiratory murmur—and it has a low pitch, it is not tubular but rather vesicular in quality, and is apt to be considerably less intense than the inspiratory sound.

Occasionally prolonged expiration may be caused by interference with the free exit of air from the lungs, as by obstruction in the larynx or bronchial tubes. In these cases it is usually associated with a deferred inspiratory murmur, in which the sound does not begin with the inspiratory act.

Exceptional.—Prolonged expiration having the pitch and quality of the healthy murmur is obtained with cavernous respiration in rare cases. In such instances its significance is ascertained by the character of the inspiratory sound and by other signs.

RUDE RESPIRATION (BRONCHO-VESICULAR OR HARSH RESPIRATION) closely resembles the sound which can be obtained directly over the bronchial tubes in a healthy chest.

The respiratory sound is raised in pitch in proportion as the tubular supplants its vesicular quality. The expiratory sound is always higher in pitch than the inspiratory, its quality is more or less tubular, and it is prolonged. The alteration in pitch and duration is in proportion to the preponderance of the tubular over the vesicular quality.

Disease may furnish all degrees of broncho-vesicular respiration from the *normal vesicular murmur* to perfect *bronchial breathing*, according to the amount of consolidation.

This sign is due to the better transmission of the vibrations from the larynx, trachea, and bronchial tubes to the surface of the chest, in consequence of the consolidation of the air vesicles, making the parenchyma a better conductor of sound-waves and rendering the bronchial tubes

more rigid, so that they transmit these waves from the upper air passages with less resistance.

The sign is obtained in *incipient phthisis* over the upper part of the lung, and in *pneumonia*, usually over the lower lobe. It is also heard in some cases of *pulmonary apoplexy*, and over a lung partially collapsed from any cause or which has been compressed for a considerable time by *fluid or air in the pleural sac*. It is most valuable as a sign of incipient phthisis.

Exceptional.—Occasionally in cases where broncho-vesicular respiration occurs, either the inspiratory or expiratory murmur may be absent; then, as in similar instances of bronchial respiration, its detection will depend on the *pitch* and *quality* of the sounds which *are* present, and upon concomitant signs.

BRONCHIAL RESPIRATION is one of the most important varieties of the healthy sounds, which may sometimes be indicative of disease. Its quality and its other elements excepting its intensity are much the same as those of normal tracheal respiration. The intensity of this sound is usually greater by far than that of the vesicular murmur, but sometimes very feeble; the pitch is high, the quality tubular, and the duration of both inspiration and expiration is prolonged, the two being of about equal length. There is an appreciable interval between the inspiratory and expiratory sounds.

Exceptional.—In bronchial respiration, either portion of the respiratory murmur may sometimes be absent.

Laennec taught that the bronchial sound was always produced in a healthy chest, but that it was not usually heard because of the intervention of air vesicles between the tubes and the chest walls. When obtained in disease, he considered the sign due simply to the better transmission of the sounds to the surface. Skoda believed that consolidation of the air vesicles surrounding the bronchus was necessary for the production of the perfect sign. Whichever of these views is correct, or whether both are in part true, matters little to us, so long as we know that the sign always indicates consolidation of lung tissue (Fig. 27). The tubular sounds in this variety of the respiratory murmur are transmitted for a considerable distance beyond the consolidated lung, which accounts for the fact that the bronchial and the vesicular elements are frequently combined in the regions immediately surrounding that which yields simply bronchial respiration.

The greater intensity of the expiratory sound in bronchial respiration accounts for the fact that occasionally we obtain a vesicular inspiratory and a bronchial expiratory sound, as the intensity of the bronchial sound drowns the vesicular in expiration.

Bronchial respiration is found in greatest perfection, in *pneumonia*, over the consolidated lung. It is obtained also in some cases of *phthisis*, but in this affection we are more apt to hear broncho-vesicular respiration.

Exceptional.—In rare cases cancer of the lung yields bronchial breathing. Pulmonary apoplexy sometimes causes the sign; it is heard over the entire chest, though more distant than in consolidation, in a few cases of pleurisy with extensive effusion.

CAVERNOUS RESPIRATION has been likened to both bronchial and vesicular. We are told by one author that it closely resembles the former, and by another that great care is necessary to distinguish it from the latter. This discrepancy is probably due to confusion in the application of the term to different signs. Flint made the distinction clear by introducing the term broncho-cavernous to designate those hollow, high-pitched sounds which, although conveying the idea of a cavity, do not correspond with true cavernous respiration. The intensity of cavernous respiration is usually feeble, so that, unless searched for carefully, it will be overlooked. The pitch is low, and the quality, instead of being vesicular or tubular, is soft and blowing or puffing. The expiratory portion of the sound is prolonged to about the same length as the inspiratory, and is even lower in pitch than the latter. The failure of some diagnosticians to appreciate the quality of this sound has caused them to deny its existence. I have occasionally heard the true cavernous murmur as just described, but I think it a very rare sign. It is produced in empty pulmonary cavities, the walls of which are so flaccid that they expand readily in inspiration and collapse in expiration (Fig. 16). It is a sign, therefore, of any of those diseases which might cause such a cavity, viz., *consumption, pulmonary abscess,* or *gangrene of the lung.*

Broncho-cavernous respiration is made up of both the bronchial and the cavernous sounds. It is usually described as cavernous, but it is higher in pitch and more tubular in quality than the latter. Its quality is not sufficiently tubular to be called bronchial, nor yet sufficiently soft and puffing to be termed cavernous. It is produced in pulmonary cavities, surrounded by lung tissue more or less consolidated; the tubular element being dependent upon the amount of consolidation. Sometimes the first part of the inspiratory murmur may be tubular in quality and the last part cavernous; again, we may obtain cavernous inspiration with bronchial expiration, due to the presence of consolidated lung tissue near the cavity. In the latter case the intense expiratory bronchial murmur probably drowns the cavernous sound which was heard with the feebler inspiratory murmur.

Broncho-cavernous respiration is the characteristic sign of the later stages of *consumption,* but it may also be produced in the cavities due to *abscess* or to *gangrene.*

AMPHORIC RESPIRATION resembles the sound produced by blowing into the mouth of an empty bottle, hence the name. It is of a metallic musical quality, and may be heard during either inspiration or expiration, or during both portions of the respiratory act, but is generally most marked in expiration. The expiratory sound is lower in pitch than that

in bronchial respiration. In this connection it is well to emphasize the necessity of studying the pitch of the respiratory sounds, for in some instances there is absolutely no other means of distinguishing between the sounds transmitted from the bronchial tubes in consolidated lungs and those heard over pulmonary cavities. The distinction in these cases is clear if we remember that the expiratory sound in the former instance is always high in pitch, in the latter always low.

Amphoric respiration occurs under the same conditions as amphoric resonance, and is frequently found in connection with cracked-pot resonance. It is due to the passage of air in and out through an opening

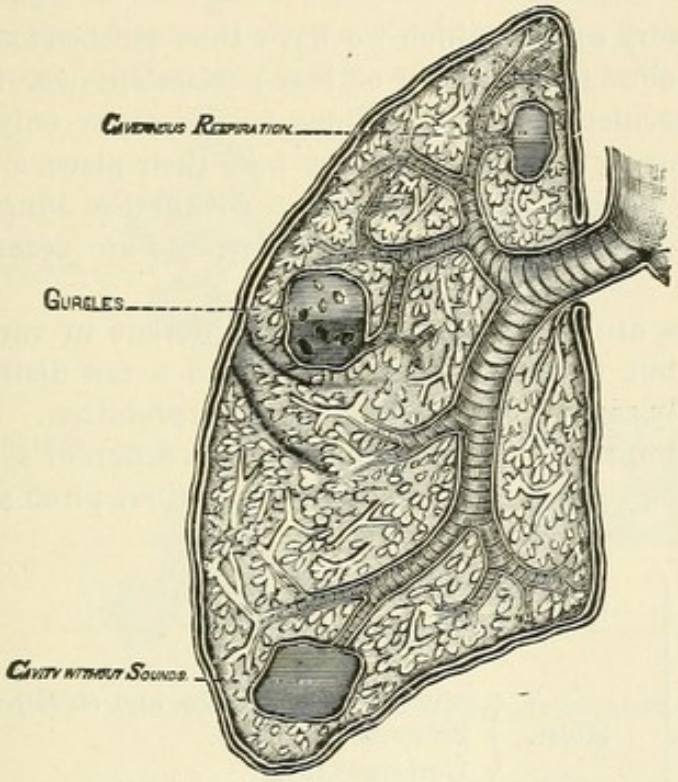

Fig. 16.—Phthisis.

from a bronchus into a large pulmonary cavity or into the pleural sac (Fig. 26). The sign is obtained most perfectly in *pneumothorax* or in *pneumo-hydrothorax*. In the latter it disappears and returns again, as the quantity of fluid rises so as to cover the opening or falls below it. This sign is also heard in phthisis when the pulmonary cavity is large and its walls are firm, so that they will not collapse in expiration.

Cavities may exist within the lungs without yielding either of the varieties of respiration which may be caused by a vomica; for example, if a cavity be filled with fluid, or if the fluid in the cavity rise above the orifice of the bronchial tube, none of these sounds will be heard (Fig. 16); but if the patient's position be changed or the amount of fluid decreased by coughing, the signs return.

CHAPTER IV.

METHODS OF EXAMINATION.—*Continued.*

ADVENTITIOUS SOUNDS.

THE auscultatory sounds which we have thus far been studying are such as may be obtained, in more or less perfection, over the healthy chest. Certain accidental or adventitious sounds occur only in disease. These may accompany normal sounds or take their place, and will vary according to their origin. Those produced within the lungs are called *râles* or *ronchi;* those upon the pleural surfaces are termed *friction sounds.*

RÂLES.—Râles are as numerous and as different in variety as the shades of color, but they may be grouped into a few distinct classes, which are generally capable of some peculiar interpretation. All of them are either dry or moist; hence we may group the different sounds under one of these heads, according to peculiarities in their pitch and quality, as shown below:

- Râles, or rhonchi,
  - Dry.
    - Sonorous râles.
    - Sibilant râles.
  - Moist.
    - Mucous râles (large and small).
    - Subcrepitant râles.
    - Crepitant râles.
  - Gurgles (large and small).
  - Mucous click.

Râles may originate in the larynx, trachea, bronchial tubes, air vesicles, or in any cavity connected with the bronchial tubes. They are produced by various conditions which interfere with the passage of air through the tubes and into the air vesicles, and may be heard in inspiration or expiration, or during both portions of the respiratory act.

DRY RÂLES are distinguished as sonorous or sibilant according to their pitch, which depends on the size of the bronchial tube in which they are produced.

Sonorous râles are usually musical, or snoring in quality, resembling the sound produced by blowing through a tube; they are sometimes cooing, sighing, or moaning in character. Their intensity varies from a sound which can be scarcely recognized to one which may be heard at

a distance from the chest, and their pitch is always low. They may be heard during both inspiration and expiration, but are most frequent in expiration. They are produced in bronchial tubes exceeding one-eighth of an inch in diameter. They are caused by the vibrations of viscid mucus or by a fold of mucous membrane, or by anything which constricts the calibre of the tube, as pressure upon its external surface by tumors, bands of cicatricial tissue resulting from former diseases, or contraction of the circular muscular fibres causing a uniform narrowing of the tube (Fig. 17). These sounds are not removed by coughing, unless caused by tenacious mucus adhering to the side of the bronchial tube. Though in the great majority of instances after coughing or after deep inspiration an individual râle may disappear, other râles will remain in some portion of the chest. This sign is obtained in greatest

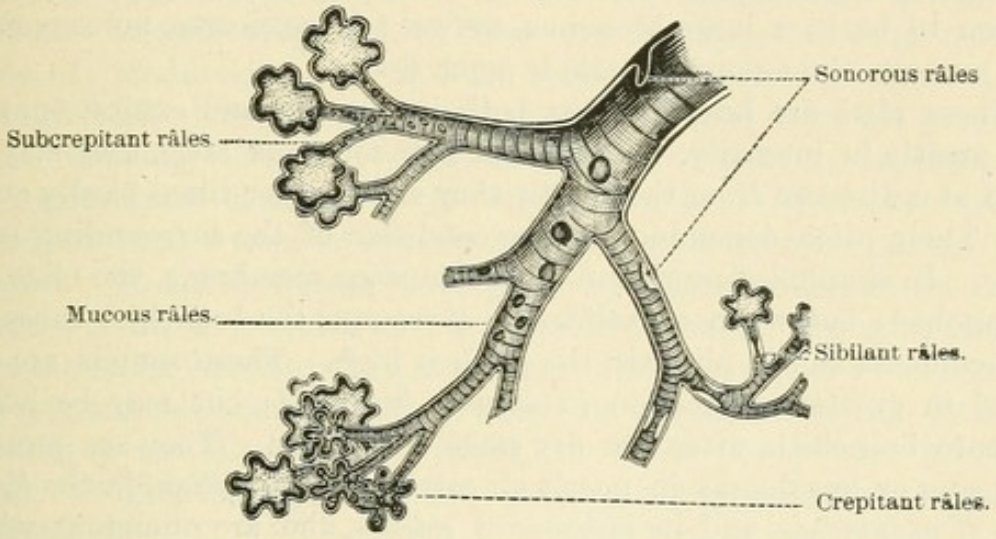

FIG. 17.—BRONCHIAL RÂLES, DRY AND MOIST, AND SUBCREPITANT RÂLES.

perfection in the early stages of *acute bronchitis* and in *asthma*. It is also heard in some cases of *chronic bronchitis*, occasionally in *phthisis*, and rarely in *pneumonia*, being in these latter instances associated with other adventitious sounds.

When obtained in phthisis, the dry râles are few in number and are associated with moist râles.

In the early stage of asthma, sonorous râles may be heard in great numbers over the entire chest.

Sibilant râles occur both in inspiration and in expiration, but are heard mostly in inspiration. They are not so intense as the sonorous sounds. Their pitch is high, and in quality they vary almost as much as sonorous râles, being sometimes whistling, sometimes hissing, and sometimes almost creaking. They are caused in the smaller bronchial tubes by the same conditions which give rise to râles in the larger bronchi (Fig. 17).

They are heard most frequently and abundantly in *asthma* and in

capillary bronchitis. In ordinary acute bronchitis they may be heard, though in limited numbers.

Sibilant râles are heard occasionally in phthisis, due then to localized bronchitis or to tubercular deposits. They are sometimes, though not often, heard in pneumonia. Occasionally, even in healthy or apparently healthy chests, we may hear two or three of these fine sounds near the borders of the lungs.

Sibilant râles may be altered, but they are seldom removed by coughing or by forced inspiration.

MOIST RÂLES are grouped as mucous, large and small, subcrepitant and crepitant, according to their characteristics.

Mucous râles, also produced in the bronchial tubes, are large or small according to the size of the tubes, and are caused by air bubbling through fluid—mucus, pus, serum, or blood (Fig. 17). If the bubbling happen to be in a large bronchus, we get a large, coarse, mucous râle; if in a smaller bronchus, the râle is much finer.

These râles are heard during both inspiration and expiration, and vary greatly in intensity. Sometimes, like sonorous râles, they may be heard at a distance from the chest; they are at other times hardly audible. Their pitch depends upon the condition of the surrounding lung tissue. In simple inflammation of the mucous membrane, the râles are low pitched; but when consolidation surrounds the bronchial tubes, as in pneumonia and in phthisis, the pitch is high. These sounds are obtained in greatest perfection in *chronic bronchitis,* but may be heard in acute bronchitis after the dry stage has passed. They are present in greater or less degree in nearly all cases of *consumption,* in the *third stage of pneumonia,* and in *pulmonary œdema,* and are numerous when *hemorrhage* has taken place into the bronchial tubes until coagulation occurs. In phthisis they are found over a limited space, due sometimes to associated bronchitis, at other times to the escape of fluid from a cavity into the bronchial tubes. These, unlike dry râles, are usually much affected by deep inspiration and coughing, by which they may be considerably altered or entirely removed.

Subcrepitant râles are moist sounds, which are produced in the very fine bronchial tubes, probably in the ultimate bronchi and those a size larger (Fig. 17). They are caused by air bubbling through fluid, and may be heard during either or both portions of the respiratory act, but are most frequently heard with inspiration. They are of comparatively feeble intensity, vary in pitch according to the condition of the surrounding tissue, and are distinctly moist and crepitating or crackling in quality.

These râles may be heard most perfectly in *capillary bronchitis* and the *third stage of pneumonia.* They are often found in *asthma* shortly after the paroxysm. They are present in *congestion of the lung,* purulent *bronchitis,* and *pulmonary œdema,* and are found over a limited por-

tion of the lung in many cases of phthisis. They occur in *brown induration* of the lungs, and are heard after *hemorrhage* into the smaller bronchial tubes, limited to the position of the hemorrhage.

The subcrepitant râle, due to circumscribed capillary bronchitis, is a sign of great value in the early diagnosis of phthisis, in which it may often be found at the apex of the lung before any other signs can be detected.

The crepitant râle is largely like the subcrepitant, but differs from the latter in two respects: it is not so moist or liquid in character, so that it is sometimes classed as a dry râle; and it is never obtained in expiration. Crepitant râles are very well imitated by rubbing together a lock of hair close to the ear. They were compared by Laennec to the sound produced by throwing salt upon a fire.

These râles are produced in the vesicles, intercellular spaces, and ultimate bronchi (Fig. 17). There are two hypotheses as to their mode of production: one is that they are caused by air bubbling through fluid within the air vesicle, just as mucous râles are produced in the bronchial tubes; the other, that they are due to the separation of the agglutinated surfaces of the capillary tubes or of the air vesicles. Which of these is true, or whether both are in part correct, has not been decided. To me they seem to be produced by separation of the sticky surfaces of the air vesicles, and the capillary bronchi. In some cases of pneumonia, for instance when associated with inflammatory rheumatism, no crepitant râle can be obtained which may be accounted for by slight viscidity of the inflammatory lymph; for if the sounds were produced by air bubbling through fluid, they would occur regardless of the nature of that fluid.

Crepitant râles are much more numerous than the subcrepitant. In listening to subcrepitant râles, we seldom seem to hear more than ten or fifteen at once; whereas with the crepitant râle we seem to hear a hundred or more with each inspiration.

Crepitant râles are obtained in perfection in the *early stage of pneumonia*, of which they are considered diagnostic. This stage lasts but a few hours; consequently in many cases of inflammation of the lung the râles have disappeared before we see the patient.

A few crepitant râles are sometimes heard in congestion of the lung and in pulmonary œdema, and they are frequently found in phthisis, in a small zone around the consolidation. In this latter case they seem to result from gradual extension of the pneumonitis, which often precedes tubercular deposit.

Crepitant râles, subcrepitant râles, and friction sounds are sometimes so much alike that it is difficult to distinguish between them. If dry crepitating sounds are numerous and heard only on inspiration, they are crepitant râles; but if dry crepitating sounds are few in number and

are heard in expiration or in both inspiration and expiration, they are likely to be friction sounds. Subcrepitant râles are more moist and not nearly so numerous as crepitant râles, and they are usually heard in both inspiration and expiration. The moist character, the number, and the time of occurrence of subcrepitant râles will enable us to distinguish them from the crepitant; and their deeper seat and their constancy will usually enable us to distinguish them from fine friction sounds—which are still fewer in number—even when the latter are moist in character.

Crepitant râles are not much affected by cough or forced respiration when due to pneumonia, but in other instances two or three full inspirations will frequently dispel them.

Exceptional.—Either crepitant or subcrepitant râles may be sometimes brought out directly after coughing where they were absent a moment previously. A sound closely resembling the subcrepitant or the crepitant râle may frequently be obtained over the thin border of the healthy lung; in these instances, only a few of the râles are heard, and they disappear after three or four forced inspirations.

GURGLES resemble large mucous râles, but are generally higher in pitch and possess a hollow metallic quality; though occurring during both portions of the respiratory act, they are most frequent in inspiration. They are produced by air bubbling through fluid in cavities which communicate with the bronchial tubes (Fig. 16). If cavities are completely filled with fluid or entirely empty, or if the level of the fluid does not reach above the opening of the bronchial tube, no gurgles will be produced. These sounds are large or small, according to the size of the cavity in which they are produced.

This sign is usually indicative of *phthisis*, but may occur in any pulmonary disease which causes excavations.

THE MUCOUS CLICK resembles an isolated subcrepitant râle, and is heard during inspiration only. The sign generally consists of a single click, or, at most, of two or three clicks. It is a sharp crackling or clicking sound, supposed to be produced in the smaller bronchial tubes by sudden separation of their agglutinated surfaces during inspiration; it is not usually affected by cough. When heard over the apex of one lung, it is a sign of considerable value in the early diagnosis of *phthisis*. Such sounds are sometimes heard over a considerable portion of the lung in *acute tuberculosis*, in extensive *chronic pneumonia*, or in the later stages of *interstitial or catarrhal pneumonia*.

FRICTION SOUNDS.—Friction sounds are produced by rubbing together of the two pleural surfaces, which are either dry from diminution of their natural secretions or roughened by exudation of inflammatory lymph (Fig. 18). These sounds are grazing, rubbing, grating, rasping, or creaking in character; sometimes dry, sometimes moist. They may be simulated by rubbing the back of the hand, while listening with the stethoscope on its palm, or by rubbing the fingers on the integument

when auscultating the chest. They are usually few in number and transitory, being heard for a few respirations, and then disappearing to return again in a few minutes; they may be heard just at the end of inspiration or at the beginning of expiration. This is the characteristic sign of pleurisy. The grazing friction sound is only heard in the beginning of the inflammation, and can be detected most frequently in the circumscribed pleurisy accompanying phthisis. Some one of the other varieties, of which the quality is of no importance, may be heard in the first and third stages of pleurisy. Care must always be taken not to mistake for this sign the sounds produced by crackling of the hairs beneath the instrument, or by the rubbing of the stethoscope, the fingers, or the

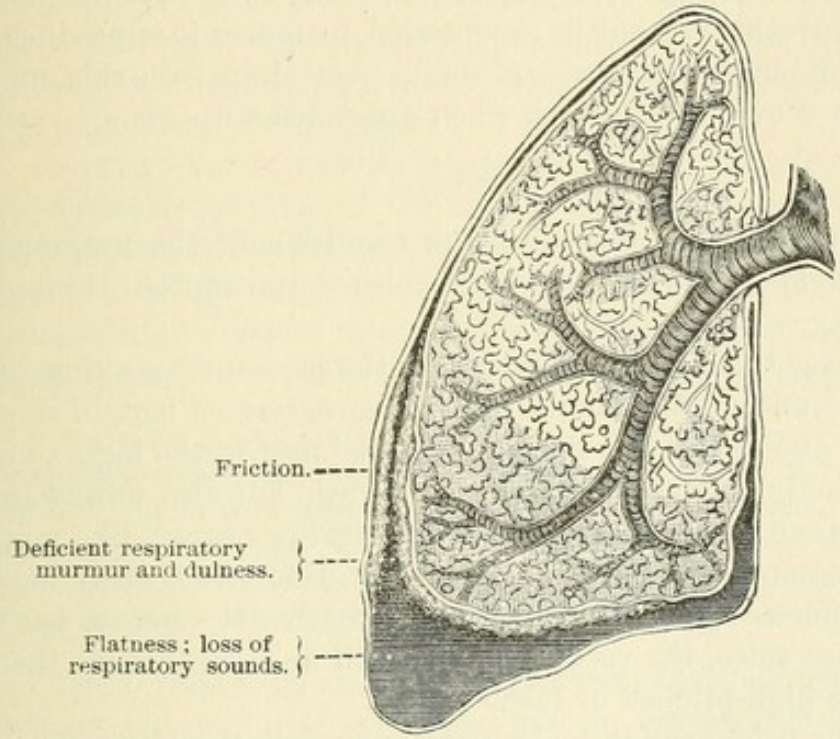

FIG. 18.—ACUTE PLEURISY. The upper part of the lung is in a normal condition, or the air cells are slightly distended. The lower part of the lung is partially collapsed. The upper surface of the fluid is not horizontal, but it conforms more or less perfectly to the natural outline of the lung.

clothing on the surface, or of the clothing or fingers on the instrument. Sounds closely resembling the friction murmur are often heard over the false ribs in a healthy chest. They seem to be produced by slight movements of the skin beneath the rim of the stethoscope.

Creaking or *crumpling* sounds are sometimes obtained over the chest, the signification of which is not fully understood. The creaking sounds are most frequently heard at the lower part of the thorax, and are supposed to be due to old pleuritic adhesions. Both creaking or crackling and crumpling sounds are sometimes obtained over the upper portion of the chest. The crumpling sounds which are heard in inspiration resemble those which may be produced by inflating a dried bladder, and are supposed to be produced from similar causes; that is, the inflation

of dry emphysematous air cells. Thompson considers these sounds indicative of syphilitic disease of the lungs. When confined to the apex, they are nearly always associated with phthisis.

METALLIC TINKLING is a clear, silvery, tinkling sound, like that produced by dropping a pin into a glass. It seems to be caused by the falling of a drop of fluid from the upper part of a large cavity on the surface of fluid below. It can sometimes be heard over one entire side, but it is usually most distinct on a level with the nipple. When the proper conditions are present within the chest—that is, a large cavity containing air and fluid—it may be produced by any agitation, such, for example, as speaking, coughing, deep inspiration, or occasionally by the act of swallowing. The sign occurs most frequently in the pleural cavity in pneumo-hydrothorax; but in exceptional instances it is produced in very large pulmonary cavities. A sound very similar to this may sometimes be heard over the stomach when distended with gas.

VOCAL SOUNDS.

Considerable information regarding the condition of the lungs can be obtained by studying the sounds of the voice as transmitted through the chest walls.

If we listen over the healthy chest while the person is speaking, an indistinct, distant, and muffled sound will be heard, termed *normal vocal resonance*. It is due to the fact that sounds produced in the larynx are transmitted not only outward through the mouth, but also downward through every branch of the bronchial tree. Vocal resonance, like most of the other pulmonary sounds, varies greatly in different healthy individuals and in different portions of the same chest. If a person has a low-pitched intense voice, the vocal resonance will be more forcible than in those who have high-pitched or feeble voices.

In studying the voice-sounds by immediate auscultation, it is desirable to close the ear which is not applied to the chest, in order to exclude sounds coming from the mouth, and it is better to have the patient count one, two, three, than to ask him questions and listen for the answers. By the latter course the examiner's attention is distracted from the sounds within the chest in the attempt to catch the patient's reply. The varieties of vocal resonance which may be heard over different regions of the normal chest are named from the parts in which they are produced; over the larynx and trachea we have laryngeal and tracheal resonance; over the bronchial tubes, bronchial resonance; and over air vesicles, the normal vesicular or, as it is usually termed, normal vocal resonance.

LARYNGOPHONY is the vocal resonance obtained over the larynx, and TRACHEOPHONY that obtained over the trachea. In these varieties the words are imperfectly articulated, but the voice is transmitted to the ear "with a force and intensity almost painful." The sounds are con-

centrated or, in other words, seem to be produced within a small area immediately beneath the stethoscope, and necessarily vary in pitch with the pitch of the individual's voice.

NORMAL BRONCHOPHONY is obtained while the person is speaking, by listening over the bronchial tubes, near the border of the sternum from the first to the third rib, or more especially directly over the main bronchi on a level with the second costal cartilages in front, or on a level with the fourth dorsal vertebra in the inter-scapular region. This occupies a position midway between normal vocal resonance and laryngophony. The sounds thus obtained are transmitted to the ear with considerable intensity, though with much less force than over the larynx; they appear to be produced immediately beneath the stethoscope, but the words seem very imperfectly articulated. Whenever this sign is obtained over any other portion of the chest, it indicates consolidation of the pulmonary parenchyma.

NORMAL VOCAL RESONANCE is obtained by listening to the voice over the vesicular portions of the lung. This sound, having no approach to articulation, is distant and diffused, seeming to come from the deeper portions of the lung two or three inches beneath the surface. As a rule, vocal resonance is always more intense upon the right side than upon the left, especially in the infra-clavicular regions.

Exceptional.—In a few instances over the right apex, even in health, the resonance very nearly approaches bronchophony. If the sounds have this character upon both sides, as they have in rare instances, they will be found most intense upon the right side, but higher in pitch on the left—a disparity due to the difference in calibre of the bronchial tubes; those upon the right side being the larger must necessarily give the more intense and lower-pitched sound.

The normal vocal resonance is generally obtained over the entire chest in men, but only over the upper part in women and children, in whom it is a sign of little value.

This sign is modified by disease, principally in its intensity, which may be either diminished or increased.

- Intensity.
  - Diminished. — Vocal sounds feeble or suppressed.
  - Increased. — Vocal sounds exaggerated.
    - Resonance which is termed bronchophony.
    - " " " ægophony.
    - " " " pectoriloquy.
    - " " " amphoric voice.

DIMINISHED RESONANCE.—Diminished resonance is usually due to much the same causes as the diminished respiratory murmur; that is, separation of the pulmonary from the costal pleura by air or fluid, as in pneumothorax or pleurisy. It also occurs in cases of extreme *emphysema*, in *pulmonary œdema*, in *bronchitis* with free secretion, and occasionally where there is *extreme pulmonary consolidation*.

The vocal sounds are mostly suppressed over fluid in the pleural sac; but just above the level of the fluid the air cells are partially collapsed, so that vocal resonance is increased. For an inch or an inch and a half below the level of the fluid the resonance is diminished in intensity, and a little lower it is nearly suppressed. Thus we are able to ascertain the height of the fluid by means of the vocal resonance as well as by percussion.

This sign is principally of value in the diagnosis of pleuritic effusion, by enabling us to distinguish between it and consolidation of the lower part of the lung.

Exceptional.—In some cases the vocal resonance may be heard distinctly all over the pleuritic effusion, though the sounds are distant and more or less muffled.

Increased Vocal Resonance.—Exaggerated vocal resonance differs from the normal voice-sounds simply in its intensity. This sign denotes more or less consolidation of the lung tissue or collapse of the air vesicles, and is usually associated with broncho-vesicular respiration.

It is a sign of considerable importance in the diagnosis of the early stage of phthisis and in discriminating between pneumonia and pleurisy.

Exceptional.—In very rare cases the vocal resonance is exaggerated in pneumothorax and in emphysema.

Bronchophony, as already noted, consists of more or less intense vocal sounds, usually imperfectly articulated, which have a peculiar degree of concentration, or, in other words, seem to be produced immediately beneath the stethoscope, instead of coming from the deeper portions of the lung. The intensity of this sign, which may be greater or less than that of normal resonance, is an unimportant element; so also is the distinctness of articulation. Its recognition depends chiefly on the characteristic concentration.

The significance of bronchophony depends upon its location. If heard over the main bronchial tubes, it may be simply a healthy sound; but if heard over vesicular portions of the lungs, it is indicative of consolidation. It is usually associated with a tubular respiratory murmur; but as it occurs with a less amount of consolidation than is necessary for true bronchial breathing, it may frequently be obtained with broncho-vesicular respiration.

Exceptional.—Bronchophony usually possesses the characteristic concentration; but when the consolidated lung is separated from the chest wall by fluid, it may sound distant.

This sign is of special value in the diagnosis of the second stage of pneumonia (Fig. 27). It is seldom obtained perfectly in phthisis, because in this disease consolidation is not usually complete.

Exceptional.—Bronchophony is occasionally obtained in carcinoma of the lung, though usually this disease involves the whole tissue, air vesicles and bron-

chial tubes alike, or it crowds the pulmonary tissue before it, thus hindering the transmission of the voice. But when the air vesicles alone are filled and the bronchial tubes remain patent, as occurs in rare cases, bronchophony may be obtained. It is also present in hemorrhagic infarctions which fill the air vesicles but leave the bronchial tubes open, and may therefore be a sign in pulmonary apoplexy.

ÆGOPHONY is a variety of bronchophony. It is a tremulous sound which has been compared to the bleating of a goat; hence the name. Like bronchophony, it conveys to the listening ear the impression of having been produced within a very limited portion of the lung; unlike the latter, it seems to come up from a considerable depth, and to tremble about the end of the stethoscope. When present, it may be most readily obtained in the inter-scapular or axillary regions. This sound is generally produced in consolidated lung tissue which is separated from the chest wall by a thin layer of fluid. It is a sign of *pleuro-pneumonia*—that is, pneumonia and pleurisy with effusion; but even in this disease it is present only a short time, and is a sign of little value. Ægophony is most frequently produced when the pleural cavity is about half filled with fluid.

In ordinary pleuritic effusions, the lung just above the surface of the fluid is more or less solidified by collapse of a portion of the air vesicles; under such circumstances ægophony may be produced providing the pleura-pulmonalis and the pleura-costalis are agglutinated just above the collapsed lung.

PECTORILOQUY differs from bronchophony in that the articulated speech is more completely transmitted. In bronchophony the voice is heard, but the words are not distinct. In pectoriloquy articulation is nearly perfect. There are two varieties of pectoriloquy: one in which the sounds are concentrated and near the ear like bronchophony, but are heard over a considerable portion of the lung; and another in which the sign is confined to a limited space and has not the degree of concentration found in bronchophony. The first of these, which is high in pitch and clanging or metallic in quality, is frequently produced by simple consolidation of lung tissue. The second, which is low in pitch and softer in quality, is always a trustworthy sign of a pulmonary cavity with smooth walls and a large opening into a bronchial tube. Well-defined pectoriloquy is not a frequent sign, but when heard the first variety is a sign of *phthisis or pneumonia*, and the second of any of those diseases which cause vomicæ, viz., *phthisis*, *pulmonary abscess* or *gangrene*, and *bronchiectasis*.

AMPHORIC VOICE is hollow and more or less musical in character. The musical quality follows the voice and is termed the amphoric echo. The words are not articulated, as in pectoriloquy. This sign occurs under the same conditions as amphoric respiration and amphoric percussion resonance; that is, over the pleural sac when containing air and

communicating freely with a bronchial tube, and over very large cavities in the lungs.

Exceptional.—There are good reasons for believing that, in rare cases, amphoric voice, as well as amphoric respiration, may be heard over a layer of air in the pleural cavity which does not communicate with the bronchial tubes.

Amphoric voice is a sign of *pneumo-hydrothorax*, in which disease it is associated with tympanitic resonance over the upper part of the chest, and ordinarily with the succussion sound. If the latter signs are absent, the amphoric voice is probably produced in a phthisical cavity.

Whispering Vocal Resonance.—Flint described the whisper resonance with considerable minuteness. He considered the signs which it furnishes of equal value with those from a loud voice; I find them of even greater importance.

The normal bronchial whisper is a term applied to sounds of a blowing or tubular character, very closely resembling the sound of forced respiration, heard in listening over the upper portion of the chest when a person is speaking in a sharp whisper. Its modifications by disease are classified as exaggerated bronchial whisper, whispering bronchophony, cavernous whisper, whispering pectoriloquy, and amphoric whisper.

Exaggerated bronchial whisper is more intense and higher in pitch than the normal sound. It is produced in lungs which are slightly solidified.

Whispering bronchophony is higher in pitch and more intense and blowing than the preceding. It has the same characteristic concentration and nearness to the ear as bronchophony with the loud voice. It may be obtained over lungs so slightly solidified as to yield only exaggerated vocal resonance when the patient is speaking aloud; therefore it can be appreciated sooner than bronchophony with the loud voice. This fact renders whispering bronchophony a most important sign in the early stage of phthisis.

The cavernous whisper is a low-pitched, blowing sound, confined to a limited portion of the chest. It is produced within pulmonary cavities under the same conditions as cavernous respiration. This sign is principally of value in the diagnosis of phthisis.

Whispering pectoriloquy differs from whispering bronchophony only in its more perfect articulation. When obtained over a small space only, this is a sign of a cavity. It is most frequently found in phthisis.

Amphoric whisper occurs under the same conditions as the amphoric voice or amphoric resonance on percussion; that is, over the pleural sac filled with air, or over very large cavities in the lung tissue.

Aphonic pectoriloquy is a term which has been applied to the voice sounds when the patient is speaking in a low tone. It has been stated that these sounds can be distinctly heard not only over consolidated or collapsed lung,

but also even when the organ in this condition is separated from the thoracic wall by a collection of air or *serum;* however, these vibrations are *not* conducted through *pus.* By studying this variety of vocal resonance, it is claimed that we may determine whether pleural effusions are of a serous or of a purulent character. I have been able to verify this statement in a few cases, but not in all.

TUSSIVE SIGNS.—The resonance of cough may sometimes be studied with advantage, especially in children. The act of coughing is often of special value in dislodging obstructions in the bronchial tubes or pulmonary cavities, and also in causing a subsequent deep inspiration which will freely inflate the air cells, thus bringing out signs which might otherwise be overlooked. The different varieties of cough are classified as laryngeal, bronchial, cavernous, and amphoric.

LARYNGEAL COUGH is usually more or less hacking in character, and often spasmodic. It is indicative of laryngitis.

BRONCHIAL COUGH is quick, harsh, and brassy. It is accompanied by a thrill or fremitus, and if severe is nearly always attended with pain beneath the sternum or along the inferior ribs, corresponding to the attachment of the diaphragm. It is generally indicative of bronchitis.

CAVERNOUS COUGH is produced under the same circumstances as cavernous respiration, and is generally associated with gurgles. It has a hollow quality and is usually very intense.

AMPHORIC COUGH is more musical and hollow in quality, is generally lower in pitch, and seems to penetrate the ear with less force than the cavernous. It is heard over very large pulmonary cavities or over the pleura when filled with air.

Sometimes large pulmonary cavities are traversed by trabeculæ which yield a peculiar twang when the patient coughs. This is of special value, as these strings prevent cavernous or amphoric voice-sounds.

Tussive signs are usually, though not always, transmitted through consolidated lung, but seldom through collections of fluid.

We may obtain considerable information about the condition of the lungs in children who cannot be induced to speak by studying the cry, which is subject to the same variations as vocal resonance in adults.

CHAPTER V.

PULMONARY DISEASES.

PLEURISY OR PLEURITIS.

PLEURISY consists of an inflammation, more or less extensive, of the serous membrane covering the lungs and lining the thoracic walls. There are three recognized varieties of this disease: the acute, subacute, and chronic or suppurative, also called empyema.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—There is first hyperæmia and reddening of the pleura with dryness from checking of its normal secretion, there is swelling from transudation of serum into the perivascular spaces, and multiplication of connective-tissue cells with loss of the normal glistening of the pleural surface due to degeneration and exfoliation of superficial endothelial cells. Then follow exudation of inflammatory lymph and effusion of serum to a greater or less extent; the former clinging to the pleural surface presents a rough, shaggy appearance; the latter gravitating to the lowest part of the pleural sac, usually holds in suspension shreds of fibrin, leucocytes, and endothelial cells. Thickening of the serous membrane results from multiplication, in it and in the fibrous exudate, of new connective-tissue cells; these mature, new blood-vessels form, making connection with the original vessels of the pleura, and organization of the exudate is the result.

Adhesions more or less extensive may form between opposing pleural surfaces, which become bound together closely by the plastic organization, or more loosely by fibrous bands and false membranes.

The pleural surface early in the inflammation may present irregular spots of ecchymosis surrounded by the more diffused redness; later, whitish spots of fibrous organization appear on the free surface. The effused serum is generally of a light yellow or greenish color, has a specific gravity of from 1,010 to 1,024, contains four to six per cent of albumen, and coagulates readily upon exposure. In these respects it differs from the fluid of hydrothorax, which contains but one per cent of albumen, its specific gravity being below 1,015. The amount of fluid varies; in acute pleurisy, it is not usually great, seldom occupying more than one-third or at most one-half of the pleural sac, and is very rarely sufficient to fill the cavity. In subacute pleurisy the quantity is often sufficient to fill the cavity and cause great distention of the side. In empyema the amount is seldom greater than in acute pleurisy.

The processes of pleuritic inflammation vary with the causes and severity of the affection. The effusion takes its character from the presence of serum, fibrin, endothelial cells, blood, and pus in varying quantity and variously combined. The products of inflammation in mild cases may be chiefly fibrinous with little or no serous effusion; hence the so-called plastic or *dry pleurisy*. If fibrinous exudate and pleural thickening are marked and serous effusion is copious, we have the *sero-fibrinous* form. If infective inflammation occur pus results, and we call it *empyema*. The purulent accumulation in these cases swarms with the characteristic streptococci and staphylococci of suppuration, and in some instances the so-called diplococci of pneumonia and bacilli of tuberculosis may be found, though they are difficult of demonstration.

Hemorrhagic pleurisy occasionally complicates purpura hemorrhagica, cancer, scorbutus, and tuberculosis, or may result from the lighting up of a new inflammation in an old pleuritis.

Serous pleuritic effusions after remaining for a time are usually gradually absorbed, but purulent accumulations never to any great extent. In the latter the fluid tends to perforate the surrounding wall either to appear externally or to empty itself into an adjacent cavity or organ. The solid portion of an effusion may be absorbed after undergoing fatty metamorphosis, but not infrequently, sooner or later it becomes the seat of tubercular degeneration; or it may become incapsulated and remain so for years; or it may be the seat of calcareous deposition. Cases are reported in which the fibrous exudate covering an entire lung had been the site of such deposit. Aside from these characteristics of an inflamed pleura, certain pathological conditions result from the effect of the process upon adjacent structures. Inflammation usually extends to the lung tissue immediately beneath the pleura, giving rise to exudation which occludes some of the alveoli. It may also by extension cause pericarditis. The pleuritic effusion may be sufficient to cause complete collapse of the corresponding lung.

The compressed lung, upon disappearance of the fluid, tends slowly to re-expand unless pressure has been too long continued, in which case carnification of the organ results, and it remains as a small, compact, leathery mass, a suitable nidus for subsequent disease. Its complete expansion in any case is apt to be limited by the formation of cicatricial bands, and the great vessels may suffer serious compression.

ACUTE PLEURISY.

For convenience of description, acute pleurisy has been divided into four stages by some authors: First, a dry stage; second, a plastic stage; third, a stage of effusion; and fourth, a stage of absorption. I prefer the division into three stages analogous to the three stages of pneumonia, calling the first the dry stage; the second, the stage of effusion; the third, the stage of absorption.

Etiology.—Acute pleurisy may be primary, or secondary to some other disease.

Predisposing Causes.—It occurs most frequently in winter and spring, in adults rather than children, and attacks preferably the male sex. Malnutrition and poor hygienic conditions favor its occurrence.

Exciting Causes.—The most common causes are exposure and rheumatism. In a weak person mental depression may be an exciting cause.

It may result from traumatism, even of slight character. It arises not infrequently from pneumonia, phthisis, pulmonary infarction, abscess, gangrene, or tumors; other causes are found in hemorrhage into the pleural cavity, pericarditis, costal or vertebral caries, abscess of the mediastinum, peritonitis, and hydatids of the liver; also in infective diseases, Bright's disease, pyæmia and septicæmia.

Symptomatology.—The usual symptoms of this disease are: A sharp, cutting *pain* in the side, aggravated by general and respiratory movements; *rapid and incomplete inspiration;* a short, *dry cough* and a hard, *rapid pulse*, with more or less disturbance of the digestive organs. Pain is especially severe on inspiration and apt to be located just beneath the nipple, though in children frequently it is less circumscribed. It is a more constant symptom in adults, but variable in duration; it usually diminishes as the general pyrexia appears, or with the occurrence of effusion.

The temperature is usually but slightly elevated the first day, 99° or 100° F. in adults, but in children 102° or 103° F. In pleuritic effusion of children, surface thermometry may reveal on the affected side higher temperature by one or two degrees, rising and falling with the increase and decrease of the effusion. While in very mild cases the subjective symptoms may be so slight as to attract little or no attention, in rare cases they may be so severe as to suggest pneumonia. Pleuritic symptoms are apt to be less marked in the feeble and cachectic. When a large effusion occurs, nausea and vomiting are frequently present and dyspnœa becomes a prominent symptom.

The most important *signs* of pleurisy are: short and catching respiration, friction fremitus on palpation, and friction sounds heard on auscultation. Over the collection of fluid after effusion has taken place, there is flatness and loss of vocal fremitus and respiratory murmur. The upper line of flatness changes with the position of the patient (Fig. 18).

In the first stage we have in the beginning simply dryness of the pleura, and shortly afterward an exudation of inflammatory lymph.

By inspection we observe jerking or interrupted and incomplete respiration, with diminution of the expansive movements of the affected side. This catching respiration results from the patient's efforts to limit inspiratory movement, in order to prevent pain. This sign, though nearly always present, is not diagnostic of pleurisy; for in intercostal neuralgia and in pleurodynia may be found similar movements.

If the patient is sitting or in a semi-recumbent position, his body

will be inclined toward the affected side. If recumbent, he is likely to be lying on the unaffected side.

Occasionally, especially in children, the patient's efforts to restrain the movements of the affected side result in temporary spinal curvature toward that side.

On palpation, no signs will be obtained in the early part of this stage; but a little later friction fremitus may frequently be detected, and the vocal fremitus may be found diminished. Pressure usually elicits deep-seated tenderness. Mensuration yields no additional signs.

Percussion yields no signs at first; but when plastic exudation has taken place, dulness, in proportion to the amount of exudation, will be elicited. The dulness is always less marked at the end of forced expiration than during normal respiration.

Auscultation early in this stage discovers a feeble respiratory murmur with jerking or cog-wheel respiration, and in some instances, just at the end of inspiration, a feeble, grazing friction sound. When plastic exudation has taken place the respiratory sounds are still more feeble, and the friction sound becomes distinct, on both inspiration and expiration, but usually most intense with the latter. This may have any of the characteristics of friction sounds, as rubbing, grazing, creaking, or crackling. It may not be obtainable except on cough or deep inspiration, and will not be heard if the inflammation is confined to the mediastinal or diaphragmatic pleura. At this stage the vocal resonance is somewhat diminished.

In the second stage of pleurisy by inspection we still observe diminished respiratory movements, but not the interrupted respiration noticed in the first stage, perhaps also an apparent increase in size of the affected side; but sufficient fluid to dilate the side of the chest is exceptional in acute pleurisy.

In palpation the vocal fremitus is absent over the effusion. Rarely, distinct fluctuation can be obtained. The apex beat of the heart will be found crowded to the right or left, according to the seat and amount of the effusion. If the pleurisy is upon the left side, the heart is crowded to the right; if upon the right side, it is displaced in the opposite direction.

Exceptional.—In very rare instances of serous effusion, the vocal fremitus is not lost.

Percussion over the lower part of the chest yields flatness, extending upward to the surface of the fluid. The height of this surface is not altered by deep inspirations or forced expirations, but its relations are changed by alterations in the patient's position, unless the effusion entirely fills the pleural sac or there are complete adhesions above its surface.

Above the fluid the resonance is exaggerated, and in exceptional cases it may have a vesiculo-tympanitic or amphoric quality.

Investigations by Damoiseau, of Paris, and more recently by the late Dr. Ellis, of Boston, show that usually, when the pleural sac is no more than one-fourth or one-third filled, the upper surface of the fluid corresponds to a curved line known as the letter S curve, termed by Ellis the curved line of flatness (Fig. 19).

G. M. Garland, in his monograph on Pneumo-dynamics, describes this curved line as follows: "Its lowest point is found behind, near the spinal column. From this point it curves upward and outward across the lateral region, where it is highest; and from this point it proceeds almost horizontally forward to the sternum." The experiments of Garland demonstrate that, instead of a gradual rising of the fluid in

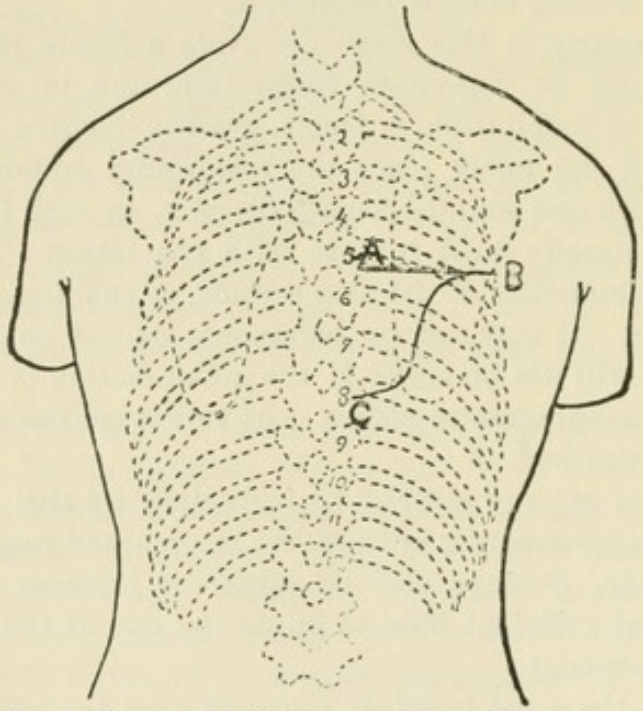

FIG. 19.—CURVED LINE OF FLATNESS IN PLEURISY, POSTERIOR VIEW (GARLAND). C. B, Letter S curve; A, B, C, triangle of dulness.

the lower portion of the chest, carrying the lung above it, and maintaining a horizontal surface, as is usually supposed, its upper line nearly corresponds to the natural outline of the base of the lung. This is supposed to be due to the elasticity of the lung, which holds the fluid in this unnatural position. I refer those interested in this matter to Garland's monograph for a complete exposition of the subject.

If a line be drawn horizontally backward from the highest point of the curved line of flatness in the lateral region to the spinal column, a somewhat triangular space will be left between it and the posterior part of the curved line of flatness. This space is termed by Garland the triangle of *dulness* (Fig. 19). It is bounded below and externally by the letter S curve, internally by the spinal column, and above by a line drawn backward from the highest point of the curved line in the lateral region. This superior boundary is not necessarily horizontal, but it

may be so considered for the sake of illustration. In this triangular space we have no fluid, but the resonance is less than above it. This dulness is due to partial compression of the lung against the spinal column. In order to recognize the curved line throughout its entire extent, we must not compare the affected with the sound side posteriorly, as it is not the distinction between resonance and flatness which we wish to obtain, but the distinction between dulness and flatness. Percussion should be made in perpendicular lines at several places, either from above downward or from below upward. By this method, we easily distinguish between the dulness over the compressed lung and the flatness over the fluid, and between the character of the resonance in these positions and that of the lung above them. Failure to recognize the true character of the percussion note in these different localities

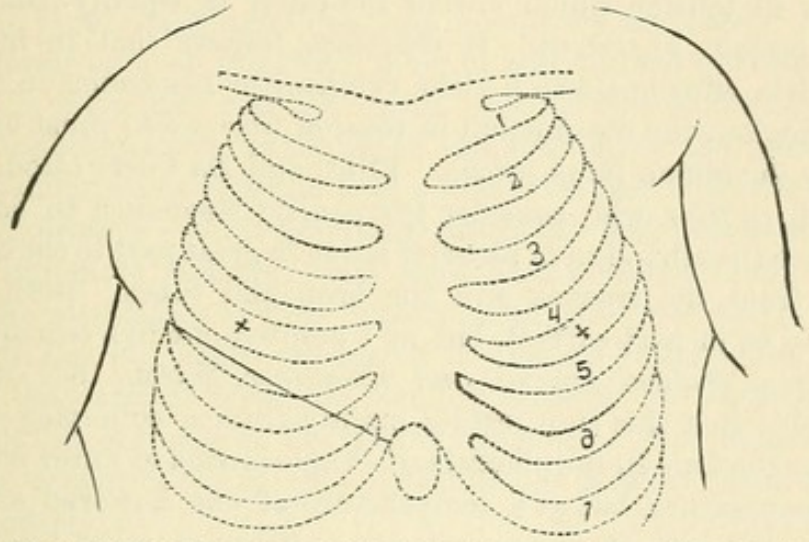

FIG. 20.—CURVED LINE OF FLATNESS IN PLEURISY, ANTERIOR VIEW (ELLIS). Letter S curve, anterior view.

has caused authors to describe the upper surface of the fluid as corresponding to a horizontal line. If we recollect that the fluid in the pleural sac conforms itself more or less perfectly to the natural contour of the base of the lung, we shall understand why the line does not undergo greater changes with alteration in the position of the patient. Suppose, for instance, that we find the level of the fluid, in front, at the fifth rib, when the patient is in the erect position; upon causing him to lie on his back, according to the generally accepted opinion, the line of flatness should still remain horizontal, and would then be found running longitudinally along the lateral region. In fact, however, this never occurs. On the contrary, the line of flatness is not likely to be depressed in front more than one or two inches by this change in the patient's position, and it will be found running more or less obliquely downward and backward, instead of longitudinally.

When the pleural cavity is nearly filled with fluid, we frequently get tympanitic resonance over its apex, especially if the patient is recum-

bent. In attempting to explain this phenomenon, we are once more confronted with the opposing statements that tympanitic resonance is low pitched and that it is high pitched. Fraentzel—who believes the tympanitic resonance to be low in pitch—in giving the reasons for this sign, quotes from Wintrich and Traube, who claim that the pitch in pulmonary percussion is dependent upon two elements: first, the volume of air beneath the point percussed; second, the tension of the lung tissue; claiming also that the pitch of the percussion sound is directly proportionate to the tension and inversely proportionate to the volume of the oscillating column of air. In other words, as the lung is diminished in volume the pitch is raised; or as it again approaches the normal size, the pitch is lowered according to the amount of air which it contains; and as the tension of the lung is increased the pitch is elevated. Therefore if the diminution in volume which raises the pitch and the diminution in tension which lowers the pitch be equally balanced, the pitch will remain unaltered. It therefore follows that in moderately large pleuritic effusions which yield tympanitic resonance in the infraclavicular region, the diminution in tension (*low pitch*) must exceed the diminution in volume (*high pitch*). Flint, and Da Costa (Medical Diagnosis, 1890, p. 265), who consider tympanitic resonance to be of high pitch, believe that this sign in pleurisy is due in great part to the conducted resonance from the trachea and the bronchial tubes. Both of these reasons may be in part correct, but, as I pointed out in a communication to the *Chicago Medical Journal and Examiner*, March, 1877, it is more than probable that this sign results mainly from a collection of watery vapor above the fluid in the pleural sac. Vaporization of water occurs even at a low temperature, but at a temperature of one hundred and one or two degrees Fahrenheit, under ordinary pressure, it takes place rapidly. This process must therefore be going on constantly when fluid collects in the pleural cavities, and as soon as the serous surfaces become so altered by inflammation that they are incapable of absorbing the vapor as rapidly as it is formed, it will collect above the fluid until the tension becomes sufficient to prevent its further formation. A cavity so formed, filled with watery vapor, must yield tympanitic resonance. I am convinced of the correctness of this theory by experiments not only with fluids outside of the body, but also on patients with the pleural cavity almost filled with fluid, and in whom when recumbent tympanitic resonance was plainly discernible, just beneath the clavicle, while on inversion of the patient so that the base of the chest was the highest, tympanitic resonance would be found over a small area at the base of the pleural sac.

Biegauski (Schmidt's *Jahrbuch*, August, 1889) calls attention to a new sign of right-sided pleurisy; increased cardiac dulness laterally appears with effusion even in small amount, caused, he thinks, by atelectasis of the middle lobe of the

lung, so exposing more of the heart. This increased dulness is said to remain for a year or more after absorption of the effusion.

By auscultation the respiratory murmur above the level of the fluid is often found slightly exaggerated. The vesicular murmur cannot be heard over the fluid excepting in a small zone near its upper level, where the sounds are feebly transmitted from the lungs. Over the fluid, vocal resonance is either lost or the voice-sounds are indistinct and distant. Sometimes consolidation of the lower part of the lung causes ægophony near the upper surface of the fluid. Often a few friction sounds may be heard in the same position, but none over the rest of the fluid.

During the third stage of pleurisy the signs denote gradual return to a healthy condition. Distention becomes less, respiratory movements are freer, and the vocal fremitus gradually appears first at the upper portion of the chest. The upper limit of the liquid, as ascertained by percussion, slowly falls until the fluid is entirely absorbed. Sometimes, over the lower part of the chest, more or less dulness persists for a long time, or the resonance may not again become normal, owing to the remaining inflammatory lymph or to thickening of the pleura, which may permanently separate the lung a short distance from the chest wall.

The respiratory sounds gradually return, at first feeble and distant, but growing more distinct, until they finally become normal. Occasionally the respiratory sounds remain harsh and tubular in quality, on account of the imperfect expansion of the air vesicles, and bronchial breathing may remain near the vertebral column for some time. Usually, as the two surfaces of the pleura again come into contact, friction sounds are obtained, which may continue for a short time only or for several months.

The heart and the abdominal organs gradually return to their normal positions, as shown by percussion and auscultation.

In some rare cases, however, when the heart is crowded to the right of the sternum by an effusion into the left pleural sac, adhesions take place which permanently retain the organ in its abnormal situation. Sometimes the absorption of a large and long-continued effusion in the right sac is followed by a permanent dislocation of the heart to the right of the sternum, due to the tendency of the surrounding parts to fill the space which should be occupied by the unexpanded lung.

If the air vesicles cannot fully expand, owing to the partial disorganization of lung tissue from long-continued compression or because the lung has been bound down by inflammatory adhesions, the chest may not again attain its normal condition. There will be consequent loss of motion and retraction of the affected side, with more or less dulness upon percussion and feeble or suppressed respiration. In the most protracted cases the upper portion of the lung becomes only partially expanded, and in this region there will be dulness upon percussion, with

deficient vesicular murmur and broncho-vesicular respiratory sounds, together with exaggerated vocal resonance.

DIAGNOSIS.—The essential points in the diagnosis of acute pleurisy are: the indistinct chills, the sharp pain in the side, friction fremitus and murmurs; flatness on percussion with change in the level of fluid by changes in the patient's position, with absence of vocal fremitus and absence or great diminution in the intensity of all respiratory and vocal signs over fluid effusions.

The differential diagnosis of pleurisy is usually easy, yet various diseases have been mistaken for it. The affections liable to cause error in diagnosis are pleurodynia, intercostal neuralgia, pericarditis, pneumonia, phthisis, collapse of the lung due to pressure on a main bronchus, cancer of the lung, aneurism of the aorta, and enlargement of the liver or spleen.

Pleurisy is only likely to be mistaken for *pleurodynia* or *intercostal neuralgia* in the first stage of the acute variety, when the pain and consequent impairment of the respiratory movements and murmur are the same as in the latter affections. The distinction may be made by remembering that the pain of pleurodynia is apt to be fugitive, shifting, and often bilateral, and is likely to be increased by slight pressure and by muscular contractions. The pain in intercostal neuralgia is confined to one, two, or three tender points along the course of the intercostal nerves; the neuralgic diathesis is commonly to be found in this affection and frequently coincident uterine disease. On the other hand, the pain in pleurisy is deep-seated, and although there is tenderness on pressure, it is not confined to isolated points along a nerve; and by auscultation we detect a friction sound which is not obtained in pleurodynia or in intercostal neuralgia. In these latter there is usually no fever.

The diagnosis between *pericarditis* and pleurisy affecting the left side is based upon the locality of the pain and the friction sounds, and the relation of the latter to the respiratory movements.

The pain of pericarditis is located in the præcordial region; that of pleurisy more laterally. The friction sound in pericarditis is heard most distinctly at the left border of the sternum near the fourth costal cartilage; that of pleurisy usually farther to the left and lower down. The friction sound in pericarditis is independent of the respiratory movements, and does not cease when the patient holds his breath. In pleurisy these sounds are not heard except during respiration.

Exceptional.—The action of the heart may cause a friction sound between the anterior portions of the left pleura which will not disappear when respiration ceases, but this is extremely uncommon.

The diagnostic points of pleurisy as distinguished from *pneumonia* are as follows:

Symptoms.

PLEURISY.	PNEUMONIA.
Chill absent or slight.	Onset with marked chill.
Temperature low, rarely above 102° F.	Fever high, 102°–105° F.
Slight prostration.	Marked prostration.
Cough hacking, dry.	Cough followed by tenacious, often bloody or rusty sputum.
Respiration jerking.	Respiration panting.
Stitch-like pain, usually below the nipple.	Pain usually duller and less intense.
Aspiration gives additional evidence of effusion.	

Inspection.

Countenance notably pale and anxious at the onset.	Countenance apt to be flushed.
Decubitus often on the affected side.	

Palpation.

Vocal fremitus diminished or absent.	Vocal fremitus increased.

Percussion.

Flatness and sense of resistance over the fluid.	Dulness rather than flatness.
Displacement of adjacent organs.	No displacement.

Auscultation.

Vocal sounds feeble.	Vocal sounds exaggerated.
Inspiratory and expiratory friction sounds prior to effusion.	Crepitant râles and later numerous moist râles.
Respiratory sounds feeble or absent over effusion.	Vesicular murmur feeble or absent, but bronchial breathing distinct in second stage.

The most distinctive sign of pleuritic effusion is absence of vocal fremitus over the affected part, instead of increased fremitus as in pneumonia.

Pleurisy is distinguished from *phthisis* by the history and by the same signs which differentiate it from pneumonia, also by the fact that phthisis, affecting the greater part of the lower lobe of one lung, will usually affect the apex of the opposite lung, whereas the signs of pleurisy are usually confined to the lower part of one side. In phthisis the signs usually progress downward; in pleurisy, upward.

Many signs similar to those of pleurisy with extensive effusion may appear in *collapse of a lung* from compression of its main bronchus, viz., loss of motion of the side, absence of vocal fremitus, dulness or flatness on percussion, and absence of respiratory and vocal signs. When these signs exist, the diagnosis must be based mainly on the position of the heart. Moderate pleuritic effusions, where no adhesion of the pleural surfaces has taken place, would be easily differentiated from the condition under consideration by changes in the level of the fluid. But where the effusion is circumscribed, or when it completely fills the

pleural cavity, this sign would not be present. In pleurisy with considerable effusion, the heart is more or less displaced toward the opposite side. This does not occur in collapse of the lung.

The essential difference in the signs of these two conditions may be seen at a glance in the following table:

PLEURISY.	COLLAPSE OF LUNG FROM COMPRESSION OF THE MAIN BRONCHUS.
Heart usually more or less displaced to opposite side.	Heart not displaced.
Side often distended. Side not retracted excepting in protracted cases.	Side not distended, may be retracted, and would always be retracted except that collapse of the air vesicles causes diminished pressure on the organ. This favors dilatation of the blood-vessels, and sometimes causes congestion with exudation which fills the air vesicles and distends the lung to its normal size.

Dulness usually begins near the middle of the lung *in pulmonary cancer*, and progresses irregularly in different directions, leaving here and there patches of normal resonance surrounded by flatness. In pleurisy flatness begins at the base of the chest and is uniform. The constitutional symptoms of the two diseases are usually different.

The occurrence of empyema with perforation of the chest walls, in the course of the aorta, might cause a pulsating tumor which would closely simulate *aneurism of the aorta*. It would be distinguished from the latter disease by the presence of signs of empyema in the lower part of the chest.

Pleurisy of the left side is distinguished from *enlargement of the spleen* by the following points. An enlarged spleen seldom encroaches much upon the thorax, and therefore causes little or no distention of the side, and no bulging of the intercostal spaces or displacement of the heart. Upon percussion, dulness is found to extend in front higher than behind, and the level of its upper surface does not materially change with changes in the patient's position. There is also a large area of flatness below the diaphragm.

Even skilful diagnosticians have frequently mistaken *enlargement of the liver* for pleuritic effusions. The differential signs will be seen in the following table:

PLEURITIC EFFUSIONS.	HYPERTROPHY OF THE LIVER.
Inspection.	
Frequently, bulging of the intercostal spaces.	There may be bulging of the chest, but the intercostal spaces are not especially prominent.
Palpation.	
Occasionally, fluctuation.	No fluctuation.

Pleuritic effusions.	Hypertrophy of the liver.
Percussion.	
Dulness extending higher behind than in front.	Dulness extending in front higher than behind, because the shelving border of the lung posteriorly intervenes between the liver and the thoracic walls.
The line of absolute flatness usually varies with changes in the position of the patient, and *is not* depressed or elevated during inspiration or expiration.	The line of flatness is not materially affected by changes in the patient's position, but *is* depressed and elevated by inspiration and expiration.
Auscultation.	
The respiratory murmur is heard in front, at a lower level than behind, and this level is not materially affected by deep inspiration.	The respiratory murmur is heard behind at a lower level than in front, and this level is depressed during deep inspiration and elevated in expiration.

Prognosis of acute and subacute pleurisy. In ordinary cases of acute pleurisy recovery usually occurs within two or three weeks, but they may lapse into the subacute and chronic forms. A permanent lesion usually remains in some part of the pleural sac after sero-fibrinous pleurisy (Loomis), frequently in the form of thickening and adhesions; these predispose to repeated attacks, resulting in greater pleural thickening, connective-tissue hyperplasia and contraction, thus limiting the function of the lung and favoring attacks of bronchitis and the inroads of phthisis.

Diaphragmatic pleurisy in the dry form is common and generally results in adhesions, which may fix the diaphragm as high in some cases as the fourth rib, usually at the seventh or eighth, thus greatly diminishing the vertical diameter of the chest cavity, rendering subsequent thoracentesis, if necessary, dangerous, and favoring rupture of the diaphragm in the sudden strain of severe bodily exertion.

Subacute pleurisy may be protracted for months, resulting in permanent crippling of the lung from compression, and it may be in emphysema of the opposite organ; or the fluid may become purulent, especially in children. Pleurisy complicating grave disorders such as pyæmia, septicæmia, or Bright's disease is obviously unfavorable. In the latter affection and in very acute pleurisy, effusion may be so rapid and copious as to cause death in a day or two.

Extreme compression of the lung in any case invites sudden congestion or œdema of its fellow, and consequent death.

Danger of sudden death from compression of the heart, according to Leichtenstern (*Deutsches Archiv für klinische Medicin*, Band IV), is greater if a large effusion occurs on the right side, probably owing to the greater weakness of the walls of the right ventricle. However, in children a large effusion on the left threatens sudden fatal syncope from its effect in twisting the great vessels.

TREATMENT.—The patient should be kept quiet in bed, and put upon an unstimulating diet unless great weakness demand the opposite. Talking should be prohibited, and all voluntary motion avoided. The respiratory movements may be restricted by strapping the side with strips of adhesive plaster running diagonally, from above downward and forward and downward and backward, and also horizontally; a broad strip of rubber plaster applied during expiration, or a wide elastic bandage, may be employed for the same purpose. When these are not used, hot poultices may be beneficially employed.

Opiates or some of the more recent analgesics, such as antipyrine, acetanilide, or phenacetin, which are to be preferred when there is much fever and in most cases where the pain is not extreme, should be given in sufficient quantity to relieve pain. Loomis recommends the application of a constant galvanic current to the affected side for the relief of pain, which continues after the subsidence of friction sounds. Sometimes the pleural sac rapidly fills with serum, and the question of aspiration will be suggested. With regard to this, the following rule is important: Do not aspirate in acute pleurisy until about the middle of the second week or until all acute symptoms have passed, unless compelled to do so to relieve great dyspnœa. In the third stage of the disease, tonics and potassium iodide, with counter-irritation by blisters or iodine, are indicated. Absorption of the fluid may also be favored, by free sweating brought about by the use of jaborandi, pilocarpine, or the hot-air bath, and by such diuretics as squills, comp. spts. of juniper, and potassium bitartrate, acetate, or iodide. Sodium salicylate, or salol in large doses, is recommended as sometimes producing prompt subsidence of serous pleurisy where other remedies prove unsatisfactory (J. Drzewiecki, *Medical Record*, July, 1889).

SUBACUTE PLEURISY.

Subacute pleurisy, also called chronic pleurisy by some authors, consists of a low grade of inflammation of the pleura, most frequently characterized by mildness of the symptoms, absence of pain, and slight constitutional disturbance with the effusion of an excessive amount of serum often completely filling the pleural cavity.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—These having been already described under the general title Pleurisy, it only remains to be said that this is pre-eminently the "pleurisy with effusion." The morbid processes occurring in the pleura are less rapid than in the acute variety; the pleural thickening and formation of fibrous tissue is more extensive; the effused liquid larger in quantity; the results of pressure more grave.

ETIOLOGY.—The causes are similar to those of the acute form, but malnutrition and tuberculosis are the most frequent.

SYMPTOMATOLOGY.—The principal symptoms are *dyspnœa, loss of appetite, emaciation, vomiting,* and more or less *cough.*

Fever of from one to two degrees is common. Pain may be slight or altogether absent.

It is surprising how great the effusion may become in this affection before the difficulty in breathing becomes noticeable.

The *signs* are those of the second and third stages of acute pleurisy with extensive effusion (Fig. 21).

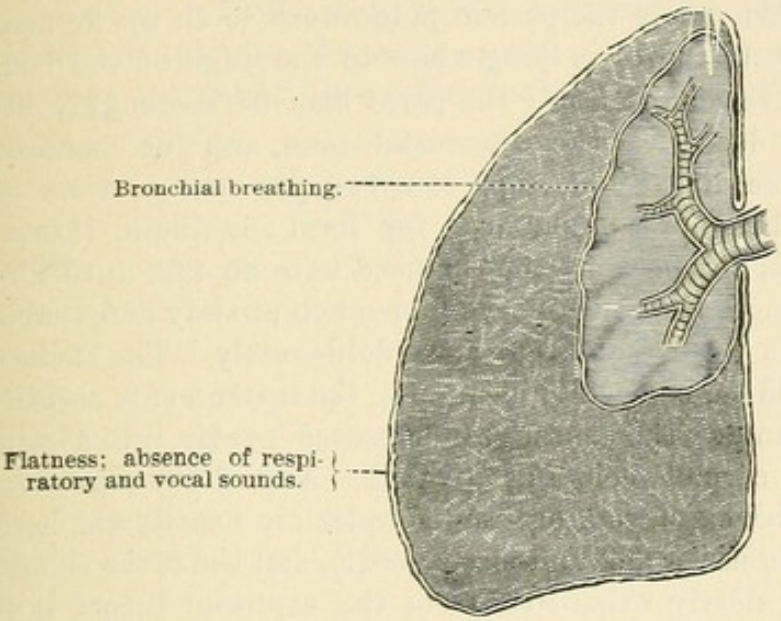

FIG. 21.—SUBACUTE PLEURISY.

DIAGNOSIS AND PROGNOSIS.—The subject of diagnosis and prognosis of subacute pleurisy has been included in that of acute pleurisy.

TREATMENT.—The indications are for *improved nutrition* and *removal of the effusion.*

Very moderate catharsis, diuresis, and diaphoresis, if employed short of exhausting depletion, especially in the more robust, are advisable, not so much to influence absorption of the pleuritic effusion as to favor improvement of the general nutritive processes. Mild counter-irritation is also useful. The diet should be nutritious, easily digested, and moderately stimulating, composed of animal and farinaceous broths, beef preparations, eggs, and in some cases such spirits as sherry and port. These, and bitter tonics, as the various preparations of hydrastis, calisaya, columbo, and gentian, combined with ferruginous remedies, and the employment of mercury and potassium iodide in alterative doses, best meet the first requirement.

If in a couple of weeks the fluid has not materially diminished, it should be withdrawn by an aspirator, providing there is sufficient to more than half fill the pleural cavity, or even when the collection is small if it causes dyspnœa or discomfort in the side. Whenever the cavity is completely filled and the heart displaced, even though no urgent

symptoms occur, no time should be lost in performing the operation. In cases of bilateral effusion, especially where there is cyanosis or great dyspnœa; when emaciation occurs with indigestion and feeble circulation; when pleural effusion complicates pericarditis, heart disease, pneumonia, severe bronchitis, or Bright's disease; or when the fluid becomes purulent—operative procedure must not be delayed. In operating, it is most convenient to have the patient sitting astride of a chair with the arms folded and resting upon the back of the chair, and the body inclined slightly forward; but if the patient is too weak to sit up, he may remain in the recumbent posture, lying close to the edge of the bed. General anæsthetics are seldom used; the parts may be thoroughly benumbed by injecting deep into the intercostal space, and just beneath the skin, with a fine needle, a few drops of a two per cent solution of cocaine, or of the solution recommended for local anæsthesia (Form. 140). It is well to tell the patient that he need have no fear until told the plunge is to be made, in order to save him much anxiety and enable the physician to make his examination more deliberately. The surface to be punctured should be surgically clean and the instruments aseptic. I find it convenient to dip the thoroughly cleansed needle into a mixture of equal parts of carbolic acid and olive oil. Any of the aspirators in common use may be employed, but the simpler are usually the best. It is generally best to use a medium-sized needle, and the cocks should be closed and the air nearly exhausted from the aspirator before it is introduced. The puncture is best made near the angle of the ribs in the sixth, seventh, or eighth interspace. It is my custom to make it high. When the pleural sac is only partially filled with fluid, we ascertain the upper surface of this, and make the puncture about an inch below it. If the operation is at the lower part of the chest, the needle is apt to strike the diaphragm, or, if this does not occur, as soon as a part of the liquid has been withdrawn, the diaphragm is forced upward against the needle, causing pain and preventing further withdrawal of fluid.

The skin should be drawn upward about half an inch by the ends of two fingers, which are then pressed firmly into the intercostal space; between them the needle is thrust inward and upward in the direction corresponding to the slant of the adjacent costal surfaces, to avoid the danger of striking a rib. When all is ready the patient should be forewarned of the sudden coming pain, and the needle plunged in until it enters the pleural cavity. The air cock is then opened and the fluid slowly withdrawn. During this procedure, if cough, pain, or dyspnœa or a feeling of constriction of the chest or weight upon the sternum occur, the aspiration should be discontinued at once, whether the fluid has all been withdrawn or not. The amount of fluid removed at one time is exceedingly variable, being from a few ounces to several pints, and not infrequently rapid absorption has been known to follow removal

of even a few drachms. The operation should be repeated within from five to ten days if the fluid reaccumulates. Usually after these measures the patient immediately improves, the appetite is better, weight increases, and the fever may entirely disappear. Subsequent treatment of the case should be of a tonic nature, and should include systematic and careful exercise of the muscles of the trunk, and breathing exercises. Recovery is sometimes greatly aided by a sea voyage or change of climate, especially to a high altitude when mountain-climbing will develop the respiratory muscles and the air cells will be expanded. The patient should be told that he must expect pain in the affected region on pulmonary and general muscular exercise, for some weeks or months.

CHAPTER VI.

PULMONARY DISEASES.—*Continued.*

CHRONIC PLEURISY OR EMPYEMA.

THE term empyema is applied to pleurisy when the inflammation is protracted and pus instead of serum occupies the pleural sac.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—If sero-fibrinous pleurisy become suppurative, the plastic elements undergo degenerative changes by the action of various micro-organisms, and are found to consist of pus cells and shreds and flakes of semi-purulent coagula immersed in serum. If the empyema be primary, leucocytes, round cells, and endothelial cells, more or less degenerate, appear on the pleural surface, to be washed by the serum to the bottom of the pleural sac. The lymphatics, cells, and pericellular spaces—in the serous and subserous tissues—contain active micro-organisms in greater or less number. The effects of pressure upon the heart and lungs in empyema do not differ from those which occur in pleurisy with serous effusion.

ETIOLOGY.—Empyema, according to Bouveret, is most prevalent during the first five years of life, and pleuritic effusions are more apt to become purulent in children than in adults. Whether idiopathic or not, it usually occurs in those of hereditary weakness or those who are debilitated by disease or irregular habits.

It may follow trauma or opening into the pleural sac of an abscess in the liver, lung, or thoracic wall. Pneumonia and typhoid fever are frequent causes, or it may complicate rheumatism, or scarlet fever and some other contagious diseases, or pyæmia or septicæmia. More recently influenza has been assigned as an occasional cause.

SYMPTOMATOLOGY.—The symptoms of empyema denote serious constitutional disturbance. The most important are: rapid pulse, dyspnœa, cough and pain, high temperature, dry brown tongue, hectic and night sweats, with loss of appetite, vomiting, and rapid emaciation.

The signs of this disease are much the same as those of subacute pleurisy, but usually the displacement of the heart and of other adjacent organs is greater in proportion to the amount of fluid. Contraction of the chest occurs when compression of the lung has so impaired its elasticity that it cannot regain its original volume after partial absorption of the fluid. The chest is then flattened on the affected side, the nipple depressed and nearer the median line.

Occasionally accompanying curvature of the spine may exist, with convexity toward the sound side. This phenomenon results because the dorsal muscles of the sound side are no longer counterbalanced by those of the affected side, which become paralyzed by the persistent pressure.

Ordinarily the level of the fluid does not vary with changes in the position of the patient, owing to the agglutination of the pleural surfaces immediately above the effusion. In this, as in other varieties of pleurisy, fluctuation is occasionally detected by palpation. Sometimes, with large effusions, especially in the left pleura, pulsation of the side is observed synchronously with the contraction of the heart. This condition is called *pulsating empyema*. If the pus breaks through the chest wall and appears beneath the integuments, the tumor thus formed generally pulsates strongly, and it might easily be mistaken for an aneurism if located in the course of the aorta instead of being at the lower part of the chest. Tumors of this kind often enlarge with inspiration and diminish in size with expiration.

Exceptional.—Rarely, empyema, instead of occupying its usual position at the base of the chest, may be confined to the upper part of the pleural sac, or to a small space about the root of the lung, or it may occupy two different and widely separated localities.

It is generally considered impossible to differentiate between serum and pus in the pleural sac; but Guido Bocelli, of Rome, claims that the distinction can be made by attention to the whispering vocal resonance. The whisper resonance, he claims, may be heard at the base of serous pleuritic effusions, but will not be conducted through pus. In making this distinction, two conditions must be secured: First, immediate auscultation must be practised, the ear being pressed firmly against the naked chest, and all external sounds excluded by closing the other ear; second, the patient must be so placed that the vibrations produced by whispering shall proceed from his mouth in a direction diametrically opposed to the listening ear.

Diagnosis.—Empyema may be suspected from the physical signs denoting pleural effusion, together with the symptoms significant of purulent inflammation, but the diagnosis can be made positive only by exploratory puncture.

Prognosis.—This is generally considered unfavorable. Chances of recovery lie in spontaneous opening and discharge of the pus, a very tedious process, or in its removal by operative procedure. Without such relief, the dangers are: death from sepsis, pyæmia, exhaustion, or from the effects of pressure upon the thoracic organs. In acute empyema, death may result within one or two weeks, but in the more chronic forms the patient may live for months, or even three or four years, or possibly longer. Children recover much more satisfactorily after operation than adults, but succumb more quickly without it. Leichtenstern considers the escape of pus in the empyema of children as an almost infallible indication of recovery. He believes that the cases of so-called spontaneous cure in children can be explained by the theory that the pleural

accumulation in these cases disappears by discharge through an opening into a bronchus.

Loomis states that when spontaneous opening occurs, about twenty per cent recover; but that when the pus has been removed by operative procedures, only about twelve per cent recover; but I have seen quite a series of cases in which evacuation of the pus by the method here recommended has been followed by recovery in about seventy-five per cent of the patients.

TREATMENT.—Pus in the pleural cavity must be removed. To this end various operations have been advocated.

Aspiration of the cavity repeated two or three times has in a few cases proved sufficient.

L. G. Fütterer, of Chicago, reported to me by personal letter six cases perfectly cured by aspiration of the chest and washing out of the cavity with a three-fourths of one per cent solution of clove oil in water that had been filtered and thoroughly boiled. This was injected and drawn off and followed by a permanent injection of a second quantity of this solution nearly equal in amount to the pus first evacuated.

Another method of treatment is by *pleurotomy*. An incision is made in the axillary region between the fifth and ninth ribs and parallel to them; double drainage tubes are inserted and a Lister dressing is applied. A convenient apparatus recommended by A. T. Cabot (Cyclopedia of the Diseases of Children, Keating, Vol. II, p. 712, 1889) is readily made from a piece of tubing cut half in two, folded upon itself and held in place through a shield by safety-pins.

Still others advise *resection of the ribs* either subperiosteal or not, and performed with various incisions and minor points of technique.

Authorities differ as to the invariable advisability of washing out the cavity. Bowditch (*Medical News*, January, 1889) claims that in two hundred and ninety-nine operations upon two hundred and fifty patients he found it necessary to wash out the cavity only once, and he considers it dangerous. De Cérenville (*Schmidt's Jahrbücher*, Band 218, Heft 1) reports six cases of epilepsy in children, following mechanical irritation of the pleural surfaces, as in irrigation, sounding, and probing. Equally high authorities favor irrigation.

A. B. Strong, of Chicago, strongly favors resection of the ribs, and reports thirteen cases (*Chicago Medical Record*, October, 1891) with only one death. Of these, however, twelve were acute and eight were in young children in whom the prognosis is usually favorable, whatever method of evacuation of the pus is adopted. He uses large drainage tubes (Fig. 23) well adapted for the purpose, readily made and easily worn.

W. M. Strickler, of Colorado Springs, Colorado (*Medical News*, May, 1887), advocates resection of the fifth, sixth, and seventh ribs, thorough digital examination of the cavity, removal with the fingers

of all fibrinous masses, separation of adhesions if necessary, and copious hot-water irrigation, followed by daily flushings. He reports excellent results in five adult cases.

Zimmerman and others consider *siphon drainage* as the most effective. A long, aseptic rubber tube is passed into the cavity through the

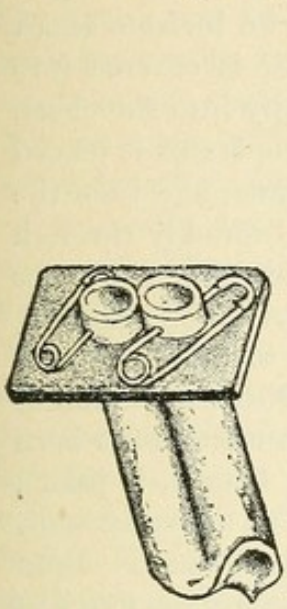

FIG. 22.—CABOT'S DRAINAGE TUBES.

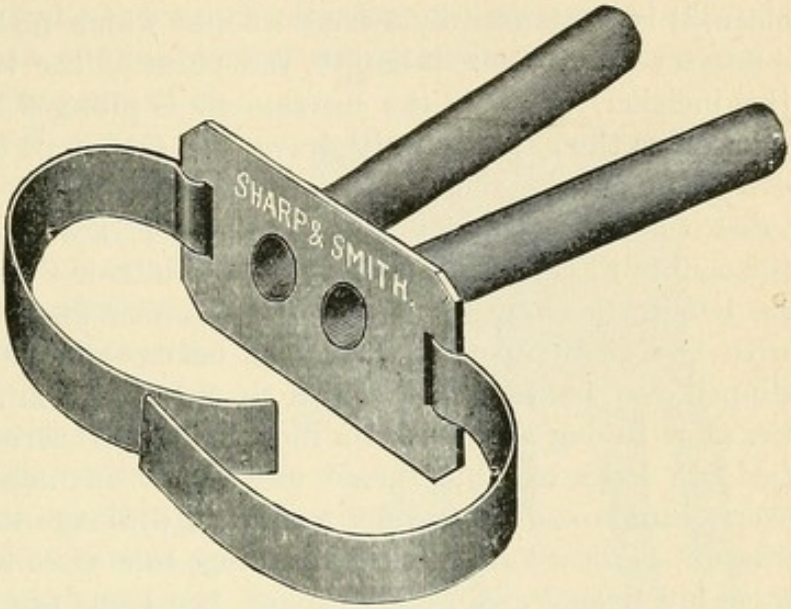

FIG. 23.—STRONG'S DRAINAGE TUBES. One-half size.

canula of a large trocar, a clamp closing the outer end of the tube. The canula is then slipped out, the tube is clamped between it and the chest wall, and the first clamp and the canula are removed. Connected to this tube is a glass one leading through a rubber stopper to the bottom of a bottle containing some antiseptic solution.

To secure a constant air-tight joint at the wound in thin patients where the tissues retract, the tube may pass through a rubber shield bound

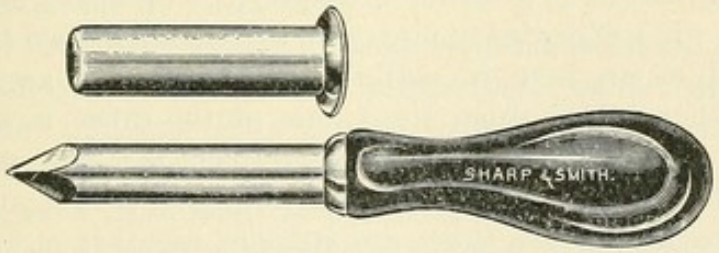

FIG. 24.—INGALS' FLAT TROCAR. One-half size. For introducing drainage tubes in empyema.

closely to the chest. Powell (*Canadian Practitioner*, 1887) successfully treated six cases by siphon drainage, using Nélaton's catheter passed through a rubber bandage fastened around the chest, and washed out the cavity by alternately raising and lowering the bottle containing a weak solution of carbolic acid.

With a single exception, I have never found resection necessary. The radical operation which I have employed with much satisfaction for many years is performed by means of a broad, flat trocar (Fig. 24) sufficiently large to admit the passage of two drainage tubes at once. If an anæs-

thetic is thought necessary, nitrous-oxide gas may be advantageously used, as its effects are quickly over; but it will usually be sufficient to inject deep into the intercostal tissues, as well as just beneath the skin, a few drops of a four-per-cent solution of cocaine such as recommended for local anæsthesia in the nose. The skin having been made thoroughly clean, it is punctured by a small scalpel, which makes an incision about a quarter of an inch in length, the point of the trocar is entered into this incision, and then the instrument is plunged boldly into the chest. As soon as the stiletto is withdrawn, the thumb of the operator is placed over the mouth of the canula to prevent the escape of pus; and then the tubes, which have been previously prepared, are slipped quickly through the canula to the required depth, the canula is withdrawn and the tubes are left in the chest. A bit of sheet rubber about three inches square, with two small openings near the centre and close together, is now slipped over the tubes and down to the chest wall. Next, a section of the same tubing about half an inch in length, through which have been tied two loops of stout thread each about an inch in length, is passed over a canula and slipped down over the drainage tube to the chest wall, where it is forced off upon the drainage tube close to the surface. Both tubes are treated alike, and through the loops are passed long strips of adhesive plaster, by which they are bound firmly to the chest wall.

The drainage tube is now perfectly under the control of the operator; it cannot possibly slide into the chest, and the adhesive straps keep it from being forced out a few days later when the tissues about it have retracted. The section of sheet rubber placed next to the chest wall acts as a valve preventing air from entering the chest at least for the first eight or ten days; that is, until the retraction of the tissue occurs about the tubing. A roller bandage is applied over the whole, the drainage tubes being allowed to protrude through it. In preparing the drainage tube, I take a piece of ordinary pure gum tubing about two feet in length and one-eighth of an inch in calibre and cut it half across near the middle; it is then folded upon itself, one of the tubes is perforated in several places extending about three inches from this cut end, the other in a couple of places, extending about one inch. About an inch and a half from this end the two tubes are stitched together at a single point with strong silk. The stitch is made through one of the perforations and knotted within the tube; then, if by any means it come loose, it is likely to be washed out. When folded upon itself and fastened in this way, one of the tubes is cut about half an inch shorter than the other, so that the operator may know subsequently which tube is perforated the greater distance from the end. About six inches from the end of the tube which is passed into the chest, a bit of thread is tied closely about it as a mark, in order that during the operation the surgeon may know how far it has been passed through the canula. Finally, the outer ends of the tubes are tied tightly, and the whole is made aseptic by soaking in a strongly carbolized solution. By thus closing the ends of the tubes, we are enabled to slip them through the canula, withdraw the latter, and

complete the operation even when the chest is much distended, without the escape of more than one or two ounces of pus.

After the dressings are completed, the drainage tubes may be bent upon themselves to seal them hermetically, while the ends are opened and connected by short glass tubes to longer rubber tubes, through which the cavity may be washed or drained according to indications. It has been my custom to wash out the pleural sac immediately with an antiseptic solution, and to have the washing repeated afterward once or twice daily for a couple of weeks, and subsequently less frequently until the sac is obliterated. This solution should be used at a temperature of 101° F. Between the washings the ends of the tubes may be bent upon themselves and tied, or they may be left hanging in a bottle containing some antiseptic solution, as thought best. When the patient is able to walk about, I usually allow drainage to go on constantly into a bottle which the patient carries in his pocket. In cases of empyema which have lasted for a long time, it is very important that about the fifth or sixth week after the operation the physician should ascertain whether the cavity is decreasing in size, which can be easily done by measuring from time to time the quantity of fluid required to fill it. Usually the pleural sac rapidly contracts until it will not hold more than four or five ounces; but after this, especially in adult cases of long standing, the contraction may be very slow. Here it becomes necessary to use stimulating injections, such as aqueous solutions of zinc sulphate, gr. ij. to iv. ad ℥ i.; iron sulphate double this strength; compound solution of iodine, ʒ ss. to ʒ i. ad ℥ i.; or copper sulphate, gr. v. to gr. xx. ad ℥ i. If iodine is used it will attack the drainage tubes so that they must be renewed every two or three days. Hydrogen peroxide, the commercial solution diluted with an equal volume of water, has been highly recommended to check suppuration, and a solution of the oil of cloves or emulsion of iodoform may be used for the same purpose. When the cavity has so far contracted as to hold not more than two or three drachms, the drainage tubes may be withdrawn about half an inch, left in this position for two or three days, then withdrawn as much farther, and so on until they are out of the pleural cavity, when the external wound readily closes.

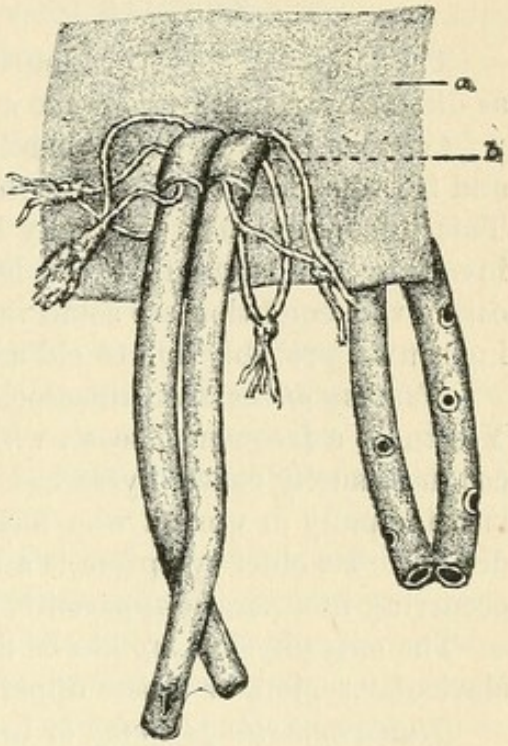

FIG. 25.—INGALS' DRAINAGE TUBES FOR EMPYEMA. *A*, sheet rubber; *B*, retaining apparatus.

The aim in the treatment of empyema is to give free exit for pus, and secure obliteration of the pleural sac by agglutination of its walls. Hence we encourage as far as possible the expansion of the lung, in some cases allowing the ribs to fall in, by resection, and bring the pleural surfaces together.

The importance of careful medical and general treatment adapted to the improvement of the patient's condition need hardly be emphasized.

PECULIAR LOCAL FORMS OF PLEURISY.

The following forms of pleurisy, though not entitled to be considered as distinct varieties, need some special consideration:

Circumscribed pleurisy usually occurs during the course of phthisis, and is responsible for many of the acute pains suffered by consumptives. This inflammation is generally limited to the small portion of pleura investing the lung where the lesions are superficial. The signs indicating this condition are some variety of friction sound, or a dry, creaking sound, probably due to old adhesions.

Pleurisy of the apex, unassociated with phthisis, is said by J. Burney Yeo to be a frequent disease, which he believes to be the cause of many coughs, usually called hysterical or stomach coughs. He has observed it principally in women who have been accustomed to wear low-necked dresses. Its chief symptom is a harsh, dry, shallow, or incomplete cough, occurring in a person apparently in good health.

The only physical *sign* to be detected is friction limited to the supra-clavicular region, or to the upper third of the scapular region.

Diaphragmatic pleurisy or inflammation of the pleura covering the diaphragm is not easily detected. According to Noel Gueneau, the following symptoms render its diagnosis more precise. Besides the pain elicited by percussion over the base of the chest on the affected side, there is a point of hyperæsthesia, due to irritation of the phrenic nerve, found at the intersection of two lines, one of which corresponds to the border of the sternum, and the other, perpendicular to it, follows and prolongs the border of the ribs. At the same time there is hyperæsthesia found between the sternal attachments of the sterno-cleido-mastoid muscles, and pain in the shoulder and in the infra-clavicular region of the same side. These are reflexes from irritation of the phrenic nerve. Neuralgia of the last intercostal nerve is also frequently present, and there is likely to be increased obliquity of the last rib on the affected side, and immobility of the hypochondrium. If the inflammation is on the right side, the liver is usually slightly depressed.

Percussion gives a high-pitched note over a narrow space, corresponding to the lower margin of the lung contiguous to the effusion.

On auscultation, the vesicular sound at the level of the collection of liquid is usually feeble, and accompanied with crepitant or mucous râles. Weakness of the inspiratory sound and prolonged expiration may

exist over the whole lung, due to compression of the bronchi by enlarged glands, which are said ordinarily to accompany this disease.

Multilocular pleurisy is rarely observed. In 1854, Wintrich wrote that it was impossible to distinguish, in the living subject, between *unilocular*, *bilocular*, and *multilocular* pleurisy, and this proposition is still generally accepted; but in a communication to the Académie de Médicine, of Paris, in 1879, Jaccoud declared the diagnosis possible when the following groups of signs are found coincidently with the ordinary symptoms and signs of pleurisy. He has observed two distinct semeiological types of the affection.

In the first, added to the ordinary signs of complete pleuritic effusions, the vocal fremitus, though lost over every other portion of the affected side, is found to be preserved along a line running forward from the spinal column, in a more or less regular semicircular course, toward the sternum, at a variable height. Vocal resonance and bronchial respiration are heard in the same locality, though wanting everywhere else.

This line indicates the position of the band of pleuritic adhesion dividing the pleural sac into two cavities. In these cases, he has found in the infra-clavicular region feeble and distant respiratory murmur and voice-sounds, with no tympanitic resonance.

In the second type, vocal fremitus, though more or less enfeebled, is obtained over the whole effusion, excepting sometimes a narrow zone of the breadth of one or two fingers, at the lower posterior part of the chest. Marked bronchial respiration and bronchophony are also found over the fluid, with perfect flatness on percussion, and no tympanitic resonance under the clavicle. In two cases he has been able to locate the fundamental partitions, by finding one or two zones where the vibrations were manifestly stronger than in other localities. The value of this diagnosis depends upon the proposition apparently established by Jaccoud's observations, that thoracentesis is not well borne in multilocular pleurisy, but that it seems rather to add greatly to the patient's danger. The essential points in the differential diagnosis between extensive pleuritic effusions of the unilocular, bilocular, and multilocular types are shown in the following table:

Unilocular pleurisy.	Bilocular pleurisy.	Multilocular pleurisy.
	Palpation.	
Loss of vocal fremitus.	Vocal fremitus preserved on a line corresponding with the band of adhesion, though lost above and below this line.	Vocal fremitus, though enfeebled, is present over the whole of the affected side, excepting a small zone at the base. Vocal fremitus is occasionally well marked in one or two limited zones corresponding to bands of adhesion.

Unilocular pleurisy.	Bilocular pleurisy.	Multilocular pleurisy.
	Percussion.	
Usually tympanitic resonance under the clavicle.	Flatness over the whole chest; no tympanism.	Flatness over the whole chest; no tympanism.
	Auscultation.	
Absence of respiratory murmur and vocal resonance, excepting over the compressed lung in the upper part of the thorax.	Bronchial respiration and bronchophony heard over a line corresponding to the pleuritic band, but wanting in other places, except over the apex, where they are indistinct.	Bronchial respiration and bronchophony marked over the seat of the whole effusion.

HYDROTHORAX.

Hydrothorax is a term applied to the presence in the pleural cavity of a dropsical effusion, which is non-inflammatory in character, thin, clear, yellow, or greenish. It has a low specific gravity, contains relatively little albumin, and coagulates less readily than an inflammatory effusion. The affection is usually bilateral, but may be confined to one side.

Etiology.—Hydrothorax may arise from any condition which impedes venous circulation, producing extensive passive congestion, as heart disease, notably mitral affection; diseases of the liver or kidneys; pressure of tumors and the like and venous thrombosis; it may also be the result of malignant disease, chronic blood-poisoning, exhausting discharges, or other morbid conditions producing general hydræmia.

The symptoms, of which dyspnœa is most marked, come on insidiously and are due to pressure of the fluid.

The signs will be similar to those of an inflammatory effusion.

Diagnosis will be based upon the signs and symptoms of the causative disease, the absence of inflammatory symptoms, the character of the fluid, and its usual bilateral position.

Prognosis will depend upon the cause.

Treatment will be directed to the primary morbid condition and to the immediate relief of the lung by aspiration.

PNEUMOTHORAX.

Pneumothorax consists of a collection of air or gas in the pleural sac, resulting from perforation of the pleura or from decomposition of pleuritic effusions (Fig. 26).

Etiology.—Air may enter the pleural cavity through a traumatic opening in the chest wall; through communication established with the stomach or œsophagus by ulceration or rupture; through openings into the lung from exploratory puncture, fracture of the ribs, or ulceration due to phthisis, empyema, abscess of a bronchial gland, or gangrene; or

through rupture of an emphysematous sac. About ninety per cent of all cases are of tubercular origin.

SYMPTOMATOLOGY.—The usual *symptoms* are sudden acute pain in the side, with severe dyspnœa and lividity of the lips and face; great prostration, accompanied with anxiety of countenance ; a clammy surface, palpitation, accelerated pulse, and in some cases collapse followed by death within a few hours. In other cases the symptoms are manifested insidiously, only becoming marked when considerable fluid accumulation has followed the entrance of air. This is the case in pneumothorax from emphysema. If it result from phthisis, the symptoms, especially pain, are very marked.

The most important *signs* are diminished movement and enlargement of the affected side; tympanitic resonance; respiratory murmur feeble or amphoric in character or wanting.

Inspection and mensuration reveal distention of the affected side, diminution or loss of the respiratory movements, with widening, and sometimes bulging of the intercostal spaces.

Palpation shows the vocal fremitus feeble or wanting, and the apex beat of the heart displaced toward the sound side.

By percussion, tympanitic or amphoric resonance is obtained over the collection of air. When distention of the side is extreme, the adjacent organs are displaced, and the tympanitic resonance, somewhat muffled and modified in quality, may be obtained for a considerable distance beyond the normal limits of the pleura.

Exceptional.—Occasionally when the tension is very great, the percussion note is so muffled as to seem almost dull. The bell sound may be obtained by percussion with two coins on one side of the cavity while the ear is placed opposite.

In auscultation, the respiratory murmur is feeble or absent according to the amount of air. The vocal sounds are altered in like manner. The respiratory murmur on the sound side is exaggerated. The heart sounds are feebly transmitted through the collection of air. Bronchial breathing may be heard over the compressed lung, in the inter-scapular space, and usually over the apex anteriorly. Amphoric respiration and voice are also obtained when a bronchial tube connects freely with the cavity of the pleura. The differential diagnosis between pneumothorax and emphysema, the only disease with which it is likely to be confounded, will be given under the latter.

PNEUMO-HYDROTHORAX.

Pneumo-hydrothorax signifies a collection of both fluid and air in the pleural sac. When the former becomes purulent, as is usually the case, the condition is termed pyo-pneumothorax. As the effusion of fluid is almost sure to follow in a few hours after the admission of air

into the pleura, the signs and symptoms of this disease and of pneumothorax are usually considered together, but the presence of both air and fluid in the pleural cavity causes some signs which are not found in pneumothorax. The splashing sound obtained by succussion is diagnostic. Metallic tinkling is also found in many instances (Fig. 26).

Inspection, palpation, and mensuration furnish the same signs as in pneumothorax or in extensive pleuritic effusions. There is absence of vocal fremitus, and displacement of the heart and adjacent organs, with distention of the side and loss of motion.

On percussion, tympanitic resonance is obtained over the air in the upper, and flatness over the fluid in the lower, part of the chest. The

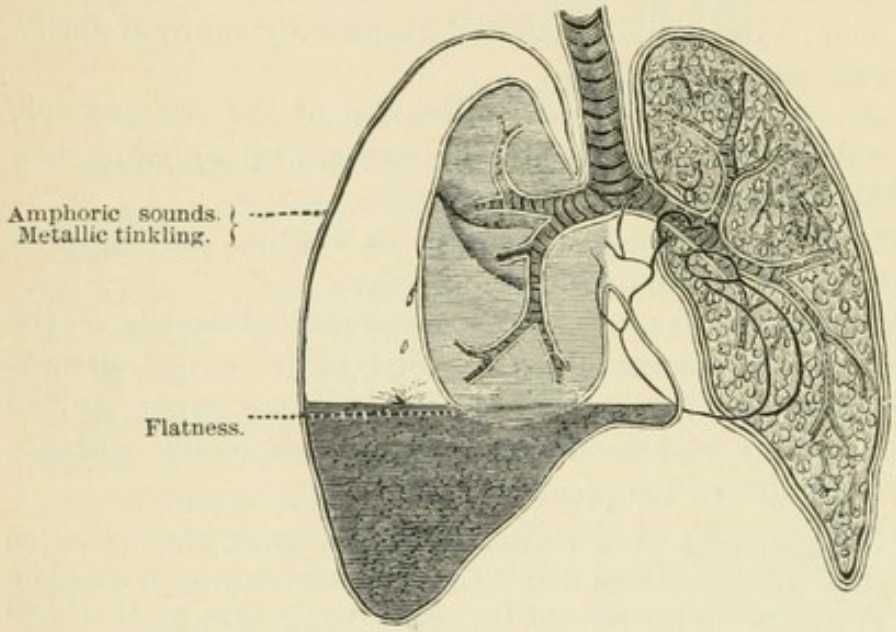

FIG. 26.—PNEUMO-HYDROTHORAX. Right lung compressed by air and fluid. Heart crowded far to the left.

line of flatness corresponding to the surface of the fluid changes with the position of the patient. Tympanitic resonance is not infrequently transmitted a short distance beyond the limits of the pleura, and even below the surface of the fluid, so that if only a small effusion is present this sign may be heard over the entire chest, and thus the presence of fluid escape our notice. Amphoric resonance is sometimes heard over the upper part of the chest.

Upon auscultation below the level of the fluid, the respiratory murmur is absent or very feeble and distant. Above this level it may be the same, or amphoric respiration may be heard. This latter may be limited to a small space near the point of perforation, which is likely to be located just in front of the angle of the fourth or fifth rib. Amphoric respiration may disappear and reappear again during the course of the disease, in consequence of the variation in the amount of fluid from day to day.

Usually bronchial respiration is heard over the compressed lung, where it lies against the spinal column.

The signs of phthisis, which in nine cases out of ten precede those of pneumothorax, are frequently found at the apex of the lung on the opposite side. Metallic tinkling is one of the signs of this disease. It may result from agitation of the fluid in coughing. The splashing sound obtained on succussion is characteristic. Vocal resonance is feeble or wanting, or amphoric, upon the affected side. The percussion resonance and the respiratory murmur upon the sound side are exaggerated.

DIAGNOSIS.—Pneumothorax and pneumo-hydrothorax are not likely to be mistaken for other diseases, though they are said to be closely simulated when there is complete catarrhal obstruction of the main bronchus on one side. They may possibly be mistaken for emphysema, chronic pleurisy, or diaphragmatic hernia.

Comparison with *emphysema* presents the following distinctive features:

PNEUMOTHORAX AND PNEUMO-HYDROTHORAX.	EMPHYSEMA.
Inspection.	
Prominence or bulging of one side, with loss of movement, especially at the lower part of the chest, but no falling in of the inferior ribs or intercostal spaces during inspiration.	Prominence of the anterior superior portion of the chest, usually upon both sides, with a characteristic lifting movement of the upper part and falling in of the lower ribs and intercostal spaces during inspiration, with frequently permanent contraction of the lower part of the chest.
Percussion.	
Tympanitic resonance over the upper part of the chest with flatness over the fluid, the line of flatness varying with changes in the patient's position. The heart is displaced to the right or left, according to the seat of the disease. Nearly always these signs are found on one side only.	Vesiculo-tympanitic resonance over the entire lung, but most marked at the superior portions; no flatness below. The heart may be covered by lung tissue, but it is not greatly displaced. The signs are usually found on both sides.
Auscultation.	
Respiratory murmur feeble or absent; if heard, the expiratory murmur is of normal duration, unless prolonged by consolidation of the lung, in which case it will be high pitched. Amphoric respiration and voice are observed if a bronchial tube connects freely with the pleural cavity. Metallic tinkling.	Respiratory murmur usually feeble and generally associated with bronchial râles. The expiratory sound is prolonged and low pitched. The respiratory sounds are sometimes harsh and tubular, but never amphoric. No metallic tinkling.
Succussion.	
Splashing sounds if fluid is present.	No splashing sound.

These diseases can be easily distinguished from *chronic pleurisy* by the physical signs obtained on percussion and auscultation. On inspection, palpation, and mensuration the signs are similar.

Pneumothorax and pneumo-hydrothorax.	Chronic pleurisy.
Percussion.	
Tympanitic resonance over the upper portion of the chest, flatness over the fluid.	Tympanitic resonance, if heard at all, is limited to a small space at the apex of the lung, usually immediately beneath the clavicle; flatness over the remainder of the affected side.
Auscultation.	
Often amphoric respiration and voice.	Never amphoric respiration or voice.

Diaphragmatic hernia is, fortunately, a rare disease. It possesses many symptoms and signs in common with pneumothorax, like which it causes distention of one side, displacement of the heart, diminished motion, tympanitic resonance, and feeble or suppressed respiration with metallic tinkling. The differential diagnosis depends mainly upon the history and the symptoms, as seen from the following table:

Pneumothorax.	Diaphragmatic hernia.
History and Symptoms.	
Usually follows phthisis or accidental perforation of pleura; the dyspnœa may come on suddenly or gradually.	Often congenital; at times dyspnœa comes on suddenly, and as suddenly disappears.
Auscultation.	
Amphoric respiration and metallic tinkling.	No amphoric respiration, and the metallic tinkling occurs independently of the respiratory movements, and is associated with rumbling of gas in the stomach or intestines, which usually form the contents of the hernia.

Prognosis.—Pneumothorax without pleuritis is rare, but when it does occur recovery not infrequently takes place. The prognosis in pyo-pneumothorax is very unfavorable. Death often occurs within a few hours or at most within a week or two. Rarely patients recover.

Treatment.—Pneumothorax and pneumo-hydrothorax call for essentially the same treatment. At first an opiate should be administered to relieve pain. When fluid has collected and dyspnœa is great, free drainage is advisable, especially if the fluid has become purulent; subsequently the case should be treated in the same manner as empyema.

Potain recommends replacing the fluid and air by sterilized air, and reports favorably (*Gazette des Hôpitaux*, April, 1889).

CHAPTER VII.

PULMONARY DISEASES.—*Continued.*

BRONCHITIS.

Bronchitis is an inflammation of the membrane lining the bronchial tubes. It affects both sides at the same time, and is therefore called a bilateral disease. Five varieties of bronchitis are recognized, viz., acute, subacute, chronic, capillary, and plastic bronchitis.

ACUTE AND SUBACUTE BRONCHITIS.

The symptoms and the signs of acute and subacute bronchitis are substantially the same, except that in the latter variety they are less marked.

Anatomical and Pathological Characteristics.—The morbid peculiarities in acute bronchitis are those of acute catarrhal inflammation affecting the larger bronchi. There is congestion, thickening, and softening of the mucous membrane; slight exfoliation of superficial epithelial cells, and hypersecretion of thin transparent mucus, frothy from admixture of air. This gradually becomes translucent, and finally yellow and viscid as more leucocytes escape from the engorged vessels. Slight ecchymoses may appear in severe cases, and the expectoration may show minute points of blood. This affection, usually confined to the larger tubes in adults, has a tendency in children and the aged to involve the capillary bronchi. The same conditions are present in subacute bronchitis, but less marked.

Etiology.—Old people and infants and those debilitated by disease or vicious habits or subjects of the gouty or rheumatic diathesis are most disposed to attacks of bronchitis, especially if exposed to improper hygienic conditions, whether of poor ventilation, defective drainage, or deficient food and clothing. It is more prevalent in climates exhibiting frequent and sudden atmospheric changes in humidity and temperature. Exposure to cold, especially when the body is overheated, or to excessive heat in a badly ventilated room is a frequent cause. Inhalation of irritating gases, particles of dust, or larger solid bodies frequently gives rise to bronchial inflammation. The occasional occurrence of the disease in seeming epidemics also suggests as the cause in some cases a micro-organism.

Symptomatology.—Bronchitis is ushered in sometimes with a chill; usually with pain in the back and extremities, attended by a sensation

of tightness or constriction in the chest, soreness beneath the sternum, a harsh cough and frothy expectoration sometimes streaked with blood.

The most important *signs* are absence of dulness and the presence of large and small, dry or moist râles on both sides of the chest (Fig. 17).

Inspection in acute bronchitis shows the chest movements normal or somewhat accelerated.

Upon palpation, the vocal fremitus is normal. If there is considerable secretion in the tubes, bronchial fremitus will be obtained, especially in children.

Exceptional.—In a few cases the movements are deficient in those parts of the chest supplied by bronchi that are partially occluded by a collection of the bronchial secretions.

On percussion, the resonance is normal.

Exceptional.—In some cases dulness is found, especially over the lower portion of the chest, due to accumulation of the fluid secretions. This dulness, however, may be removed by coughing and free expectoration.

By auscultation in subacute bronchitis we frequently hear simply a harsh and somewhat bronchial sound without râles. In acute, and in many cases of subacute bronchitis, sonorous and sibilant râles (Fig. 7) are obtained in the early part of the disease, and the vesicular murmur is more or less obscured by these signs. After from twenty-four to forty-eight hours, the secretions from the mucous membrane become abundant, and then the dry give place to large and small, moist, mucous râles. The intensity of these râles varies; sometimes they are feeble, at other times they may be heard at quite a distance from the chest. These signs are seldom continuous. Often they are heard during a few respirations, and are then displaced by deep inspiration or by forced expiration or cough. Mucous râles, even when numerous, may sometimes be entirely removed by free expectoration.

Some of the bronchial tubes may become so filled with mucus as greatly to diminish the intensity of the vesicular murmur, or even to suppress it in those portions of the lung supplied by the occluded bronchus.

Exceptional.—If the disease affect the smaller tubes, the vesicular murmur may be inaudible over the entire chest.

Vocal resonance is not altered.

CHRONIC BRONCHITIS.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—Continued inflammation of the bronchial mucous membrane produces thickening and irregularity of its surface. The surface is occasionally paler than normal and of a grayish color, but is usually of a deep pink or red and sometimes of a purple hue. The congestion may be diffused or in

patches, and the surface may be marked by numerous ecchymoses. The muciparous glands become swollen, and their secretions become thin and profuse or viscid and scanty, partially plugging the mouths of the ducts. These secretions may be serous, muco-purulent, or purulent, sometimes also fetid. The superficial epithelial cells degenerate and exfoliate to some extent, and rarely small ulcerations occur. The elastic longitudinal fibres and muscular coat of the bronchial wall become hypertrophied, the latter finally undergoing fatty degeneration. With loss of muscular tone and elasticity, bronchial dilatation may occur; in the larger tubes the soft parts may bulge out between the cartilages, forming sacculi in which secretions tend to collect and undergo putrefaction. The cartilaginous rings sometimes become calcified. As the process extends, the outer fibrous coat suffers hypertrophy, perhaps followed by peribronchitis with progressive induration, contraction and diminution in the calibre of the smaller bronchi. These morbid processes may result in asthma, bronchiectasis, emphysema, fibroid phthisis, lobular pneumonia, or dilatation of the right ventricle causing general venous congestion with consequent derangements of the gastro-intestinal and renal systems.

ETIOLOGY.—Chronic bronchitis occurs most frequently in those of middle age or advanced life, but occasionally in children. Generally malnutrition referable to a gouty or rheumatic diathesis or to the cachexia of chronic alcoholism, syphilis, or malaria is ultimately accountable.

Not infrequently, however, some pulmonary, cardiac, hepatic, or renal disorder, producing chronic pulmonary venous congestion, is the predisposing cause. The exciting causes are those of acute bronchitis, successive and obstinate attacks of which are invited by the constitutional bias.

Regarding the cause of the putrid condition sometimes developed in chronic bronchitis, Josef Lumniczer (*Wiener med. Presse*, May, 1889) has shown that the decomposition is probably due to a short curved bacillus, one and one-half micromillimeters in length, easily stained with aniline and cultivated at 96.8°–100.4° F., when it develops the peculiar fetid odor characteristic of the disease in man and the lower animals.

SYMPTOMATOLOGY.—The most constant symptoms are cough and expectoration, the character of which varies greatly according to the course of the inflammation, its cause, and the peculiarity of the individual. In severe cases and those of long standing, dyspnœa and labored breathing become prominent symptoms, and sometimes there is complaint of a feeling of soreness over the larger bronchi. Mild cases are characterized by slight or moderate cough with some muco-purulent expectoration.

The so-called winter cough, almost or quite absent in summer but recurring with the return of cold weather, may be mild at its beginning, but is apt to increase in severity from year to year.

In other subjects of bronchitis, cough and expectoration are more constantly present, but are variable in character. In certain cases, aptly termed bronchorrhœa, expectoration is very profuse, amounting sometimes even to two quarts in twenty-four hours, more or less serous in quality, but occasionally purulent. On the other hand, in so-called dry catarrh, expectoration is scanty and viscid; small, tough, translucent masses are expelled with extreme difficulty during severe paroxysms of cough accompanied with great muscular effort, reflex laryngeal spasm, choking, venous congestion of the face and neck, and perhaps vomiting.

The *signs* of chronic bronchitis differ from those of the acute affection principally in the greater abundance of mucous râles and in the scarcity of dry râles.

DIAGNOSIS.—The different varieties of bronchitis may be readily distinguished from each other by the history. They are liable to be mistaken for asthma, emphysema, pulmonary hemorrhage, and phthisis.

From *asthma*, bronchitis is distinguished by the symptoms and by the history. The spasmodic character of asthma, its sudden appearance, the great dyspnœa, and the history of former attacks are sufficient to establish the diagnosis.

The physical signs in these two diseases differ rather in degree than in kind, as shown in the following table:

BRONCHITIS.	ASTHMA.
In the early stage, dry râles, comparatively few in number. Later, during the second or third day, these give place to large and small mucous râles.	During the paroxysm, sonorous and sibilant râles are very abundant. The following day either the respiratory murmur may be normal, or an abundance of moist râles, due to the attendant bronchitis, may be present.

Simple bronchitis can be easily distinguished from well-marked cases of *emphysema*, but the latter disease is usually associated with more or less inflammation of the bronchial mucous membrane. The distinctive points in the two diseases are as follows:

BRONCHITIS.	EMPHYSEMA.
Inspection.	
Form and movements of the chest natural.	Prominence of the upper portions of the chest, barrel-shaped, with more or less constant expansion of the superior ribs, which are elevated in inspiration as though united in a single bone. Depression of the soft parts in inspiration, notably above the clavicles and sternum and at the lower portions of the chest.

Bronchitis.	Emphysema.
Percussion.	
Resonance normal. In exceptional instances slight dulness, especially over the lower part of the chest.	Vesiculo-tympanitic resonance more or less marked.
Auscultation.	
Vesicular murmur sometimes incomplete. The expiratory murmur not prolonged. Numerous râles.	The respiratory sounds feeble, but expiration greatly prolonged. Comparatively few râles.

Bronchitis is distinguished from *pulmonary hemorrhage* by the history and character of the sputa. The physical signs are identical, except the absence in the latter of dry râles, with the harsh quality of respiration often found in bronchitis.

Before the days of auscultation and percussion, chronic bronchitis was often mistaken for *phthisis*, but at present the physical signs render their distinction comparatively easy. They differ in the following particulars:

Bronchitis.	Phthisis.
Inspection.	
Form and movements of the chest natural.	Very early in the disease more or less depression over the affected region, with lessened expansion.
Palpation.	
Rhonchial fremitus, with normal vocal fremitus.	Vocal fremitus exaggerated.
Percussion.	
Resonance normal.	More or less dulness over the affected regions.
Auscultation.	
Râles found in this disease are equally diffused over both lungs. Expiratory murmur not notably prolonged. Resonance natural.	Râles and other signs of consolidation localized, limited to the portion of lung affected. Broncho-vesicular respiration and exaggerated vocal resonance.
Microscopic.	
No bacilli of tuberculosis in the sputum, nor elastic fibres.	Tubercle bacilli; elastic fibres.

Prognosis.—Acute bronchitis generally terminates in recovery within a few days or at most two weeks, even without treatment. It is seldom serious except in infants and the aged, or very feeble patients in whom it not infrequently develops into the capillary form. In the diathetic or cachectic, oft-repeated acute attacks are apt to occur and lead to chronic bronchitis. This latter form, though in itself rarely fatal, is

not easily curable and gradually tends to the development of asthma or more serious conditions, such as emphysema, bronchiectasis, atelectasis, and fibroid phthisis. Emphysema is peculiarly liable to result from dry catarrh of the bronchi.

TREATMENT.—In many cases the *acute* disease may be aborted, if seen early, by a hot stimulating draught at bed-time and the application of sinapisms over the chest; or a ten-grain dose of Dover's powder, quinine, or phenacetine, eight grains of antipyrine, five of acetanilide, or a moderately full dose of jaborandi or its active principle pilocarpine. Failing in this, we may use with advantage small doses of opium or of aconite; or troches of morphine, antimony, and ipecac compound (Form. 32); or a combination of morphine, ammonium chloride, and tartar emetic (Form. 1); or troches of compound licorice mixture (Form. 34) until the expectoration becomes free. Subsequently for cough it will be found beneficial to administer extract of cannabis indica (Allen's) gr. $\frac{1}{8}$ to $\frac{1}{6}$, extract of hyoscyamus (alcoholic) gr. $\frac{1}{2}$ to i., extract of nux vomica gr. $\frac{1}{4}$ to $\frac{1}{2}$, quinine hydrobromate gr. i. to ij., monobromated camphor gr. ij. to gr. iij. every four to six hours. Ammonium carbonate with small doses of morphine (Form. 5) is also useful. If the cough is not very troublesome, we may give potassium chlorate, ʒ ss. to ʒ i. daily in divided doses. Tonics may be required until resolution is complete.

The subacute form of the disease is treated in essentially the same manner.

Chronic bronchitis is often dependent upon some constitutional disease or diathesis which should receive our first attention, together with improvement as far as possible of the hygienic surroundings, and the correction of vicious habits. If it is due to the dartrous diathesis, arsenious acid, gr. $\frac{1}{40}$ to gr. $\frac{1}{20}$ three times a day, is especially indicated.

For the rheumatic or gouty diathesis, one or more of the following remedies may be given from three to five times a day: Potassium acetate gr. xv., resin of guaiac gr. x. to xv., or its ammoniated tincture ʒ ss. to ʒ i., potassium iodide gr. v. to x., tincture of colchicum ♏x. to xx. Even in these chronic conditions, salicylic acid or sodium salicylate is sometimes very beneficial, as also salol. In some instances undoubted benefit is derived from phytolacca. In a large percentage of these cases the digestive organs will be found at fault, and the greatest good will follow a judicious use of laxatives and the administration of remedies which will correct gastric and intestinal indigestion.

Many patients having the gouty or rheumatic diathesis are subject to eructations of gas or sensations of weight and fulness of the stomach shortly after eating, or to flatulence. The indications here are to hasten digestion and prevent decomposition of food. To this end I have often found of great service a capsule containing the following, given before meals and at bed-time or before and after meals according to the severity of the case:

℞ Capsici, ... gr. ss.
Hydrastinæ hydrochlorat., ... gr. ⅔.
Extract. nucis vomicæ, ... gr. ⅓.
Acid. salicylici, ... gr. ij.
Papain (Carica papaya), ... gr. iij.
M. Inclose in capsule.

The hydrochlorate of hydrastine here used is the article commonly known as such in medicine, but in pharmacy and chemistry it is more correctly termed hydrochlorate of berberine.

When the digestive trouble is mainly gastric, the salicylic acid is preferable to prevent decomposition; but if flatulence is a prominent symptom, salol will be found efficacious. Of the digestive agents, papaine is to me most satisfactory, but sometimes pepsin, pancreatin, and ingluvin are useful.

If the affection originates in syphilis, potassium iodide in full doses, with mercury bichloride, will have the best effect.

When the disease is of simple catarrhal origin, potassium chlorate, ʒ i. daily in divided doses, is one of the best internal remedies. Preparations of squill, senega, yerba santa, and eucalyptus are sometimes beneficial. Vegetable and mineral tonics, cod-liver oil, and preparations of malt are indicated for debility.

Persistent counter-irritation sometimes aids greatly in promoting a cure.

Locally, inhalations similar to those recommended for diseases of the throat (Form. 62, 63, 67, 69, 72, and 73) are beneficial, and in some instances, particularly where there is free secretion, great relief is obtained from the inhalation of thymol gr. ss. to i. to ʒ i. of liquid albolene.

Cough may be relieved by small doses of morphine and ammonium carbonate (Form. 5), by troches of morphine, or cannabis indica and terpin hydrate compound (Form. 33), and often by sedative inhalations (Form. 53–59). For dyspnœa, the nitrites in some form are specially beneficial. Great care should be taken on the part of the patient to avoid damp feet, exposure to night air, cold drafts, overheated atmosphere, and the inhalation of irritating substances.

When practicable, change of climate is often highly beneficial. When the bronchial secretions are profuse, the patient is likely to obtain most benefit in a higher altitude with dry atmosphere; if the secretions are scanty or tenacious, a moist climate with an equable temperature like that found at the seashore in Southern California or along the coast of the Gulf of Mexico is more salutary.

CAPILLARY BRONCHITIS.

Capillary bronchitis consists of an acute inflammation of the mucous membrane lining the capillary bronchial tubes. It usually results from extension of inflammation affecting the larger bronchi, and it affects both lungs at once.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—Evidence ren-

dered by autopsies indicates that capillary bronchitis without accompanying inflammation of the air vesicles is very rare. In most cases the mucous membrane of the larger tubes is first involved, and during the progress of the disease the small tubes become more or less blocked with secretion; this has a valve-like action, which prevents air from entering some of the alveoli during inspiration, but allows it to escape in expiration, so that these air cells collapse, and as a result the cells in adjoining lobules are correspondingly distended. The lung consequently has an irregular mottled appearance, from interspersed sunken atelectatic patches and elevated distended air sacs.

ETIOLOGY.—The etiology of capillary bronchitis is that of acute bronchitis, it usually resulting, in children and the aged, from extension of inflammation from the larger tubes.

SYMPTOMATOLOGY.—The principal symptoms, in addition to those found in acute bronchitis, are severe dyspnœa with lividity of the surface and great prostration, following marked febrile reaction and accompanied by rapid respiration and a weak pulse.

The principal *signs* are: absence of dulness, occasionally exaggerated resonance and sibilant or subcrepitant râles on both sides (Fig. 17).

By inspection, respiratory movements are found to be rapid, and the countenance shows the effects of imperfect aeration of the blood as the disease advances.

Palpation occasionally yields a rhonchial fremitus, due to disease in the larger bronchial tubes.

Percussion obtains a resonance normal or slightly exaggerated over the lower portions of the chest. This exaggeration is due to emphysema of a portion of the air vesicles, which results from complete occlusion of some of the smaller tubes, with collapse of their terminal vesicles, and consequent dilatation of the surrounding air cells.

Auscultation usually furnishes signs of general bronchitis, and in addition to these, early in the course of the affection, sibilant râles are found in great abundance, which a little later are replaced by subcrepitant râles. These subcrepitant râles, when numerous and attended by the symptoms already mentioned, may be taken as a positive sign of capillary bronchitis, but a few are frequently heard over the lower portion of the chest, simply from gravitation of fluids, or of the products of inflammation from the larger bronchial tubes.

Occasionally a few subcrepitant râles are heard, near the borders of the lung, even in health.

Subcrepitant râles, when confined to the apex or to the base of one lung, usually indicate that the capillary bronchitis producing them is either of tuberculous or of emphysematous origin.

DIAGNOSIS.—Capillary bronchitis is attended by signs similar to

some of those found in asthma, pneumonia, or pulmonary œdema. This disease may be distinguished from *asthma* by the history.

Capillary bronchitis cannot be mistaken for the first or second stage of *lobar pneumonia* if we bear in mind that neither of these stages causes many sibilant or subcrepitant râles, which are abundant in bronchitis, and that both stages are attended by marked dulness, while in bronchitis resonance is either unaltered or exaggerated. From the third stage of lobar pneumonia this disease is distinguished by the signs obtained by palpation, percussion, and auscultation, as follows:

CAPILLARY BRONCHITIS.	LOBAR PNEUMONIA.
Palpation.	
No increase in the vocal fremitus.	Vocal fremitus increased.
Percussion.	
No dulness; occasionally exaggerated resonance.	More or less dulness.
Auscultation.	
Subcrepitant râles over both lungs; these râles are of low pitch.	Subcrepitant râles confined to one side, over the affected lung; these râles are high in pitch.

It is difficult to distinguish between capillary bronchitis and *lobular pneumonia*, with which it often coexists; but the diagnosis may be made fairly certain by attention to the following points:

CAPILLARY BRONCHITIS.	LOBULAR PNEUMONIA.
Symptoms.	
Moderate fever. Moderately accelerated respiration.	High fever. Very rapid respiration.
Percussion.	
No dulness, but possibly exaggerated resonance.	Limited unchanging spots of dulness may sometimes be detected, though, as the disease usually occurs in children, in whom dulness is difficult to detect, this sign is liable to escape observation.
Auscultation.	
Multitudes of fine dry or moist râles over every part of the chest.	The râles are limited in area unless the two diseases coexist. Bronchial breathing can occasionally be detected.

Capillary bronchitis is distinguished from *pulmonary œdema* by the following symptoms and signs:

CAPILLARY BRONCHITIS.	PULMONARY ŒDEMA.
History.	
Febrile symptoms. Usually shows an antecedent acute bronchitis several days in duration.	No febrile symptoms. This affection usually follows some protracted disease, as typhoid fever, or affections of the heart or kidneys.

CAPILLARY BRONCHITIS.	PULMONARY ŒDEMA.
Percussion.	
Resonance normal or exaggerated.	Dulness over the lower part of both lungs.
Auscultation.	
Usually numerous râles in the larger tubes.	Signs of general bronchitis frequently absent.

Capillary bronchitis is distinguished from *phthisis* by the history of the case, and by the fact that the subcrepitant râles of the latter affection are limited to a smaller portion of the chest, which is usually over the apex of one lung.

PROGNOSIS.—This disease in severe cases may prove fatal within eighteen hours, but usually it extends over four or five days. The rate of mortality, though differently estimated, is extremely high, especially for the aged and for infants under one year. When following whooping-cough or measles, or complicating any serious organic trouble, or occurring in delicate children, the prognosis is also unfavorable. Convalescence in any event is apt to be tedious and recovery incomplete, attended by more or less permanent crippling of one or both lungs by collapse of the alveoli and hyperplasia of the connective tissue. The prognosis should therefore always be guarded.

Death generally results from asphyxia, and its approach is indicated by signs of extensive involvement of the lungs, difficult expectoration, cessation of cough, dyspnœa, cyanosis, or the symptoms of collapse. A temperature of 105° F. or more, if long continued, is very unfavorable.

TREATMENT.—Opiates should *not* be used in this disease excepting in very small doses. Early in the disease, ammonium chloride with syrup of ipecac will be useful; but after two or three days, more benefit will be derived from ammonium carbonate. Inhalations of steam, or steam impregnated with sedative remedies, have a soothing effect on the inflamed bronchi (Form. 53–59). Ammonium iodide in small and often repeated doses is sometimes a most efficient remedy. Strychnine, gr. $\frac{1}{30}$ to $\frac{1}{20}$, is a valuable remedy in this affection, as soon as symptoms of exhaustion supervene. Alcoholics should be used to sustain the strength, if the ammonium carbonate does not seem sufficient. Cough and any spasmodic tendency may be relieved by camphor or the bromides.

In children it is necessary to watch carefully the secretion of urine in order to avoid a frequent cause of dyspnœa; digitalis internally and cataplasms over the kidneys are usually effective in promoting free renal secretion (Simon: *Medical News*, January, 1890).

The most efficient remedies are ammonium carbonate and strychnine, with large jacket poultices kept constantly warm and moist and covering the whole chest. The diet must be nourishing.

PLASTIC BRONCHITIS.

Synonyms.—Pseudo-membranous, croupous, exudative, or fibrinous bronchitis.

Bronchitis is sometimes complicated by exudation of fibrinous matter, with the formation of false membrane or plastic casts in the smaller air tubes and their ramifications and occasionally in the larger bronchi. This affection may be acute or chronic.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—The affection is generally chronic, and usually involves the smaller bronchi only. It is most frequently circumscribed, but may be diffuse in acute cases, and is marked by exudation from the surface of the bronchial mucous membrane of fibrinous material, forming casts, which have a laminated structure, the layers being separable when dry. This substance is composed of coagulated albumin (soluble in alkali), containing leucocytes and fat globules, sometimes octahedral crystals, a few red corpuscles, and epithelial cells. It is firm and of a white, gray, or yellow color, occasionally specked with blood. Seemingly the mucous membrane beneath it is not seriously implicated, but may be either congested or pale.

ETIOLOGY.—The ultimate cause of plastic bronchitis is not as yet known. Though poverty, exposure, and feeble health are mentioned as favoring its occurrence, excepting diphtheria, no particular diseases or conditions have been ascertained to bear special causal relation to it.

Authorities differ as to its comparative frequency relative to age and sex. According to Peacock it more often affects men (Transactions of the Pathological Society, Vol. V, London).

SYMPTOMATOLOGY.—The prominent symptoms are: hacking cough with scanty expectoration, followed, after a varying interval of from a few hours to several days, by a sense of constriction in the chest, and dyspnœa which may be very severe. The cough gradually increases in severity, the expectoration becomes more abundant and perhaps tinged with blood or accompanied with profuse hæmoptysis, and finally small fragments of the fibrinous matter are brought up or, after severe paroxysms of cough, complete casts of the bronchi. These casts may be solid or hollow, varying in diameter up to half an inch and in length from a fraction of an inch to six inches, the counterpart of the branching bronchial tree.

The physical *signs* are those of ordinary bronchitis, superadded to which are the signs due to partial or complete obstruction of some of the bronchial tubes, viz., weakness or absence of the respiratory murmur, with dulness where portions of the lung are collapsed. These signs may lead to an erroneous diagnosis of *pleurisy* or of *pneumonia.* From the former, plastic bronchitis is distinguished by absence of catching respiration, pains, and friction sounds; by the speedy occurrence of

dulness with loss of the respiratory murmur and vocal signs, and by the presence of signs of bronchitis in other parts of the chest.

We distinguish it from *pneumonia* by the absence of bronchial breathing, and, when collapse of the lung occurs, by the sudden accession of the signs of consolidation. The differentiation from ordinary bronchitis rests entirely upon the expectoration of fibrinous casts.

Prognosis.—The mortality in the acute form is about fifty per cent, death occurring in from five to fifteen days. Though complete recovery from chronic plastic bronchitis is rare, death simply from this form is equally so.

Treatment.—During the acute attack or during exacerbations of the chronic form of plastic bronchitis, the treatment should be essentially the same as that for membranous croup.

Stirling recommends inhalations of lime water, strong or dilute or combined with a two to five per cent of sodium bicarbonate, in which the casts are soluble. Turpentine, cubebs, and copaiba tend to render them more plastic.

At other times, potassium iodide will afford some relief. The general health must be maintained and all causes of cold avoided.

A warm climate is advisable, and if possible a sea voyage.

DILATATION OF THE BRONCHIAL TUBES.

Synonyms.—Bronchiectasis or bronchicatasis, knife-grinder's rot, filer's phthisis, cirrhosis of the lungs. It is sometimes termed fibroid phthisis.

Anatomical and Pathological Characteristics.—Dilatation of the bronchi is usually associated with fibrous induration of the lungs or with vesicular emphysema. It is generally found in the smaller tubes over the middle or the lower portion of the lung, more frequently on the right than on the left side.

The affection may be general or partial, single or multiple, and may be fusiform, cylindrical, or saccular. The bronchus so affected may continue of normal calibre on each side of the enlargement; it may be narrowed or obstructed on either the distal or the proximal side; or obliterated on both. The walls of such a cavity frequently show atrophy of the mucous membrane, with its secreting glands, or they may present a surface more or less irregular and granular. The submucous elastic tissue is hypertrophied, the muscular coat normal, atrophied, or its fibres widely separated. The cartilages may be thickened or may have partially disappeared, but the connective-tissue elements are greatly hypertrophied, and the adjacent interstitial lung tissue is involved in the same process.

Etiology.—Bronchiectasis may arise from increased pressure within the bronchi or from weakening changes in the walls or surrounding lung

tissue. It may be the result of alveolar collapse or atelectasis or stenosis of the bronchi from any cause, but chiefly from chronic bronchitis, also from phthisis and occasionally from old pleuritic adhesions.

SYMPTOMATOLOGY.—Patients affected with bronchiectasis often have the general appearance and symptoms of phthisical subjects. The principal distinctive symptom is the expectoration of opaque, purulent, and extremely offensive sputum, which is very abundant, measuring sometimes three pints in twenty-four hours.

The principal *signs* are: more or less dulness, and a harsh inspiratory murmur with numerous râles, all of which signs may rapidly change.

Inspection shows imperfect expansion of the chest, prolonged, labored expiration, with more or less fixity of the chest walls, and depression of the intercostal spaces.

The signs obtained by palpation, percussion, and auscultation vary greatly at different times, according to the amount of fluid in the tubes or cavities. This variation in the signs is of itself almost diagnostic of the disease.

By palpation, the rhonchial fremitus may or may not be obtained. The vocal fremitus may be normal, but it is sometimes increased, at other times diminished.

By percussion, some dulness is usually obtained over the affected lung. This is sometimes removed by free expectoration, and may then be followed by vesiculo-tympanitic or perhaps a cracked-pot resonance. Dulness is apt to be located at the middle or lower part of the lung, and is most common on the right side. Light percussion usually elicits dulness, when a more forcible stroke would produce a somewhat tympanitic sound.

On auscultation, we sometimes find the respiratory murmur suppressed over a considerable portion of the lung, while round about it the sounds may be harsh and loud. A little later, free expectoration having emptied the bronchial tubes and cavities communicating with them, respiration may become broncho-vesicular and intense, where at first it could not be heard. The respiratory murmur is often associated with numerous adventitious sounds of every variety from the dry, sibilant râle to gurgles.

Vocal resonance is subject to similar changes, and from the same causes.

DIAGNOSIS.—Bronchiectasis is most likely to be mistaken for *phthisis*, from which it can only be distinguished by attention to the expectoration, and to the mutability of the physical signs. The distinctive features between the two are as follows:

BRONCHIECTASIS.	PHTHISIS.
Palpation.	
Fremitus changeable.	Exaggerated vocal fremitus not universal, but when present usually constant.

Bronchiectasis.	Phthisis.
Percussion.	
Dulness, or vesiculo-tympanitic resonance, often changing from one to the other during the examination.	More or less dulness, which remains constant.
Auscultation.	
The signs are usually found over the lower or middle portions of one or both lungs, and change rapidly as the result of deep inspiration or cough.	The signs for several months are usually confined to the upper portion of one lung. They are not materially altered by cough or by deep inspiration. They are confined to a more limited space than the signs of dilatation of the bronchi.

Prognosis.—Bronchiectasis runs a chronic course, and, though not fatal in itself, is inductive of other pulmonary disease, especially predisposing to putrid bronchitis, and gangrene or abscess of the lung. It is incurable and, being secondary to chronic bronchitis, old pleuritic adhesions and thickening, atelectasis or fibroid phthisis, its prognosis depends upon that of the associated disease.

Hectic, rapid pulse and progressive emaciation with night sweats are unfavorable symptoms, but these symptoms, attended by most abundant fetid expectoration and great asthenia, giving the appearance of the last stage of consumption, sometimes disappear in a partial recovery, so that the patient lives in fairly good health for a year or two.

Treatment.—In bronchiectasis, cod-liver oil, calcium chloride, and vegetable tonics are generally demanded. Some of the preparations of eucalyptus globulus or grindelia robusta are occasionally beneficial, as are also copaiba, turpentine, senega, and squills. Potassium or ammonium iodide and arsenic are also useful. Inhalations of turpentine, camphor, iodine, and carbolic acid are frequently useful in checking or altering the secretions (Form. 66, 67, 68, 70, 71, 73). Counter-irritation should be tried.

ASTHMA.

Asthma is a spasmodic affection of the respiratory apparatus, chiefly characterized by paroxysmal attacks of dyspnœa.

Anatomical and Pathological Characteristics.—There are no recognized morbid changes peculiar to asthma. It is a functional disorder or neurosis dependent upon some physical condition not yet thoroughly understood. Many hypotheses have been advanced to explain the mechanism and cause of asthmatic dyspnœa.

Though none of them have become entirely adequate theories, the bronchial spasm hypothesis is the one most commonly accepted. According to this, the dyspnœa is due to spasm of the annular muscular fibres of the bronchi which narrows their calibre and obstructs the pas-

sage of air. That bronchial constriction occurs in asthma is proved by the constant presence of sibilant râles.

Some, with Wintrich, consider spasm of the diaphragm as accountable for the difficult breathing.

Weber and others hold that it is due to vasomotor relaxation producing congestion and tumefaction of the bronchial mucous membrane. Crystals and spirals found in the sputum by Leyden and Curschmann, and supposed to be causative, as irritants to the bronchial mucous membrane, have been ascertained to be present not alone in asthma, but also in many pulmonary disorders.

ETIOLOGY.—Although the ultimate cause of asthma is unknown, certain predisposing conditions are recognized; according to Salter, heredity is to be traced in forty per cent of all cases; others claim a smaller percentage (Lazarus in *Deutsche medicinische Zeitung*, 1887).

The neurotic temperament seems to favor it, particularly if coupled with plethora; also the rheumatic and gouty diathesis. It is common to all ages. Its victims are most often males, those preferably of the upper class. Soltmann thinks it especially common among the Hebrews (Shattuck: Cyclopedia of Diseases of Children, Keating). Asthmatics usually suffer most in winter, and the attacks occur generally at night. Its exciting causes may be considered as those acting directly as irritants to the terminal fibres of the vagus or sympathetic in the bronchial mucous membrane, and those acting reflexly from a greater or less distance. Bronchitis is the most frequent exciting cause of asthma. An asthmatic attack may arise from inhalation of dust, smoke, fog, and other vapors, pungent fumes, odors from certain plants, pollen, and emanations from animals. Indeed, the list of substances capable of exciting an asthmatic paroxysm is long.

Different patients are affected each in his own peculiar way, one by the presence in the atmosphere of one substance or condition, another by one totally different. The diseases and conditions which by reflex impression upon the bronchial nervous mechanism excite the asthmatic paroxysm are also very numerous and varied. Not infrequent causes are found in irritation of the upper air passages by impalpable particles diffused in the atmosphere or by such deformities as septal deflection, exostoses, nasal polypi, and hypertrophy of the tonsils.

Asthma has been attributed to the pressure from a hypertrophied thyroid, an aneurism or other tumors, or from enlarged bronchial glands. It is frequently due to some disorder of the alimentary tract, such as gastric indigestion or neurosis, duodenal catarrh, hepatic torpor, constipation, intestinal worms, or hemorrhoids. It may be due to abdominal tumors or derangements of the genito-urinary system, as for example calculi, prostatic enlargement, enuresis, spermatorrhœa, sexual abuse, and, in women, ovarian, uterine, and vaginal disease. Diseases of the heart, of

the kidney, or of the brain may cause asthma, as may also certain skin diseases—eczema, urticaria, and herpes, for example. Poulet describes an epileptiform variety of asthma (*Journal de Médecine de Paris*, 1889). It seems sometimes to occur from presence in the blood of poison, such as the uræmic, gouty, rheumatic, or malarial (Robinson, *Medical News*, 1890), or certain chemicals presumably acting through the circulation upon the respiratory centres. But back of all these favoring conditions and exciting causes is something, as yet unknown, which is an important if not the chief etiological factor in the production of the disease. Cases occur in which the most careful examination fails to find any predisposing or exciting cause.

Symptomatology.—Asthma is characterized chiefly by paroxysms of dyspnœa, with stridulous respiration and the evidences of deficient aeration of the blood. In some instances an attack may be foretold by sensations of mental depression, drowsiness, or irritability, or their opposites; or by hyperæsthesia, headache, a sense of constriction of the throat or chest or frequent desire to gape or sneeze. Some attacks begin with coryza, which may develop into bronchitis. Usually the onset is sudden; the patient awakes from sleep, wheezing and perhaps gasping for breath, with a sense of thoracic constriction, and if it be his first attack he fears imminent suffocation. Breathing becomes more labored, accompanied by venous turgescence, congestion of the face and neck, bulging and suffusion of the eyes, dilatation of the nostrils, and profuse perspiration. The pulse decreases in strength with the severity and duration of the paroxysm. The paroxysms usually last from two to four hours, but the attack sometimes terminates in a few minutes. It may occasionally continue for weeks. Recurrence of the affection results in some patients only from certain exciting causes, in others more or less periodically—daily, weekly, monthly, or yearly.

Diurnal attacks are rare. Frequently the paroxysm terminates in a mild bronchitis. Between attacks the condition of asthmatic patients varies in degree from a condition of apparent health to the state of more or less constant suffering from the disease or its sequelæ.

The principal *signs* are labored and wheezing respiration, attended by numerous sonorous and sibilant râles, which may be heard, and often felt, over the whole chest.

The patient is usually found in the upright position. Respiration is labored, inspiration being short and jerking, and expiration prolonged. The dyspnœa is chiefly expiratory. The respiratory motion of the chest is greatly diminished. Severe cases show the signs of deficient oxygenation of the blood.

Inspection, palpation, mensuration, and percussion yield no distinctive signs. The resonance may be normal or slightly exaggerated.

By auscultation we obtain jerking or cog-wheel respiration, with a great variety of sonorous and sibilant râles. The respiratory murmur

is usually harsh and more or less tubular, the vesicular element being suppressed. Vocal resonance is normal.

DIAGNOSIS.—During a paroxysm, asthma may be mistaken for cardiac dyspnœa, capillary bronchitis, or spasmodic laryngeal affections. From the first, it may be distinguished by the history, by the absence of cardiac signs and by the presence of a great number of sonorous and sibilant râles.

Asthma differs from *capillary bronchitis* in its history, and in some of the signs obtained by inspection and auscultation, as shown in the following table:

ASTHMA.	CAPILLARY BRONCHITIS.
Symptoms.	
A sudden attack, with usually a history of former paroxysms. Febrile symptoms not marked.	Dyspnœa comes on gradually, usually preceded by acute or subacute bronchitis. Febrile symptoms pronounced.
Inspection.	
Respiration labored, but not greatly accelerated.	Respiration not only labored, but also rapid.
Auscultation.	
Sonorous and sibilant râles, usually followed by large and small mucous râles.	Mucous râles likely to precede the sibilant râles, and the sibilant to be followed by the subcrepitant.

Spasmodic affections of the larynx are distinguished as follows:

ASTHMA.	SPASMODIC LARYNGEAL AFFECTIONS.
Dyspnœa, expiratory.	Dyspnœa inspiratory.
Râles.	No râles.
No local laryngeal signs.	Laryngeal signs sometimes positive.
No change in voice.	Voice altered.

After the paroxysm, the signs of asthma are like those of bronchitis, but they last only a few hours.

Asthmatic symptoms often occur during the progress of pulmonary emphysema; but these two diseases may be easily distinguished from each other by the history. *In emphysema,* as in cardiac disease, dyspnœa is permanent, and aggravated by exercise; while in asthma the dyspnœa usually comes on during the hours of rest.

PROGNOSIS.—Asthmatic paroxysms are very rarely fatal. One attack predisposes to others, and the disease is usually obstinate. Hope of complete cure is good in proportion to the youth of the patient, absence of organic disease, short duration of the attacks, infrequence of recurrence, immunity from distress during the intervals, and the presence and discovery of a removable cause. Chronic asthma tends to the development of emphysema, chronic bronchitis, and dilatation and hypertrophy of the right cardiac ventricle.

TREATMENT.—During the paroxysm, the most effectual internal treatment consists of the administration of morphine and chloral (Form. 2) repeated every half-hour or every hour until relief is obtained. This may be combined with half a drachm of fl. ext. of grindelia robusta, which is sometimes beneficial. The nitrites in the form of nitroglycerin gr. $\frac{1}{100}$, or nitrite of amyl ♏ij. to v., repeated every twenty minutes for two or three doses, or apomorphine gr. $\frac{1}{10}$ internally every two hours, frequently prove effective. Weill (*La France Médicale*, March, 1889) through experiments, confirmed by others, found that inhalation of carbon dioxide greatly relieved cough and dyspnœa and cut the paroxysm short.

Two or three cups of strong hot coffee will frequently abort an attack, if taken when the first symptoms are noticed. The severity of the paroxysms may be greatly modified by small doses of belladonna, hyoscyamus, or hyoscyamine gr. $\frac{1}{140}$ to $\frac{1}{120}$ hypodermically; or by potassium bromide or camphor. Fuming inhalations of arsenious acid or potassium nitrite alone or combined with other antispasmodics such as stramonium, hyoscyamus, or tobacco, give speedy relief in some cases (Form. 132–138). Galvanizing the pneumogastric nerve, with the positive pole beneath the mastoid process, and the negative pole on the epigastrium, will promptly relieve some cases.

If either bronchitis or pneumonia supervenes, it should receive treatment similar to that recommended when it occurs as a primary disease. The general treatment of asthmatic patients should be supporting. Between the paroxysms an effort should be made to prevent their recurrence. The most efficacious remedy for this purpose is potassium iodide, but in some cases ammonium iodide, grindelia, eucalyptus, arsenious acid, or resin of guaiac will be found useful.

In all cases a complete history should be obtained and a thorough examination made to ascertain, if possible, the existence of any disorder which might cause a reflex bronchial spasm. Such disorder should be corrected; thus, it will often be possible to prevent or cure an attack by attention to the alimentary canal.

It should be remembered that asthma may result from the rheumatic or dartrous diathesis, and that it is often caused by bronchitis or emphysema, as well as by purely nervous affections. The treatment must therefore meet the conditions of each case.

If all medicines fail, a change of climate should be tried. The climate of Colorado is perhaps the most frequently beneficial to these patients, but very slight changes may be sufficient to prevent a recurrence of the attacks; therefore "each patient must be a law unto himself" in this regard. By repeated trials, most cases will find localities where they will be free from asthmatic attacks.

PULMONARY EMPHYSEMA.

Pulmonary emphysema is an abnormal inflation of the lung, due to over-distention of its air vesicles or accumulation of air in the tissues about them; in the former cases it is commonly termed vesicular, in the latter extra-vesicular or interlobular emphysema.

Etiologically it is also called primary or secondary, compensatory and vicarious.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—Post-mortem opening of the chest in a well-marked case of general emphysema reveals the lungs abnormally pale, much distended so as to meet or overlap anteriorly, their surfaces bearing the imprint of the ribs, their borders rounded. They do not collapse. The heart may be displaced downward and toward the median line. The lung feels softer than normal and puffy to the touch. Indentation made by digital pressure remains for some time.

There is loss of elasticity, diminished crepitation, and greater buoyancy in water. Dilated air sacs may be seen protruding from the surface as rounded, hemispherical, or spherical elevations and of a grayish hue. Air may be pressed from the distended sacs, which upon section appear as cavities scattered through the lung, varying in size from a millet-seed to a hen's egg. In mild or beginning emphysema there may be simply extreme distention of the alveoli, with little or no destruction of their walls. As the process continues, two or more air cells coalesce, by the rupture of their common septa, forming cavities of variable size. The walls of these are here and there constricted and roughened by ragged projections which mark the location of former alveolar partitions. The capillary plexus is consequently partially destroyed. In the interlobular form, secondary to vesicular emphysema, air escapes from the vesicles into the interstitial connective tissue forming other cavities. The process may extend along the blood-vessels of the interlobular septa to invade the mediastinal, cervical, and finally the subcutaneous connective tissue.

Probably rupture of the alveolar walls is dependent in most cases upon a primary fatty or fibroid degeneration. Senile emphysema, so called, results from atrophy of lung tissue; here the lungs are diminished in size and generally pigmented. Emphysema is generally bilateral, but may be confined to one lung or to a single lobe. When due to forced expiration, with obstruction in the trachea, larynx, or glottis, it is most marked along the anterior border of the upper lobes. In addition to these morbid changes, the bronchi communicating with the cavities are the seat of more or less bronchitis and bronchiectasis. Virchow, as reported in 1889, had never seen tubercles in an emphysematous lung and only one case of pneumothorax (*Berliner klinische Wochenschrift*, 1889).

But both these conditions may accompany it. Pneumonia occasionally complicates it, and dilatation and hypertrophy of the heart, with resulting changes in the lungs, liver or kidneys, are not uncommon.

Etiology.—Emphysema may occur at any age. It is, however, most common in those beyond middle life, and more frequent in men than in women. Heredity seems to play an important part in the etiology; but whether the disease is largely due to hereditary transmission of a special weakness of lung tissue, or to primary malnutritive changes of a fatty or fibroid nature, is an open question. It occurs in the aged from natural atrophy accompanying general senile decline. Forced inspiration may cause over-distention or rupture of air vesicles, whose elasticity is already impaired. The usual cause is the exertion, after deep inspiration, of powerful expiratory efforts with closed glottis or with more or less obstruction of the respiratory passages from other causes. Hence, the disease not infrequently complicates asthma and the cough of chronic bronchitis or pertussis, and may result from excessive use of certain wind instruments, or from straining efforts as in lifting, childbearing, or defecation. Local compensatory emphysema occurs in the air vesicles adjacent to lung tissue that is collapsed or consolidated or whose larger bronchi have been obstructed. Obliteration of the air vesicles of one lung wholly or in large part, from pneumonia, phthisis, infarction, and the like, or from pressure by pleuritic effusion, may produce compensatory emphysema in the opposite organ.

Symptomatology.—The prominent symptoms are constant dyspnœa, increased on exertion, associated often with the symptoms of bronchitis or asthma, or of both.

The prominent *signs* are: lifting of the sternum in inspiration, barrel-shaped chest; vesiculo-tympanitic resonance, and prolonged expiration.

Inspection in well-marked cases finds the countenance dusky, the eyes prominent, the nostrils dilated, and the sterno-cleido-mastoid muscles standing out like whip-cords in their efforts to aid in respiration. The shoulders are elevated and drawn forward, the neck is apparently shortened, and the individual seems to stoop, which gives him the appearance of old age. The margins of the scapulæ sometimes stand out like wings, and there is an increase in the antero-posterior diameter of the chest, giving the rounded barrel-shaped appearance. During inspiration, there is no expansive movement of the upper ribs, but they are elevated as if the chest walls were composed of a single bone. In marked cases of this disease, there is with inspiration falling in of the soft parts of the chest above the clavicles and sternum; the intercostal spaces at the upper part of the chest are wider and more distinct than usual; and there is retraction instead of expansion of the false ribs during inspiration. Early in the disease, these signs are not present. Venous pulsation is sometimes seen in the jugulars.

Occasionally among old people, in cases known as atrophous emphysema, the intervesicular septa are destroyed by atrophy and the vesicles coalesce. The volume of the lung is thereby more or less diminished, so that the disease causes no distention of the chest. In such cases, no signs would be obtained on inspection, except perhaps retraction and an increased obliquity of the lower ribs, with considerable diminution of the space between them and the crest of the ilium.

By palpation, the apex beat of the heart is frequently found below its normal position, and nearer the median line.

Vocal fremitus may be exaggerated, diminished, or normal.

Mensuration shows us the exact increase in the antero-posterior diameter of the chest, and the deficient expansive movement in inspiration.

Percussion yields vesiculo-tympanitic resonance, usually most marked over the upper part of the left lung. Percussion over the præcordia may show diminished area of superficial cardiac dulness, or the entire region may yield pulmonary resonance, due to the expansion of the border of the left lung, so that it completely covers the heart.

Deep inspiration or forced expiration will not materially affect the pulmonary resonance, as it would in health.

On auscultation, the vesicular murmur is impaired, the inspiratory sound being deferred, and consequently shortened, and the expiratory sound being prolonged, so that the ratio between the two may be reversed, making the expiratory sound equal in length to the inspiratory, or even three or four times as long. In typical, uncomplicated cases, both sounds are low in pitch; but harsh, blowing sounds from the bronchial tubes are often heard, especially during inspiration. A peculiar dry, crackling sound, closely resembling fine pleuritic friction, is often heard just at the end of inspiration or at the beginning of expiration. It is produced in the walls of the air-vesicles.

Gerhardt (*Berliner klinische Wochenschrift*, 1888), in four cases of emphysema, heard fine bubbling, crackling sounds in the cardiac region synchronous with the heart-beat, evidently from displacement of air in the mediastinal connective tissue by the cardiac impulse.

In rare cases, especially in the aged, the inspiratory and the expiratory sounds are of equal duration, exaggerated in intensity, harsh and tubular in quality, and high in pitch. This is probably due to atrophy of a portion of the lung tissue.

Vocal resonance may be either increased or diminished.

The heart-sounds are usually feeble, and those at the apex are displaced downward and inward, by the intervention of the emphysematous lung between this organ and the surface of the chest. The cardiac sounds and impulse are often abnormally distinct in the epigastric region, due to displacement of the heart and to dilatation of the right ventricle. Dilatation of the ventricle may cause tricuspid regurgitation with a valvular murmur.

Diagnosis.—The diseases likely to be mistaken for emphysema are: dilatation of the lung from acute tuberculosis, and pneumothorax. When confined to one lung, emphysema may be mistaken for any of the diseases which usually cause feeble respiration. In such cases, the normal murmur of the sound side is liable to be mistaken for exaggerated respiration, and the feeble murmur of the emphysematous lung for the normal sounds. Error may be avoided by remembering that the feeble respiratory murmur of emphysema is characterized by *prolonged* expiration, and that resonance over the affected lung is more marked than that of the sound side; while in nearly all diseases causing feeble respiration, from obstruction in the air passages or from interference with the free expansion of the lung, the expiratory sound is *shorter* than the inspiratory, and the resonance is less intense than on the sound side. Emphysema of one lung, or of a single lobe of one lung, is a rare affection; but when it does occur, great care is necessary to avoid errors in diagnosis.

Bilateral emphysema is differentiated from *pneumothorax* by the signs furnished upon inspection, percussion, and auscultation, as follows:

Emphysema.	Pneumothorax.
Inspection.	
Usually bilateral.	Very rarely bilateral.
Prominence of both sides, especially of the antero-superior portion of the chest, with falling in of the soft parts during inspiration.	Uniform distention of one side, no sinking in of the soft parts during inspiration.
Percussion.	
Vesiculo-tympanitic resonance on both sides.	Tympanitic resonance on one side only.
Auscultation.	
The respiratory murmur vesicular in quality, and expiration prolonged.	The respiratory murmur feeble or suppressed, or amphoric.

Emphysema of a single lung is distinguished from pneumothorax by the following signs:

Emphysema of one lung.	Pneumothorax.
Percussion.	
Vesiculo-tympanitic resonance.	Tympanitic resonance more or less intense, with absence of the vesicular quality.
Auscultation.	
The inspiratory murmur delayed, the expiratory sound prolonged.	The vesicular murmur feeble or absent, but, if heard, regular in rhythm. The respiration may be amphoric.

R. Thompson states that in *acute tuberculosis*, as numbers of the air

vesicles become filled with the tubercular deposit, the adjoining cells become distended so as to cause physical signs, especially in front, almost identical with those of emphysema. The distinctive features of the two diseases may be seen in the following table:

EMPHYSEMA.	ACUTE TUBERCULOSIS.
History.	
Affection gradually developed.	Comparatively rapid accession.
Symptoms.	
Constitutional symptoms often slight.	Constitutional symptoms similar to those of typhoid fever.
Inspection.	
Cyanosis; labored expiration; chest enlarged.	Pallor; respirations rapid but not labored; chest not enlarged.
Percussion.	
Vesiculo-tympanitic resonance more or less marked over whole chest.	Vesiculo-tympanitic resonance in front, but actual dulness behind.
Auscultation.	
Expiratory murmur prolonged and low in pitch.	Expiratory murmur not much prolonged and higher in pitch than normal.

Some signs produced by *fibrosis* or *fibroid disease* of both lungs are liable to cause it to be mistaken for emphysema. The distinction may be readily made from the following signs:

EMPHYSEMA.	FIBROID DISEASE OF BOTH LUNGS.
Inspection.	
Fixity of the chest with bulging, except in the atrophous form.	Fixity of the chest with flattening.
Palpation.	
Vocal fremitus usually diminished.	Vocal fremitus markedly increased.
Percussion.	
Vesiculo-tympanitic resonance.	Usually dulness, but occasionally resonance approaching tympanitic in quality.
Heart covered by lung tissue, as shown by resonance.	Heart uncovered, causing increased area of superficial dulness.
Auscultation.	
Low-pitched respiratory sounds, though sometimes considerable harshness from affection of the bronchi.	Absence of respiratory murmur at times. In other instances, rude respiration.

Emphysema and *bronchial asthma* are not likely to be mistaken for each other, especially if the following points are remembered:

EMPHYSEMA.	ASTHMA.
History.	
Dyspnœa constant.	Dyspnœa paroxysmal.

Emphysema.	Asthma.
Inspection.	
Chest barrel-shaped.	Chest normal.
Heart displaced.	Heart not displaced.
Auscultation.	
Few râles present unless bronchitis complicate, when râles are moist.	Abundant dry râles, sibilant and sonorous.

Prognosis.—A lung once emphysematous never recovers. Mild cases dependent upon causes which may be early removed may be greatly relieved by the general improvement of the patient and the compensation offered by the remaining normal lung tissue. Though in itself not a dangerous disease, well-marked emphysema insures the patient much distress, unfits him for active life, and greatly predisposes him to more serious disease. Bronchitis, though frequently a cause of the disease, is a common effect. Bronchiectasis, asthma, and pleurisy are likewise frequent complications.

Heart disease with disorders of the liver, kidneys, spleen, and alimentary tract which are its common sequelæ, naturally results from chronic obstruction to pulmonary circulation, and is therefore an important element in prognosis. Pneumonia, tuberculosis, and hemorrhage are rarely observed in emphysematous foci, but may occur in parts not so affected.

Treatment.—As the changes in the lung tissue in this disease are due in part to general malnutrition, our first aim in treatment must be to improve the general condition. Remedies of most service for this purpose are tincture of iron, cod-liver oil, and occasionally small doses of quinine and strychnine.

Chronic bronchitis usually coexists, and should receive treatment similar to that already mentioned under the head of treatment of pneumothorax and pneumo-hydrothorax. Potassium iodide is the most serviceable single remedy in this disease. It should be given in doses of gr. v. to xx., three or four times a day for a long time. Arsenious acid long continued has been found beneficial. Asthmatic symptoms are to be treated as spasmodic asthma. Cough may require anodynes. Expiration into rarefied air has benefited some cases.

The patient must avoid all causes of cold or asthmatic attacks, and should live if possible in a climate where he will be most free from dyspnœa. High altitudes are not to be recommended for these cases.

CHAPTER VIII.

PULMONARY DISEASES.—*Continued.*

PNEUMONIA.

Synonyms.—Peripneumonia, peripneumonia vera. Popularly known as lung fever or inflammation of the lungs. There are two recognized varieties of this disease: *lobar pneumonia*, in which the greater part or the whole of one lobe, or the whole lung, is affected, and *lobular pneumonia*, in which the inflammation is confined to a single lobule, or to groups of lobules scattered through the lungs. According to the origin and character of the disease, its various manifestations collectively have also been termed primary or secondary pneumonia, or bilious, gastric, typhoid, latent or walking, intermittent, hypostatic, tubercular, scrofulous, rheumatic, gouty, puerperal, or metastatic pneumonia—varieties, so called, which require no special description. Though different cases vary more or less in their origin and anatomical characteristics, as well as in a few of their clinical features, to attempt to differentiate between them by their physical signs would only be confusing. I shall therefore consider at length only lobar and lobular pneumonia, and but briefly mention, under their respective headings, special variations of the disease, and the signs which are accounted valuable in enabling us to differentiate them.

LOBAR PNEUMONIA.

Synonyms.—Acute pneumonia; croupous pneumonia; acute sthenic pneumonia.

Lobar pneumonia consists of an inflammation of the vesicular structure of the lungs, with accumulation of inflammatory exudation in the air cells, whereby they are filled and rendered impervious to air.

Anatomical and Pathological Characteristics. — Croupous inflammation of the lung is characterized by three stages—first *engorgement*, second *red hepatization*, third yellow or *gray hepatization;* it may terminate in resolution, in suppuration diffuse or circumscribed, in gangrene, in chronic pneumonia, or in tuberculosis. During *engorgement* the lung is increased in size, is of a dark red or bluish color, with perhaps faint patches of subpleural ecchymoses and the affected tissue does not collapse. It is doughy in consistency, pits on pressure, and is heavier than normal. From the cut surface oozes a reddish sero-albuminous

fluid, with darker blood from the capillaries. *Microscopically* the vessels lining the alveoli are found crowded with blood corpuscles and so distended as to encroach upon the lumen of the air sacs, which contain serum, corpuscles, and a few epithelial cells.

In the stage of *red hepatization* the organ is darkly mottled, in color resembling the liver; the serous surface may be markedly ecchymotic and the seat of fibrinous exudation. The lung is larger, heavier, and firmer than normal; it sinks in water, is friable, non-crepitant, and may show the imprint of the ribs. The cut or torn surface is bathed in a reddish serous fluid, and appears granular from the projection of small, dark red masses of coagulum from the alveoli. These become more prominent on pressure and are easily removed upon scraping the surface.

Microscopically these masses are seen to consist of granular epithelial cells and red and white corpuscles, held within a fibrinous coagulum.

In the third stage red hepatization gradually gives place to *yellow* or, in markedly pigmented lungs, to *gray hepatization.* The red color of the former stage disappears owing to fatty degeneration of the alveolar contents, to anæmia produced by the pressure within the alveoli, and to breaking up of the red corpuscles with some absorption of their hæmatin. The lung in this stage is still larger and heavier than in the preceding stage, it is more mottled with gray and yellow, more fragile, and is non-crepitant. Section reveals a surface more uniformly gray or dirty yellow and less granular, from which exudes a viscid fluid of like color.

Microscopic examination shows pus cells, fat globules, pigment, micro-organisms, and a detritus of fibrin and red corpuscles. The morbid conditions causing these appearances are located chiefly in the air sacs. In addition, the mucous membrane of the smaller bronchi is usually congested and not infrequently these are the seat of plastic, fibrinous casts sometimes extending to the larger tubes. Œdema of the parts adjacent to the inflammatory focus is usually present and may also involve the opposite lung. Acute compensatory emphysema is likewise occasionally present. The bronchial glands enlarge and sometimes suppurate.

Pleuritis occurs if the pneumonia is superficial. Pericarditis is most common in pneumonia of the left lung, evidently from direct extension, but it is not an uncommon accompaniment of right-sided pneumonia. Inflammation or at least marked congestion of more remote structures—the alimentary tract, liver, spleen, kidneys, brain, and spinal cord—are not uncommon associate morbid phenomena. Under favorable conditions, resolution occurs, incident to rapid fatty degeneration of the alveolar contents, which become more fluid and disappear partly by expectoration, partly by absorption. Gradually air re-enters the vesicles, which resume their function, congestion subsides, and pulmonary œdema slowly disappears. In unfavorable cases suppuration may supervene upon the third stage; the lung then becomes more uniformly yellow, boggy, and very fragile, and the fluid from the torn surface is decidedly

purulent. There is also more or less purulent infiltration of the perivesicular tissues. Resolution may slowly follow this diffuse suppuration, or numerous abscesses may form by rupture of the interalveolar septa and formation of limiting walls of granulation tissue. These in turn, by progressive ulceration in the line of least resistance, may terminate in perforation of the pleura or pericardium, or may empty themselves into the bronchi and close by cicatrization; or their contents remaining encapsulated may undergo caseous change and receive calcareous deposit. Diffuse or circumscribed *gangrene* occasionally occurs, invited in some cases by antecedent bronchiectasis or putrid bronchitis (Orth, Diagnosis in Pathological Anatomy, p. 145). In rare cases acute pneumonia terminates in a *chronic form*, characterized pathologically by large increase in the interstitial connective tissue which obliterates the alveoli and smaller bronchi of the affected part, making it firm, dense, and airless. Finally, the pneumonic area is liable to infection with the tubercle bacillus. In order of comparative frequency pneumonia affects the right lower, the left lower, and the right upper lobe. According to Minot, the disease in children originates oftenest in the right upper lobe, least frequently in the right lower. Double pneumonia occurs in from five to fifteen per cent of all cases, but most frequently in the aged (Loomis' Practical Medicine, p. 102; Cyclopædia of Diseases of Children, p. 589).

ETIOLOGY.—Climates and seasons most subject to sudden marked changes of temperature, occupations subjecting the individual to abrupt changes from heat to cold, and such hygienic conditions as bad ventilation and sewerage, poor food and clothing, and habits which enervate are all favorable to the occurrence of pneumonia. Though robust health and a fine physique seem at times to offer to it no barriers, yet most diseases which exhaust vitality and diminish local resistance predispose to pneumonia. In this category are included a previous attack of pneumonitis, the acute infectious diseases, alcoholism, uræmia, acute rheumatism, and disorders of the blood. Diseases of the heart producing chronic pulmonary congestion, and severe traumatic injuries to the chest, are also predisposing factors.

Recent investigations by Fraenkel, Weichselbaum, Friedländer, Netter, Sternberg and many other careful observers suggest that pneumonia is an infectious disease, the primary exciting cause of which is a specific micro-organism; and that in most instances the diplococcus pneumoniæ of Fraenkel is that germ. According to these writers, it can be proved to exist in over 90 per cent of all cases, in the tissues and fluids of the local pulmonary inflammation; and it has also been found at the seat of complicating meningitis, pleuritis, pericarditis, synovitis, and otitis. Friedländer's micrococcus, the typhoid bacillus, and other specific germs may also in some cases excite pulmonary inflammation. Delafield (*New York Med. Jour.*, 1890) regards pneumonia as an infective inflammation dependent upon individual susceptibility, a primary exciting cause of inflammation and a pathogenic bacterium some one of which factors takes precedence at different times. Facts recorded by Wolff (*Zeitschrift der Bakt.*, 1890), Jaworski

(*Jour. Am. Med. Ass.*, Dec., 1889), Kuhn (*Berlin klin. Woch.*, April, 1889), Matheson (*Brooklyn Med. Jour.*, April, 1889), Wagner (*Am. Jour. Med. Sci.*, 1889), Wells (*Med. Regist.*, Feb., 1890; *N. Y. Med. Jour.*, March, 1890), Mosler (*Deut. med. Woch.*, Nos. 13 and 14, 1890; *Med. Press and Circ.*, Sept. 25, 1890), and others strongly suggest its contagious character under some conditions.

SYMPTOMATOLOGY.—The chief symptoms are a severe initial chill, followed by fever which attains great intensity in a few hours and as suddenly subsides between the fifth and the tenth days; these are usually attended by pain in the side, dyspnœa, cough with clear tenacious and subsequently rusty sputa, great prostration, and frequently delirium.

In some cases these active features are preceded several days by dull pains in the head, back, and limbs, dizziness, lassitude, and perhaps alimentary disorders. Usually the onset manifests itself abruptly by severe rigors, which may last for two or three hours. In children there may also be initial convulsions, delirium, and gastric disturbance. The temperature in uncomplicated pneumonia is characterized generally by a rise to 103° or 105° F. at the invasion, followed by slight morning remissions and evening exacerbations till the day of crisis, when it either declines gradually or falls suddenly to normal or one or two degrees below. The highest point is commonly reached on the second or third day, but may occur just before the final fall.

The pulse ranges from 100 to 120 beats per minute, or much higher in serious cases, and is the most important index in pneumonia. It becomes rapid and feeble depending upon the severity and duration of the attack, and may be intermittent, especially in old age.

Sharp lancinating pain below the nipple, increased by cough and deep inspiration, is a common symptom, probably due to concomitant pleuritis. It may be absent or slight in old age and when the pneumonia is deep seated. It tends to diminish and disappear by the third or fourth day. Very severe headache during the first two or three days is an almost constant symptom. Delirium, usually of the mild, incoherent type, is most frequent in old people, children, and drunkards; in the latter it may take the violent form. Muscular tremors are common in convalescence. Convulsions often occur in children either at the beginning of the disease or just before death. Respiration is shallow and increased in rapidity, in severe cases even to sixty or seventy counts to the minute. Dyspnœa is usually an early and prominent symptom, but may be absent, even with greatly accelerated breathing.

Cough of a short, hacking character is commonly an early symptom, but is exceptionally absent. It may disappear just before death. The expectoration, at first frothy, becomes translucent, tenacious, and viscid, and later of a red or brownish-red brick-dust or rusty color from admixture of blood. In some grave cases the sputum is more watery and dark purple, like prune juice. Rusty sputum commonly appears within the first two or three days, but may be absent till the tenth or twelfth,

and then present in but slight degree. Rarely, it is entirely absent. During resolution the sputum is more profuse and yellow or greenish. Digestive disorders, vomiting, and diarrhœa occur sometimes at the invasion.

The essential *signs* in the order of their occurrence are: diminished movement of the side, some dulness and crepitant râles, followed by marked dulness, bronchial breathing, and bronchophony. These signs are succeeded in favorable cases by subcrepitant râles and a gradual return of the healthy signs (Fig. 27).

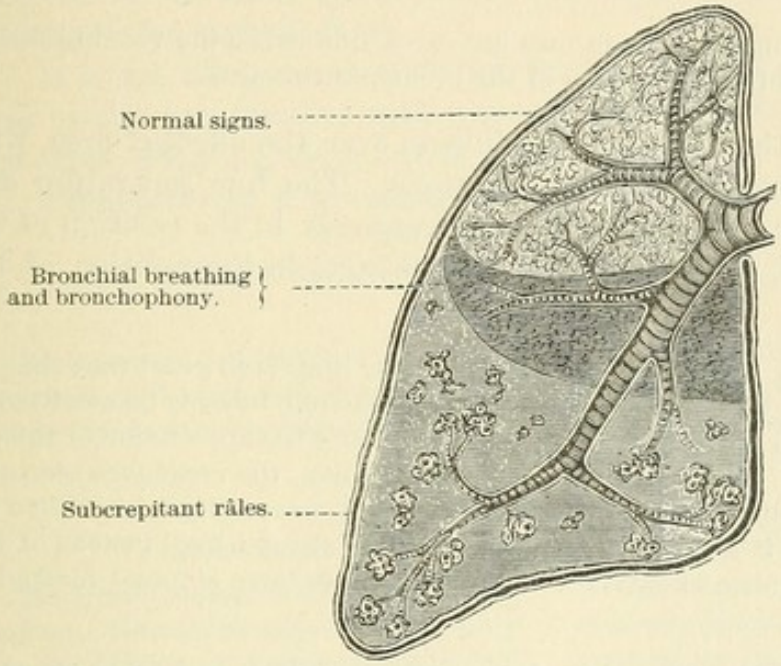

FIG. 27.—The upper lobe indicates healthy lung tissue; the middle lobe represents the second stage of pneumonia (red hepatization), and the lower lobe illustrates the third stage (gray hepatization).

For convenience we describe the signs in three groups corresponding to the three stages of the disease. The *first* stage, beginning with the inception of the disease, continues until the air vesicles are completely filled. From this point the *second* stage continues throughout the period of consolidation or red hepatization. The *third* stage, that of gray hepatization, continues from the beginning of resolution until convalescence is complete.

As *signs of the first stage*, inspection finds the movements of the chest somewhat diminished over the affected organ.

Palpation in the early part of this stage yields only negative results; later, the vocal fremitus is increased.

Percussion early in this stage elicits slight dulness, which gradually increases as the stage advances.

On auscultation, while there is congestion only, before inflammation has become fairly established, the respiratory murmur is feeble. As exudation takes place, crepitant râles occur in great numbers at the end of inspiration. When these râles are well marked and persistent, they may be regarded as pathognomonic.

When pneumonia is associated with inflammatory rheumatism, the crepitant râle does *not* occur. Subcrepitant are sometimes associated with the crepitant râles, but the latter greatly predominate.

As consolidation progresses, respiration becomes broncho-vesicular and finally bronchial.

As *signs of the second stage*, inspection and palpation show that the movements are still deficient on the affected side, and exaggerated on the opposite side. Vocal fremitus is exaggerated.

Exceptional.—Consolidation in rare instances diminishes the vocal fremitus, in consequence of complete occlusion of the bronchial tubes.

In percussion there is marked dulness over the affected area, with exaggerated resonance over healthy portions. The line separating dulness from vesicular resonance usually corresponds to the position of the interlobular fissure, and is not altered by changes in the position of the patient.

Exceptional.—In rare cases the density of the lung is so great that the percussion sound caused by vibration of air in the bronchial tubes is transmitted to the surface with such peculiar distinctness as to justify the appellation of tubular resonance. In some instances of extreme consolidation, the resonance seems almost amphoric. In such cases the solid sounds would of necessity be mistaken for hollow sounds, were it not for their pitch, which is always high instead of low like the proper resonance of cavities. In rare cases, flatness is found instead of dulness.

By auscultation there are found no crepitant râles, but in their place we find bronchial or broncho-vesicular respiration, varying in degree with the amount of consolidation. There is also coexisting bronchophony and whispering bronchophony. A few moist and dry bronchial râles are apt to be heard in this stage.

Exceptional.—In rare cases a few crepitant râles may be heard in this stage. In other instances, the bronchial tubes of larger size may be filled by the inflammatory lymph, so that the vocal resonance is diminished instead of being intensified, and all respiratory sounds may be suppressed.

Early in *the third stage*, the signs are the same as in the second stage, with the addition of a few subcrepitant râles. As the stage advances, vocal fremitus becomes gradually lessened, dulness diminishes over the inflamed portion of the lung, bronchial breathing slowly gives place to broncho-vesicular breathing, and this finally to the normal respiratory murmur. Subcrepitant râles appear early in this stage, and continue, often associated with mucous râles in the larger bronchi, until resolution is nearly complete.

The crepitant râle also occasionally reappears; it is then known as the crepitant râle redux.

Bronchophony, which was present in the second stage, gradually

gives place to exaggerated vocal resonance, and this, in turn, to the normal sounds of the voice.

DIAGNOSIS.—Pneumonia is to be diagnosticated from pleurodynia, intercostal neuralgia, pleurisy, pulmonary œdema, collapse of the air vesicles, hydrothorax, phthisis, and bronchitis; also, in children, from meningitis on account of the delirium, occasional contractions of the posterior cervical muscles and other convulsive phenomena. In the aged or debilitated, on account of the typhoid symptoms and occasional absence of the usual symptoms of inflammation of the lung, it may be mistaken for typhoid fever.

It is not likely to be mistaken for *pleurodynia* or *intercostal neuralgia* by any one familiar with physical diagnosis, as these diseases yield no signs excepting those due to pain.

From *pleurisy* it is distinguished by the following features:

PNEUMONIA.	PLEURISY.
Symptoms.	
Deep-seated, comparatively dull pain, marked chill, high temperature, cough with viscid or rusty sputum.	Pain superficial, and lancinating, usually absence of marked chill and high temperature, absence of rusty and viscid sputum.
Signs.	
First Stage.	*First Stage.*
Moderate dulness with feeble respiration. Numerous crepitant râles only on inspiration, and exaggerated vocal resonance.	Resonance normal. Respiratory murmur feeble or absent. Ordinarily grazing or creaking friction sounds, both inspiratory and expiratory; but occasionally transitory crepitating friction murmurs few in number as compared with crepitant râles usually heard during three or four inspirations, then disappearing, to return in a few moments.
Second Stage.	*Second Stage.*
Vocal fremitus exaggerated. Dulness marked with no change of the upper limit by changes in the position of the patient. Bronchial respiration and bronchophony.	Vocal fremitus absent. Flatness instead of dulness. The line of flatness changes with changes in the patient's position. Usually absence or marked feebleness of all respiratory and vocal sounds.
Third Stage.	*Third Stage.*
Subcrepitant râles in addition to the harsh respiration, exaggerated vocal fremitus, and resonance, and dulness of the second stage.	Friction fremitus and murmur; absence of râles. Respiratory and vocal signs feeble or nearly normal. More or less dulness.

There is a liability to mistake *pulmonary œdema* only for the first and third stages of pneumonia. The diagnosis is generally easily made

if we recollect that œdema is usually a bilateral, and pneumonia a unilateral disease. In œdema, the dulness is slight, and occurs on both sides; while in pneumonia it is marked, and commonly found only on one side.

Crepitant râles are few in œdema and nearly always associated with larger moist râles. In the first stage of pneumonia crepitant râles are very abundant, and seldom associated with other moist sounds.

Subcrepitant râles in œdema are heard upon both sides, and are not high in pitch or metallic in quality. In pneumonia they are found only on one side, and are high in pitch and usually metallic.

Œdema usually follows some protracted disease, as, typhoid fever. Pneumonia is generally a primary affection, and is attended by marked febrile symptoms which are absent in œdema.

Pneumonia is distinguished from *pulmonary collapse or atelectasis* by the history and *ensemble* of physical signs, rather than by any pathognomonic characteristics. The points of distinction are shown in the following table:

PNEUMONIA.	PULMONARY COLLAPSE.
History.	
Usually a primary affection involving only one lung.	Generally a sequel of bronchitis, often involving both lungs.
Percussion.	
Marked dulness.	Moderate dulness, frequently vesiculo-tympanitic resonance in the vicinity.
Auscultation.	
In the first and third stages, crepitant and subcrepitant râles. Second stage, bronchial breathing; exaggerated respiration over healthy lung. Râles and other abnormal signs usually confined to one lung or one lobe of that lung.	Few if any crepitant or subcrepitant râles. Bronchial breathing over collapsed lung; prolonged emphysematous expiration near it. Râles due to bronchitis over both lungs. Other signs due to collapse more apt to affect both lungs and not likely to involve an entire lobe of either.

The distinction between pneumonia and *hydrothorax* is shown below:

PNEUMONIA.	HYDROTHORAX.
Unilateral dulness, and the respiratory and vocal signs of consolidation.	Bilateral flatness, with absence of respiratory and vocal signs.

To distinguish pneumonia from *phthisis*, a knowledge of the history and the symptoms is frequently essential. Many physicians, where the signs of pneumonia have continued for more than four or five weeks, consider the case one of consumption; but this rule will not always hold good. The distinctive features between these two diseases, as they ordinarily present themselves, may be seen in the following table:

PNEUMONIA.	PHTHISIS.
An acute affection usually involving the greater portion of the lower lobe of one lung and giving rise to the signs of consolidation.	A protracted disease coming on insidiously, nearly always beginning at the apex of the lung, and at first involving only a limited amount of tissue; giving rise, first, to the signs of slight and subsequently to those of greater consolidation.
Symptoms.	
Breathing panting. Marked pyrexia terminating in crisis.	Breathing hurried but natural. Irregular and intermittent temperature.
Microscopic.	
Pneumococci.	Tubercle bacilli.

Phthisis following upon pneumonia will be distinguished from prolonged cases of the simple inflammation by the history and by the physical signs obtained on repeated examinations, and in most cases by finding tubercle bacilli in the sputum.

Any one familiar with physical diagnosis cannot mistake *bronchitis* for the early stages of pneumonia. The râles of the resolving stage of pneumonia might be mistaken for those of bronchitis; but there is no danger of error if we remember that the latter is a bilateral disease and causes little or no dulness on percussion, and that, when dulness does occur, it disappears after cough and free expectoration.

Though in some cases the symptoms of pneumonia are like the symptoms of *meningitis* and *typhoid fever*, the diagnosis is readily made by careful physical examination.

PROGNOSIS.—Uncomplicated pneumonia usually runs its active course in from five to ten days. The symptoms increase till the day of crisis, when they suddenly remit or subside by lysis. The crisis, usually occurring anywhere from the fifth to the ninth day, is marked by a sudden fall of temperature, often to one or two degrees below normal, accompanied by decrease in severity of the other symptoms, and followed by sleep, or in children by stupor. There is also not infrequently a critical hemorrhage from the kidneys, bowels, or nasal mucous membrane, and usually a profuse perspiration occurs. In the feeble or aged the critical discharge may occur as diarrhœa.

The mortality in pneumonia ranges from ten to twenty per cent, varying in different seasons and years, but in the weak and aged averaging much higher. The prognosis is worse for women than for men, for infants than for adults under sixty. In persons over sixty, and in those addicted to the excessive use of alcoholic stimulants, the disease is exceedingly fatal. In general, fatality is proportionate to the extent of lung tissue involved and to the severity of the fever. Double pneumonia usually terminates in death, and pneumonia of the apex is said to

be especially unfavorable in the aged and in children. Complicating pericarditis, valvular disease of the heart, Bright's disease, diabetes, pleurisy, tuberculosis, emphysema, and pulmonary abscess or gangrene greatly lessen the chances of recovery. The most prominent unfavorable symptoms are as follows: A pulse in adults above 120 beats to the minute, in children above 130, or marked irregularity in its rhythm; rapid respiration with low temperature; fever above 104° F. for more than forty-eight hours; a gradual rise of temperature after the fourth, or continued fever beyond the tenth day; delirium and coma, or in children convulsions occurring late; signs of collapse at any stage of the disease; hæmoptysis or copious prune-juice expectoration; suppression of the sputum in the third stage or its becoming fetid. Death occurs from asphyxia or more frequently from heart failure.

TREATMENT.—Within the first ten or fifteen hours from the inception of the attack, a blister will sometimes prevent further development of the inflammatory process; but patients are seldom seen by a physician early enough to allow of the use of this agent. Calomel administered in grain doses every hour until its purgative effects are produced is said to abort some cases, but it should not be given to debilitated patients.

For the first two or three days, small doses of aconite or veratrum viride are very useful. They should be given often, in just sufficient doses to keep the pulse nearly down to its natural rate; they must not be continued after the third day. During the same period fluid ext. of ergot, in doses of ℥xx. to xxx. every three or four hours, is often very useful, relieving congestion and checking the inflammation.

After the second day quinine in doses of three to five grains every three to five hours is the best antipyretic. In the inception of the disease, phenacetine, gr. v. to x., or antipyrine in similar doses are often productive of the best effects in relieving fever; but as soon as the heart begins to weaken, they should be employed, if at all, with the greatest caution. It is unsafe to use them continuously, and seldom desirable to administer more than three or four doses of either in the beginning of the disease, or more than one or two small doses during any twenty-four hours after the second day of the attack. It should be remembered that phenacetine is less depressing to the heart than antipyrine, but apparently possesses only about one-half the antipyretic power.

During the active stage of inflammation, large, hot jacket poultices, enveloping the whole side, are beneficial if they can be kept constantly and thoroughly applied; otherwise they do harm. When poultices cannot be managed satisfactorily, an oil-silk jacket should be employed, with warm clothing. The constant application of heat or cold produces the same results in acute inflammations; therefore, in some instances when the temperature is high, excellent results may be obtained by the application of cold over the affected organ; preferably by means of the coil

of rubber tubing through which a current of ice-water is kept circulating. From the very first, the patient should keep perfectly quiet, neither moving nor speaking excepting when absolutely necessary.

Very small doses of opium or moderate doses of chloral are sometimes necessary to relieve pain and restlessness, but either must be given very carefully, and opium is especially objectionable when the evidence of imperfect aeration of blood is distinct. Many patients have undoubtedly been hurried to the grave by the injudicious use of opium in this disease.

Where there is much prostration, and the heart is weak, strychnine gr. $\frac{1}{30}$ to $\frac{1}{20}$ or tincture of nux vomica in full doses with or without tincture of digitalis every three or four hours is very important. Alcoholics or ammonium carbonate are required in the same condition; and if œdema of the lungs appears, alcoholic stimulants in large and oft-repeated doses are of the utmost importance.

The ammonium salt is evanescent in its effects, but acts promptly.

Ammonium iodide, ammonium chloride, calcium chloride, liquor potassæ, or potassium acetate are useful in the later stages to favor resolution and prevent caseation. Late in the disease counter-irritation is beneficial. Cathartics and blood-letting should not be employed excepting in rare instances, in robust patients. When patients are much prostrated and delirious, great care should be taken to prevent them from sitting up or getting out of bed, for this will sometimes cause immediate death.

Liquid diet should be given regularly during the height of the attack; as a rule, a half pint of milk or its equivalent being given every three hours.

The experiments of G. and F. Klemperer (*Berliner klinische Wochenschrift*) on the curative effects of the blood-serum of immune animals, or antipneumotoxin, are extremely interesting, but as yet the results are not authenticated.

LOBULAR PNEUMONIA.

Synonyms. — Catarrhal pneumonia; broncho-pneumonia; disseminated pneumonia. Chronic, interstitial, or interlobular pneumonia is often included in this term.

Lobular pneumonia is an inflammation of single lobules or groups of lobules scattered through the lung, preceded and accompanied by bronchitis.

Anatomical and Pathological Characteristics.—The surface of a lung, which is the seat of catarrhal pneumonia, if the disease is superficial, presents rounded, isolated, reddish-brown or gray spots, often slightly raised, varying in size up to that of a walnut. These may be confined to a lobule or may be scattered over one or both lungs. At these points crepitation is diminished or absent, the lung is more fria-

ble and cannot be inflated. Section reveals a mottled appearance due to isolated dark brownish areas of consolidation, interspersed, in advanced stages, with others of a lighter hue; from the former, thick, reddish secretion escapes, from the latter, it has more of a milky appearance; pus may also be pressed from the bronchioles. The granular formations characteristic of the red hepatization of croupous pneumonia are absent in the catarrhal form. Here the nuclei of consolidation are composed of scattered groups of bronchioles with their immediately related vesicles. Inflammation commencing in the bronchioles involves the air vesicles by direct extension or by aspiration into them of irritating secretions. The microscope shows some of the alveoli collapsed, but the majority are more or less filled with serum, leucocytes and epithelial cells with varying degree of fatty degeneration according to the duration of the disease. The local effects of this inflammation are similar to those of croupous pneumonia, except that the products of catarrhal pneumonia contain much less fibrin and fewer red corpuscles. The walls of the bronchioles are thickened and infiltrated with round cells, and their epithelium is largely exfoliated. Their calibre is in some places contracted, in others dilated. The small tubes are always blocked with catarrhal secretion. There is also usually present more or less peribronchitis. The alveolar walls are congested.

The alveoli adjacent to these areas of consolidation may be emphysematous and are often the seat of congestion and œdema. The pleura over them may be inflamed. The pulmonary lymphatic glands are commonly enlarged. Catarrhal pneumonia terminates in resolution, suppuration, gangrene, or in chronic fibroid induration, or the products may undergo caseous or tubercular degeneration.

Etiology.—Lobular pneumonia is most common in infancy before the third year, and in advanced age. Bad sanitary conditions, poor food and shelter, and debility are predisposing factors. It is always secondary to affections of the smaller bronchi, and hence arises from exposure to the exciting causes of bronchitis. It is apt to follow influenza and the bronchitis which complicates contagious diseases, especially measles and whooping-cough.

Symptomatology.—The essential symptoms are rapidity of the pulse and of respiration, usually with high temperature and troublesome cough and emaciation, occurring in the course of a bronchitis.

The pulse, at first strong, frequently becomes feeble and compressible and runs up to from 140 to 160 per minute, and the respirations from 60 to 80. The temperature gradually rises with irregular exacerbations and remissions to 104° or 105° F., and in fatal acute cases may go two or three degrees higher. The cough loses its bronchial character and becomes hacking and painful, and is followed by but little expectoration which may be streaked with blood.

The most important *signs* are deficient respiratory movements, slight

and occasionally "patchy" dulness, with deficient vesicular murmur and, on forced inspiration, numerous poorly defined mucous clicks. When only a limited number of lobules are affected, a diagnosis cannot be accurately made; but if several lobules are involved, the signs become quite distinct.

By inspection we shall usually observe rapid but imperfect respiratory movements, with very slight expansion of the ribs during inspiration, but considerable elevation of the chest walls, especially at the upper part; and at the same time falling in of the soft parts of the chest and retraction of the lower ribs, as in pulmonary emphysema. The inspiration is often shortened and the expiration prolonged.

When several inflamed nodules exist, especially if they are located near the surface of the lung, palpation will discover exaggerated vocal fremitus.

Upon percussion, dulness will be found, varying in degree with the amount of consolidation. This is nearly always limited to the inferior and posterior portions of the chest, and usually occurs on both sides; but the disease may be confined to one lung or to the upper lobes of the lungs.

By auscultation more or less broncho-vesicular or bronchial respiration with exaggerated vocal resonance and moist high-pitched râles will usually be found over the lower part of the lungs. Likewise, over the upper and anterior portions of the chest we ordinarily find the signs of pulmonary emphysema, viz., vesiculo-tympanitic resonance, with a prolonged and low-pitched expiratory murmur.

After protracted or repeated colds, the occurrence of a feeble vesicular murmur, with several illy defined mucous clicks on forced inspiration, should cause us to suspect lobular pneumonia. The mucous clicks in these cases are due to retention of the catarrhal products in the air cells.

High-pitched bronchial râles are also significant of consolidation. In children, some of the alveoli are often completely choked, so that few râles are produced. In adults, the inflammatory products are more fluid, and consequently râles are more abundant.

DIAGNOSIS.—The diagnosis of lobular pneumonia is very difficult, unless a considerable number of lobules are affected. Even then, the disease cannot always be detected by the physical signs alone, but, as in some cases in other pulmonary affections, the history and symptoms must be weighed with the signs, before a positive opinion can be formed. For example, in a child suffering from bronchitis, if the respiration suddenly becomes accelerated, the temperature elevated, and the cough, which may previously have been loose and easy, becomes dry, hacking, and painful, we have good reason to think that the vesicular portion of the lung has become involved in the inflammatory process. If, in addi-

tion to these symptoms, the signs of consolidation which have just been enumerated are present, the diagnosis may be considered certain.

The distinctive features between *capillary bronchitis* and lobular pneumonia may be found under the differential diagnosis of capillary bronchitis.

Lobular pneumonia is often preceded and accompanied by collapse or atelectasis of many of the air vesicles; for this reason the signs of the two diseases are usually considered identical. If any considerable amount of tissue is involved, and the two conditions are not combined, a differential diagnosis can be made by attention to the following symptoms and signs:

Lobular pneumonia.	Pulmonary collapse.
Symptoms.	
Temperature suddenly increased; cough becomes dry and paroxysmal.	The elevation of temperature, and the cough, which are incidental to the associated bronchitis, are not materially affected by collapse of the air vesicles.
Inspection.	
Falling in of the lower portions of the chest, which may have been noticed in bronchitis, partially disappears.	The inverted action of the inferior ribs is increased in proportion to the extent of atelectasis.
Palpation.	
Vocal fremitus is increased.	The vocal fremitus is not likely to be increased, but, on the contrary, it may be diminished.
Percussion.	
Uniform dulness, or distinct patches of dulness, usually marked over the lower portions of the chest.	The dulness is not so distinct, and there is occasionally vesiculo-tympanitic resonance. The dulness usually occurs first at the border of the left lung, where it overlaps the heart; and shortly afterward at the base of the lungs. From the latter position it has a tendency to spread upward in an elongated, somewhat pyramidal form along the lines of the intervertebral grooves, in which position it may reach as high as the apex of the lung.
Auscultation.	
The respiratory sounds generally harsh or broncho-vesicular in quality, never wholly tubular. The mucous râles of bronchitis usually heard over the entire chest; but, in many instances, finer moist râles are obtained,	The respiratory sounds usually feeble. The râles of bronchitis are less likely to be present than in lobular pneumonia, and are seldom heard over the collapsed lobules. Sometimes deep inspirations may bring out a few crep-

limited to a small space immediately over the inflamed lobules. When the finer bronchi are dilated, as sometimes happens in this disease, the râles become coarse and somewhat metallic if the dilatations are surrounded by consolidated lung.

itant râles, which are heard with three or four inspiratory acts, and then disappear.

The differential diagnosis between lobular pneumonia and *lobar pneumonia* appears below:

LOBULAR PNEUMONIA.	LOBAR PNEUMONIA.
Symptoms.	
Begins with a bronchitis.	Begins with chill.
No chill.	Pain in the side.
No crisis.	Terminal crisis.
Signs.	
Often over both lungs but in small, scattered areas.	Usually confined to one side and to one large area.
Dulness not marked.	Dulness marked.
Mucous with smaller râles.	Crepitant and subcrepitant râles.
Broncho-vesicular voice and breathing.	Bronchial voice and breathing.

The following is the differential diagnosis between lobular pneumonia and *acute tubercular phthisis:*

LOBULAR PNEUMONIA.	ACUTE TUBERCULAR PHTHISIS.
Symptoms.	
In children and the aged.	In young adults.
Initial bronchitis.	Initial pyrexia precedes the physical signs.
Hæmoptysis not common.	Hæmoptysis common.
Emaciation and exhaustion very rapid.	Emaciation less rapid.
Signs.	
Most marked in lower and posterior parts.	Most marked at apex.
No tubercle bacilli.	Sputum sometimes contains tubercle bacilli.

PROGNOSIS.—This disease may terminate fatally within two or three days, or may extend over many weeks or months, ending in resolution and recovery, or in purulent infiltration, or in cheesy or tubercular degeneration and death: or the inflammation may cause extensive new connective-tissue formation in the interalveolar septa and about the bronchial tubes, eventuating in fibroid phthisis, which may extend over several years.

The disease is most fatal in infants, especially when following whooping-cough or measles, and in aged or greatly debilitated subjects. Death results in from 30 to 40 per cent of all cases, some authors placing the mortality even higher. Among the grave symptoms are: extension of

the bronchitis and increasing cyanosis; irregularity of the respirations and inefficient, feeble cough with cessation of expectoration; a rapid, feeble pulse; temperature exceeding 104° F., and stupor or convulsions in the later stages of the disease.

TREATMENT.—Lobular pneumonia is nearly always a secondary affection, due to extension of the inflammatory process from the bronchial mucous membrane in consequence of debility. Bearing this in mind, we avoid all depressing remedies such as antimony, aconite, or veratrum viride, and very early commence the use of stimulants.

Quinine is the best remedy to moderate the fever. Alcohol should be given according to the amount of depression. The rule is to give as much as can be borne without causing head symptoms. Ammonium carbonate or ammonium iodide are very useful, not only for the stimulation which they afford, but also for their beneficial effects in removing the products of inflammation.

Sedative inhalations are useful early in the attack, and at a later period stimulant inhalations and counter-irritation are beneficial. If the patient emaciate, calcium chloride, tincture of iron, and cod-liver oil are indicated. A change of climate is advisable if recovery does not take place within eight or ten weeks.

PECULIAR FORMS OF PNEUMONIA.

Several somewhat peculiar forms of pneumonia merit passing consideration, though they are not distinct varieties of the disease. These are: interstitial pneumonia, typhoid pneumonia, bilious pneumonia, pneumonia due to cardiac disease, and pneumonia from Bright's disease.

The treatment of these forms is essentially the same as that for the diseases with which they are associated, combined, as occasion may seem to require, with the resolvents and expectorants indicated in lobular pneumonia.

CHRONIC OR INTERSTITIAL PNEUMONIA (sometimes termed catarrhal pneumonia) will be described under the head of Fibroid Phthisis.

TYPHOID PNEUMONIA is a term that may be applied to a certain complication. If pneumonia complicates typhoid fever, or *vice versa*, the symptoms of the one disease are associated with and somewhat modified by those of the other, and the resulting prostration is marked. The secondary pneumonia is here indicated by increased rapidity of the pulse and respiration, with signs of consolidation. Cough and sanguinolent sputum are rarely present.

The expression typhoid pneumonia also refers to pneumonia of a sthenic and usually fatal form, frequently epidemic among soldiers and others subject to unhealthful sanitary conditions. The chief features are extreme exhaustion and constant tendency to collapse, although the

local pulmonary signs may be but slight. Symptoms like those of septicæmia may be prolonged for months.

Peculiarly viscid subcrepitant râles may be heard, few in number and found irregularly at the base or apex of the lung.

BILIOUS PNEUMONIA, which is most common in malarial districts, is, in addition to the symptoms of typical croupous pneumonia, characterized by jaundice, greenish, viscid, and inodorous stools, with other evidences of hepatic and gastric disorder, and a fever record intermittent in type, the febrile exacerbations being sometimes preceded during the early part of the day by chilly sensations and coolness of the ends of the nose, fingers, and toes.

PNEUMONIA ARISING FROM DISEASE OF THE HEART, especially from marked mitral lesions, presents many features similar to those of lobular pneumonia. The invasion is usually slow, seldom preceded by rigors. There is a chronic cough, with expectoration which seldom becomes rusty or tenacious. The signs may appear in scattered patches, which change their seat from day to day, but are usually found over the lower lobes of both lungs.

There is some exaggeration of the vocal fremitus, slight dulness, and blowing though not strictly bronchial respiration, with exaggerated vocal resonance.

PNEUMONIA FROM BRIGHT'S DISEASE may not differ materially from ordinary acute pneumonia, or it may begin in collapse of portions of the vesicular structure, and present characteristics similar to those of lobular pneumonia.

ABSCESS OF THE LUNG.

Abscess of the lung consists of a circumscribed collection of pus within the pulmonary parenchyma. It is usually characterized by pain, rigors and fever, and later by expectoration of a small amount of blood immediately followed by a large quantity of pus, which escapes within a few hours. These abscesses are rare excepting when secondary to tuberculosis, pyæmia, or embolism, in which cases they are usually multiple and must be considered as incidental to the primary disease. They may also result from the entrance of foreign bodies into the air passages, obstruction of the bronchi by tumors, or from suppuration of the bronchial glands; also from perforating abscesses from below the diaphragm or from the mediastinum. The pulmonary abscesses which chiefly interest us are those resulting from acute pneumonia.

SYMPTOMATOLOGY.—The abscess usually follows within a few days, upon some exposure, and occurs during the acute stage of the inflammation, being preceded by the chill and fever of acute pulmonary inflammation; but sometimes it occurs after the pneumonia has subsided. The formation of pus is commonly attended by rigors which are followed

by hectic fever. Pain is usually present in the beginning. There are irregular chills, and the temperature fluctuates during the day two or three degrees. In the milder cases, though the pulse is rapid, the pyrexia is not pronounced, and spontaneous opening and cure may be expected in the course of a few weeks.

Unless the patient dies of exhaustion, the abscess commonly opens within ten to twenty days, the profuse purulent discharge commonly being preceded by a few drops of blood or bloody pus. Often from half a pint to a pint of yellowish or greenish, though occasionally brownish, pus is expectorated within a few hours. The pus, excepting in cases of gangrene or where it has been long retained, is not particularly offensive.

Occasionally the abscess ruptures into the pericardium or pleura. The sputum commonly contains small yellowish or dark-colored bits of lung tissue visible to the naked eye, which upon microscopic examination are found to contain elastic fibre.

The signs are: dulness, with feebleness or absence of the respiratory murmur over the abscess, combined with indistinct râles and sometimes bronchial breathing in the lung tissue about it, and after escape of pus, for a short time the signs of a cavity.

Diagnosis.—The affection is liable to be mistaken for bronchitis, pneumonia, or acute or chronic pleurisy. The most important features in the diagnosis are: the symptoms of acute pneumonia followed by irregular chills and fever; dulness more or less circumscribed, but apt to be more distinct than that of pneumonia and less than that of pleurisy; atypic respiratory and vocal signs, and finally sudden expectoration of a large quantity of pus in which may be found elastic fibre.

Bronchitis is distinguished by absence of the initial chill and subsequent rigors, slight fever, absence of dulness on percussion, and the presence of bilateral râles; and by the character of the expectoration.

Pneumonia yields very similar symptoms and signs, but seldom causes the irregular chills and fever. In pneumonia the dulness may be less or more marked according to the size of the abscess and the amount of healthy lung tissue between it and the surface; but eventually the dulness in case of abscess becomes more distinctly circumscribed. In pneumonia distinct crepitant and subcrepitant râles or bronchial breathing are practically always present, while over a pulmonary abscess there may be a feeble normal murmur or absence of respiratory sounds, or there may be irregular bronchial râles, which are likely to be most distinct in a zone surrounding the abscess.

Acute pleurisy may be differentiated by the presence of friction sounds and fremitus, but absence of vocal fremitus. In it there is more decided dulness, and less distinct respiratory and vocal signs than in abscess, and there is no hectic fever. When there is much effusion, change of the level of flatness by changing the patient's position and displacement of the heart differentiate it from abscess.

Chronic pleurisy, or empyema, when general, can be easily distin-

guished from abscess of the lung, but when circumscribed the signs are not characteristic until a microscopic examination of the pus reveals elastic fibre in the case of abscess but none in empyema.

PROGNOSIS.—The affection may prove fatal within two or three weeks or may be prolonged for months. If the abscess opens spontaneously it will usually do so within three weeks. Many cases die of exhaustion, some by infection of other parts, and still others by repeated pneumonias developing about the purulent cavity; yet a considerable number recover. The cases caused by pyæmia, gangrene, tuberculosis, embolism, are necessarily grave.

TREATMENT.—Commonly the profession favors expectant treatment with tonics and ample nourishment, but when the abscess can be located, especially if near the chest wall, the question of surgical interference must be considered. Knowing the danger of the operation and remembering that many cases recover spontaneously, I believe that the greatest good to by far the greatest number will be obtained in most cases by pursuing the expectant plan for at least three or four weeks; but when we have reason to believe that there is a single abscess near the surface of the lung, when sufficient time has been given for spontaneous opening, and when progressive emaciation and hectic fever indicate the retention of pus, it is safer for the patient to open the abscess from without.

Aspiration alone or combined with washing out the cavity with a disinfecting solution will prove curative in a considerable number of cases and should be tried first, but if it fails the surgeon, with antiseptic precautions, should cut down and resect a portion of one or more ribs. If the lung is found not adherent to the pleura it should be drawn up and stitched to the external pleura, where it will become firmly attached within a few hours. Then (or at once if the two surfaces of the pleura are adherent) an opening should be made through the lung tissue to the cavity by means of the thermo-cautery, and a large-sized drainage tube introduced. Strong's tubes spoken of in treating of empyema (Fig. 23) are well adapted for this purpose. The cavity should subsequently be managed as those of other abscesses, and the patient sustained by tonics and nutritious diet.

CHAPTER IX.

PULMONARY DISEASES.—*Continued.*

PULMONARY HYPERÆMIA.

Pulmonary hyperæmia signifies an excess of blood in the pulmonary vessels. It may be general or local, active or passive. It possesses no distinctive physical signs unless associated with pulmonary œdema or bronchial hemorrhage.

Anatomical and Pathological Characteristics.—Lungs which are the seat of *active* hyperæmia are redder, slightly heavier, and less crepitant than normal. An unusual amount of arterial blood escapes on section. The capillaries are distended, the alveolar epithelium is swollen, and the bronchial mucous membrane may be injected. Œdema may accompany a local active hyperæmia. Active hyperæmia may speedily disappear or it may terminate in inflammation.

In *passive* hyperæmia or congestion, the lungs are of a dark red or purple color, the dependent parts showing marked post-mortem staining of a darker hue; the organs are heavier and less crepitant than normal, and the flow of blood on section is copious and dark, but mixed with air. The capillaries are engorged, distended, and tortuous; the air sacs contain serum with blood corpuscles, leucocytes, and epithelial cells more or less granular. The connective tissue is usually slightly œdematous and shows small extravasations. In severe and continued congestion these changes are exaggerated, there is greater thickening of the alveolar walls, engorgement of the vessels, œdema, collapse of some of the air sacs, and decrease in the amount of air in the lung, which is of dark red color dotted with lighter points of extravasation, partially decolorized. The fluid from the cut surface is more watery. This condition is termed splenization. Prolonged obstruction to the pulmonary circulation due to mitral disease results in *brown induration*. Here, in addition to the capillary engorgement and alveolar changes, there is extensive pigmentation of the lung along the lymphatics and vessels and about the connective-tissue cells, from deposit of brown granules of hæmatin derived from the degenerate red corpuscles and carried thither by the leucocytes. There is also marked connective-tissue hyperplasia. The lung is consequently dark brown in color with yellowish and reddish patches due to extravasations in various stages of decoloration. It is larger, heavier, firmer, less œdematous, and drier than a splenoid lung.

Hypostatic congestion signifies passive hyperæmia of dependent parts, usually bilateral and due to cardiac weakness in those long confined to bed by exhausting diseases.

ETIOLOGY.—*Active* hyperæmia may be due to increased cardiac action from violent exercise, medicinal stimulation, mental excitement, and certain neuroses, or to local irritation from inhalation of pungent gases, foreign bodies, and hot or cold air; or to diminution of inter-alveolar pressure in the rarefied atmosphere of high altitudes or during inspiratory expansion of the chest with obstructed air passages, as in croup, œdema glottidis, and tumors of the larynx. Lastly, interference with the circulation in one part of the lung may cause compensatory or collateral hyperæmia of the other parts.

Passive pulmonary hyperæmia is due either to inefficient propulsion of the blood through the lung from weakness or inefficiency of the right heart or to obstruction in the pulmonary artery or to interference with the outflow of blood from the lung owing to valvular disease or weakness of the left heart or pressure on the pulmonary veins.

SYMPTOMATOLOGY.—We can best recognize pulmonary congestion by considering its history and symptoms, in connection with the physical signs. For example, if a patient is attacked with sudden dyspnœa after extreme physical exertion or exposure to the influence of a rarefied atmosphere, as in high altitudes, pulmonary congestion should be suspected; and if the dyspnœa is attended with a profuse watery and blood-stained expectoration and the signs of œdema, we may be positive of our diagnosis.

In such cases percussion reveals slight dulness over the lower portions of the chest.

Auscultation reveals a feeble respiratory murmur, crepitant râles, and usually an abundance of large and small mucous râles.

Accentuation of the *second* sound of the heart, at the pulmonary orifice, has been considered by some authors diagnostic of pulmonary congestion; but this sign cannot be relied on, as it may be only relative, due to feebleness of the aortic sound; moreover, this accentuation is a common sign in cardiac disease.

In the congestion of the lung which immediately precedes pneumonia, physical examination reveals very slight dulness, with feebleness of the respiratory murmur and, possibly here and there, a crepitant or subcrepitant râle. This condition, however, is not usually included under the head of pulmonary congestion.

PROGNOSIS.—Active pulmonary hyperæmia may cause death within a few hours from œdema or hemorrhage, or it may terminate in pneumonia. It is ordinarily amenable to early and prompt treatment. Mild cases are usually of short duration and recover spontaneously. Passive hyperæmia is more serious, but the prognosis depends largely upon the gravity of the cause. Chronic cases due to heart disease are liable to sudden fatal attacks of œdema.

TREATMENT.—When the congestion comes on suddenly, full doses of ergot should be given. Bleeding will be found useful in cases of extreme plethora. Dry or wet cupping over the chest is sometimes beneficial. A blister will occasionally prevent the supervention of inflammation. If the heart is weak, it should be stimulated; and if pulmonary œdema coexist, alcoholic stimulants should be given freely and a hydragogue cathartic may be administered.

BROWN INDURATION.

The *symptoms* of brown induration are those of the causative initial disease, with cough and hæmoptysis.

The principal *sign* is dulness, limited mostly to the second intercostal space near the sternum. There are also exaggerated vocal resonance, broncho-vesicular or bronchial breathing, bronchophony, and occasionally pectoriloquy.

This affection may be differentiated from other pulmonary diseases by the position of the dulness and the presence of the symptoms and signs of mitral disease.

TREATMENT will aim to relieve the cardiac affection. Ammonium carbonate and chloride, moderate doses of digitalis and tincture of nux vomica, are especially indicated, and counter-irritation may be beneficial.

PULMONARY HEMORRHAGE.

Pulmonary hemorrhage includes hemorrhage from the bronchi (bronchorrhagia) and from the parenchyma of the lung (pneumonorrhagia). The chief symptom is hæmoptysis. This term, used loosely, in a broad sense denotes spitting of blood, whether in large quantity as from the rupture of an aneurism into the air passages, or in small amount, merely streaking the sputum of chronic bronchitis, or as found in the rusty or prune-juice expectoration of pneumonia. Properly, it signifies the raising of more or less pure blood from vessels bleeding into the larynx, trachea, bronchi, or alveolar structure.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—The appearance of the lung after pulmonary hemorrhage depends upon the extent of the hemorrhage, its cause, and the time at which the organ is inspected. If post-mortem examination is made soon after bronchial hemorrhage, the lung in general may be anæmic, marked by isolated bright red spots at points where blood has gravitated or has been drawn into superficial alveoli. On section, coagula may also be found blocking the bronchi. If these collections in the air sacs and tubes are numerous or large, the lung to that extent will be heavier, less crepitant, and less apt to collapse. Its cut surface will show red, firm patches or nodules re-

sembling infarcts, from which sero-sanious fluid escapes. The bronchial mucous membrane may appear almost normal, or ecchymotic, red, swollen and softened. If the examination be made long after death, there may be little or no remaining evidence of an abnormal condition, or the coagula in the air sacs may be partially decolorized. The hemorrhage may in some cases give rise to lobular pneumonia.

If hemorrhage has come from an abscess or tubercular cavity, an eroded vessel or ruptured aneurism may be found in the wall of the cavity or in one of the trabeculæ traversing its space. Brown induration of the lung also will often be found, with the evidence of hemorrhage due to mitral disease of long standing.

In other cases, atheromatous, fatty, or amyloid degeneration of the vessels may mark the seat of parenchymatous hemorrhage. Rarely subpleural hæmatoma and hæmothorax are present.

ETIOLOGY.—All those conditions which weaken the walls of the pulmonary blood-vessels predispose to hæmoptysis. They include tuberculosis, abscess, and gangrene, which diminish the local support of the vessels; also changes in the vascular walls, such as atheromatous, fatty, or amyloid degeneration, and atrophic changes incident to hæmophilia, purpura, scorbutus, and the infectious diseases; also heart disease and other conditions which produce chronic over-distention of the pulmonary vessels. The usual exciting causes are muscular exertion, coughing, loud speaking, or concussion from a blow or fall. Other cases occur from penetrating wounds, but in quite a large percentage of cases, no exciting cause can be discovered.

SYMPTOMATOLOGY.—The chief symptom is expectoration, usually of arterial blood, more or less frothy; perhaps immediately preceded by a sensation as of warm fluid trickling beneath the sternum. This may follow severe cough or strain and without premonition, or may be preceded by coldness of the extremities, congestion of the face, headache, dizziness, thoracic oppression, or palpitation.

Hæmoptysis may be followed by nausea and vomiting, and is apt to occasion considerable mental shock. Large and small bronchial râles are present in most cases during active hemorrhage, and may remain for several hours. Feeble respiration is sometimes noticeable and dulness may be present, though frequently no signs whatever can be detected by the most careful examination.

DIAGNOSIS.—Hæmoptysis may be mistaken for hæmatemesis, epistaxis, or hemorrhage from the gums or the pharynx. The distinctive features are as follows:

HÆMOPTYSIS.	HÆMATEMESIS.
History.	
Usually history of pulmonary or heart disease, especially phthisis.	Usually gastric or hepatic disease.

HÆMOPTYSIS.	HÆMATEMESIS.
Symptoms.	
A preceding thoracic oppression or premonitory sensation of trickling fluid beneath the sternum.	A preceding sense of pain or fulness.
Blood expelled primarily by cough. Vomiting secondary if present.	Blood expelled primarily by vomiting.
Subsequent cough and bronchial râles.	Chest signs negative.
Character of blood.	
Usually bright red and frothy from admixture of air.	Usually dark clotted or grumous; may be mixed with food.
Alkaline reaction.	Acid reaction.

In epistaxis inspection of the nares and post-nares with reflected light reveals the course of the blood and perhaps its origin. *Hemorrhage from the gums or the pharynx* can generally be readily recognized by careful inspection.

PROGNOSIS.—Pulmonary hemorrhage, though rarely immediately fatal, is in most cases indicative of phthisis. A single hemorrhage may amount to a pint or more, and continue from a few minutes to several hours. As a rule it is followed by others. In most instances it is followed by the occasional expectoration of a small amount of clotted blood for two or three days. Frequent recurrence, or severe hemorrhage if not fatal, results in anæmia or may cause lobular pneumonia. When occurring in phthisis, hæmoptysis seems occasionally to check its course temporarily; commonly the patient expresses a feeling of increased well being. Rarely, it is followed by a more rapid progress of the disease. It is a fatal symptom if due to ruptured aneurism, and serious if complicating pulmonary abscesses, gangrene, malignant growths, or when accompanying the infectious diseases or grave dyscrasia and occasionally when resulting from heart disease.

Death may occur from depleted circulation, asphyxia, or from gradual exhaustion due to anæmia or to secondary pneumonia.

TREATMENT.—The patient should be kept perfectly quiet until all bleeding ceases.

The most efficient remedies for checking the hemorrhage are full doses of ergot, gallic acid, or lead acetate and opium.

The hemorrhage may sometimes be checked by the inhalation of a spray from a weak solution of liquor ferri subsulphatis—℥ x., aqua ad ℥ i.

In estimating the value of any remedy for this purpose it must not be forgotten that the bleeding will usually cease in a short time whether remedies are used or not. Loomis relies more upon aconite and opium than upon styptics. If ice is applied to the chest, it should be done with great care, as it seems to favor the supervention of bronchopneumonia after hemorrhage (Loomis' Practical Medicine, p. 95).

PULMONARY APOPLEXY.

Synonyms.— Diffuse pulmonary hemorrhage, pneumonorrhagia, hemorrhagic infarctus.

Pulmonary apoplexy is a rare affection, consisting of extravasation of blood into the lung tissue. It usually occurs in the lower lobes.

Since apoplexy etymologically refers to loss of consciousness incident to rupture of a cerebral artery, this term is not aptly applied to interstitial pulmonary hemorrhage; usage, however, has authorized it.

Anatomical and Pathological Characteristics.—Pulmonary apoplexy consists of an escape of blood into the parenchyma of the lung from a ruptured vessel, attended by more or less laceration and infiltration of the tissues, according to the size of the damaged vessel, the cause of the injury, and the condition of the lung.

The lung is relatively heavier and firmer than normal, and contains no air in the affected portion. Not infrequently several extravasations exist from the bursting of vessels in different parts of the organ.

The resulting clots or hemorrhagic infarcts, as distinguished from embolic infarcts, are of pyramidal form, the bases of the pyramids appearing superficially beneath the pleura as dark red or almost black patches, the sides corresponding to the inter-lobular boundaries; occasionally the pleura is also torn, and blood escapes into the pleural sac. The cut surface is firm but moist and of uniformly dark color in the early stages, but later the clots gradually become decolorized. Hemorrhagic infarcts somewhat resemble true embolic infarcts, but are usually larger and more sharply defined. Apoplectic extravasation may cause death immediately or from subsequent suppuration or gangrene. It may end in resolution, complete or accompanied by cicatricial contraction, or may undergo caseation, calcification and encapsulation.

Etiology.—Hemorrhagic infarctus in the lung is usually the result of pulmonary hyperæmia acting upon vessels already the seat of degenerative changes. Such changes frequently give rise to multiple aneurisms which give way on sudden or prolonged intra-vascular pressure. A severe blow or a wound of external origin may cause diffuse hemorrhagic infiltration or it may result from erosion of a vessel by ulceration.

Symptomatology.—This affection is usually, though not invariably, attended with dyspnœa and hæmoptysis, the expectorated blood containing small dark clots.

The principal *signs* are: more or less dulness, feeble or bronchial respiration, and mucous râles.

When the coagula are few in number, and small or deep-seated, percussion yields no signs; but if they are numerous, or lie superficially, dulness will be more or less marked.

Upon auscultation, mucous, subcrepitant, and possibly well-marked crepitant râles will be detected in and about the extravasations, until coagulation of blood has taken place. Afterward, respiration will be feeble or suppressed over the extravasations; or bronchial breathing and exaggerated vocal resonance may be obtained, if a large clot lies in apposition with a bronchial tube.

DIAGNOSIS.—The diagnosis of pulmonary apoplexy must be based upon the history and the character of the sputa, in connection with the signs found upon percussion and auscultation. It is not likely to be mistaken for any other disease except pneumonia, from which it can easily be distinguished by the history and by the expectoration.

TREATMENT.—The treatment should be mainly directed to the cause of the hemorrhage. Removal of the blood-clot is probably hastened by the administration of potassium iodide, or liquor potassæ and other alkalies. Counter-irritation is useful in some cases a few days after the accident. Quiet must be maintained for two or three weeks to prevent a recurrence of the attack. If pneumonia or pleurisy supervene, they should be treated essentially the same as when they occur alone.

PULMONARY THROMBOSIS AND EMBOLISM.

PULMONARY THROMBOSIS consists of the gradual obstruction of a blood-vessel in the lung by a coagulum formed *in situ*. It occurs in the pulmonary artery or some of its branches, as a result of local non-inflammatory vascular degeneration or of inflammation in the surrounding lung tissue.

PULMONARY EMBOLISM consists of a sudden obstruction of a vessel by a foreign body, usually a fragment of a cardiac valvular vegetation or of a thrombus in some of the systemic veins. Embolism may occur in the pulmonary artery by lodgment of a thrombotic fragment from the veins of the abdomen or lower extremities or it may occur in the bronchial arteries by an obstruent brought from the mitral or aortic valves.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—Pulmonary *embolic* infarcts are usually multiple and occur near the surface of the lung, especially in the posterior part of the lower lobe. In form and gross appearance they resemble *hemorrhagic* infarcts, but they depend upon obstruction of a blood-vessel, instead of rupture. At the apex of this infarct, usually at the bifurcation of an artery, an embolus is generally to be found about which a secondary thrombus has formed. The conical form of the infarct corresponds to the distribution of the branches of the occluded vessel on the distal side of the obstruction. These being no longer supplied with blood by the main vessel, become engorged, according to Cohnheim, by regurgitation of blood from the veins, but according to Litten this is due to a small amount of arterial blood still

supplied to the part by arterioles, which, however, are not sufficient in size and number to afford adequate collateral circulation. The changes in the part, consequent upon engorgement and stasis, are: migration of leucocytes, deterioration of the tunica intima, diapedesis of red corpuscles and engorgement or collapse of the air cells with thinning of their walls. About the infarct is a zone of active hyperæmia. Embolic infarctus may terminate in resolution or cicatrization, but if infected in abscess or gangrene. Rarely caseation and calcification with encapsulation occur.

ETIOLOGY.—*Thrombosis* may be due to local vascular degeneration or inflammation extending from the adjacent lung tissue, especially in connection with feeble heart power.

Embolism may be due to loosened fragments from the cardiac valves or from systemic venous thrombi or to fat-granules drawn into the open veins at the site of a fracture or crushing injury to the long bones.

SYMPTOMATOLOGY.—The principal symptoms are sudden, severe, and sometimes paroxysmal dyspnœa, turbulent heart action, and pulsation of the jugulars, from yielding of the tricuspid valve. Exaggerated resonance is sometimes detected, owing to cutting off of the blood supply from some of the pulmonary lobules, and consequent distention of the air cells. In the same locality, the respiratory murmur will be feeble or suppressed.

DIAGNOSIS.—Neither the symptoms nor the signs of these conditions are sufficiently well understood to enable us to make a positive diagnosis in every instance. Most reliance must be placed on the symptoms and history.

PROGNOSIS.—The prognosis is unfavorable in proportion to the amount of lung damaged by the emboli or thrombus, and is always very grave if the emboli be infected. Small infarcts may undergo resolution. Death occurs from collapse, apnœa, or from secondary pneumonia, sepsis, or phthisis.

TREATMENT.—The treatment must be expectant.

PULMONARY COLLAPSE.

Synonyms.—Apneumatosis and atelectasis. The latter term, though referring to the same anatomical condition as the former, is more properly applied to air cells which remain in the fœtal condition after birth, not becoming distended with air.

Pulmonary collapse is a condition of the lungs in which air cells which have formerly been inflated have collapsed, and returned to a quasi-fœtal state.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—Both the acquired and the congenital forms may involve the whole or part of one lung or a part of each; the collapsed air cells being en masse or in isolated lobules or groups of lobules scattered through the organ. In

order of frequency, the parts affected are: the lower margin of the lower lobes of both lungs, the tongue-like prolongation of the left upper lobe, and the posterior portions of the lower and upper lobes of both lungs near the spine. The collapsed parts correspond externally to small irregular areas depressed below the general surface of a reddish-blue, violet or grayish-blue color. The cross-section is dark red, smooth, tough, airless, and the part readily sinks in water. Recently collapsed air sacs may be inflated, but if this condition long persists, distention becomes impossible and the parts subsequently undergo fatty or fibroid change or become the seat of tuberculosis. The surrounding lung tissue is not infrequently emphysematous or œdematous; the bronchi which are still pervious are frequently dilated. Permanent and extensive collapse from prolonged compression results in a dense, solid, fleshy condition of the lung, termed carnification.

Etiology.—The affection is most frequent in early childhood. It is always preceded by inflammation of the bronchial mucous membrane, the secretions from which collect in some of the smaller bronchial tubes, where, acting as ball valves, they obstruct the entrance of air during inspiration, but permit its escape in expiration. Ultimately the air cells to which the obstructed bronchus is distributed become in this manner completely emptied of air and collapsed.

Congenital atelectasis occurs in weak and sickly infants or may be due to premature delivery, and it may result from accidents in birth, such as the inspiration of amniotic and other fluids.

In children, more or less permanent collapse is apt to follow an attack of bronchitis, whooping-cough, measles, typhoid fever, severe diarrhœa, or any other exhausting disease. Disease of the brain or spinal cord interfering with the pneumogastric nerve may cause it. Collapse of the lung may be due to the pressure of mediastinal or intra-pulmonary tumors, or to effusion into the pleural sac.

Symptomatology.—The essential symptoms are: great prostration; pallor or duskiness of the skin, which hangs in loose folds on the emaciated limbs; rapid, feeble pulse and coldness of the extremities; a feeble, insufficient cough; great dyspnœa, without the lividity which usually attends this symptom, and rapid respiration, rising in young children from sixty to eighty per minute, with an altered rhythm in the respiratory acts. In this alteration of rhythm the pause follows inspiration and precedes expiration, instead of occurring between expiration and inspiration, as in health.

The chief *signs* are: retraction of the intercostal spaces and lower ribs during inspiration, dulness over the collapsed lung when the apneumatosis is considerable, and feeble or absent vesicular murmur, usually with harsh or bronchial respiration over the affected parts.

Inspection reveals the rapidity of respiration and its changed rhythm and retraction of the intercostal spaces and lower ribs during inspira-

tion. The latter is a very important sign, but it also occurs in other diseases.

In children the signs of percussion are not so reliable as in adults, but when the disease is well marked, more or less dulness will be found over the affected portions, usually first at the base of the lungs, then at their anterior borders, and finally along the spinal column. If a whole lobe is involved, dulness like that of pneumonia will be present. Not infrequently the collapsed cells are so scattered through the lungs, and the adjacent cells are so distended, that the affection may be quite extensive without giving any signs on percussion.

By auscultation, harsh or bronchial respiration may be heard over the collapsed cells instead of the vesicular murmur.

Usually portions of the lung immediately surrounding the affected lobules remain pervious to air, so that the vesicular murmur is not entirely lost; the sounds from the air vesicles are then mingled with those from the bronchi, causing broncho-vesicular respiration. Ordinarily, numerous bronchial râles are present, which may completely mask the vesicular murmur.

DIAGNOSIS.—Pulmonary collapse is most likely to be mistaken for pneumonia or pleuritic effusions.

The diagnosis in many cases must depend mainly on the symptoms, as the signs are by no means distinctive. Whenever dulness occurs, its rapid appearance, within twenty-four or thirty-six hours succeeding the signs of bronchitis, is an element of great value in diagnosis.

In pulmonary collapse there are few if any crepitant râles, which are considered pathognomonic of *pneumonia*. In the latter disease there is not the retraction of the chest noticed in collapse, and dulness is usually greater and the bronchial breathing more marked than in the disease under consideration. The fever symptoms are more marked in pneumonia.

The features that distinguish *pleurisy* from pulmonary collapse are the flatness instead of dulness on percussion, change in the level of flatness and absence of vocal fremitus, and feebleness or absence of respiratory sounds over pleural effusions.

PROGNOSIS.—Mild atelectasis in the new-born, not dependent upon congenital defect, may be corrected if restorative measures be early applied and long continued. If of long duration, or when in adults due to extreme compression, the affection is liable to be permanent and to cause more or less emphysema and finally to give rise to lobular pneumonia or phthisis.

Atelectasis following bronchitis and whooping-cough is especially fatal. According to Loomis (Practice of Medicine, p. 158), twenty-five per cent of the total mortality in young infants results from atelectasis following bronchitis.

TREATMENT.—Having fairly established the respiratory functions at

birth by the ordinary methods of the obstetrician, it must not be forgotten in the subsequent treatment of this condition that debility is the chief factor in its production. Treatment must therefore be supporting from the first. We must also attempt to remove the secretions from the bronchi, so as to prevent implication of other air cells. With this in view, a non-depressing emetic may be given when the debility is not very great, but it is generally unsafe to repeat it. In mild cases expectorant doses of ipecac are useful. In severe cases ammonium carbonate or ammonium iodide with alcoholic stimulants are indicated. Counter-irritation of the surface by means of vigorous friction or sinapisms is useful in most cases. The diet should be nourishing, but not too concentrated. Concentrated nourishment is apt to derange the digestive organs, and do more harm than good.

PULMONARY ŒDEMA.

Pulmonary œdema consists of an interstitial extravasation of serum with effusion into the vesicular portion of the lungs, which renders the cells and bronchioles correspondingly impervious to air.

Anatomical and Pathological Characteristics.—Pulmonary œdema may occur either ante mortem or post mortem; a given case can only be settled by reference to the history, and the symptoms and signs present before death. It affects most frequently the dependent parts of the lungs, but it may involve the whole or any part of one or both. In well-marked œdema, the pleura is moist, and its cavity may contain serum. The lung does not collapse on opening the chest, and is abnormally light colored, unless the œdema is due to hyperæmia. It is heavier than normal, and pits on pressure. The serum oozing from the cut surface is frothy in proportion to its admixture with air; very slightly so if the alveoli and bronchioles are almost completely filled with serum. It has a reddish tinge if the affection is due to hyperæmia, is always albuminous, and usually contains alveolar epithelium, but unless due to hyperæmia it holds but few intra-vascular cellular elements.

Etiology.—Pulmonary œdema is probably due in every case to one of three causes, viz.: abnormal permeability of the vascular walls from changes incident to certain diseases; increase of intra-vascular pressure from active or passive hyperæmia, or change in the character of the blood; two or all of these factors may co-operate in its causation.

It is not infrequently associated with general dropsy dependent upon cardiac or renal disease. It may occur from heart failure in the course of acute general disease such as typhoid fever, or in purpura, scorbutus, anæmia, and other chronic affections.

It may occur in one lung or a part of a lung from the presence in the other parts of collapse or consolidation; and hence it often complicates pneumonia, phthisis, or pressure from tumors or pleuritic effusion.

SYMPTOMATOLOGY.—The chief symptoms are dyspnœa, increased rapidity of respiration, and cough with frothy expectoration.

The principal *signs* are very moist subcrepitant râles, with more or less dulness over the base of the lungs.

Inspection, palpation, and mensuration yield no characteristic signs. Respiration is increased in frequency.

By percussion, dulness is obtained on both sides over the most dependent portions of the lungs.

On auscultation, there is a feeble respiratory murmur, which may be slightly broncho-vesicular, with abundant moist and crackling subcrepitant râles. These sometimes resemble the crepitant râles of pneumonia, but they are more moist, not so numerous, and are usually heard in expiration as well as in inspiration. The vocal resonance may be increased.

DIAGNOSIS.—Pulmonary œdema is liable to be mistaken for the first and third stages of pneumonia, for hydrothorax, and capillary bronchitis. The distinctive signs between these diseases are as follows:

PULMONARY ŒDEMA.	PNEUMONIA, FIRST AND THIRD STAGES.
Percussion.	
Slight dulness upon both sides.	Dulness more or less marked, usually confined to one side.
Auscultation.	
Mucous and subcrepitant râles on both sides.	Crepitant and subcrepitant râles on one side.

PULMONARY ŒDEMA.	HYDROTHORAX.
Palpation.	
Vocal fremitus may or may not be increased.	Vocal fremitus absent.
Percussion.	
Moderate dulness, the upper level of which does not vary with changes in the patient's position.	Flatness, the upper line of which varies with the changes in the patient's position.
Auscultation.	
Subcrepitant râles.	Absence of the respiratory murmur and râles.

Pulmonary œdema is distinguished from *capillary bronchitis* by the history, the presence of considerable dulness on percussion, and by absence of the signs and symptoms of general bronchitis.

PROGNOSIS.—The prognosis is always grave in pulmonary œdema accompanying general dropsy. Œdema is frequently the cause of death in pneumonia. Extreme dyspnœa with bubbling râles and rapidly developing cyanosis coming on in such affections indicates a fatal termination.

TREATMENT.—The treatment of this condition will depend upon the

disease with which it is associated. If it results from Bright's disease, sudorifics and cathartics will be necessary to stimulate the other emunctories. Diuretics will also be useful in some cases, but the crippled kidneys cannot respond readily to our efforts to increase their functional activity.

If the condition is dependent upon disease of the heart, digitalis will be specially useful. If it results from debility, induced by low forms of disease, general stimulation is very essential, and diuretics and sudorifics are indicated.

If it results from pulmonary congestion, active counter-irritation by sinapisms or dry cups should be made, and diuretics, sudorifics, and cathartics should be simultaneously employed, care being taken not to exhaust the patient.

Digitalis, scoparius, potassium acetate, and ammonium acetate are the best diuretics. Jaborandi and the hot-air or vapor bath are the most suitable means to cause sweating.

Saline cathartics, and elaterium or euonymus may be employed when it is desired to act on the bowels.

When patients are greatly depressed from protracted disease, care should be taken to prevent pulmonary œdema, by frequently changing their position from the back to the sides, and *vice versa*.

PULMONARY GANGRENE.

Pulmonary gangrene is a putrefactive necrosis of lung tissue, resulting from pneumonia, septicæmia, or local injuries.

Anatomical and Pathological Characteristics. — Gangrene usually occurs at the lower part of the lung, and, according to Flint, on the posterior aspect of the upper portion of the lower lobe. It is usually confined to a few lobules, but sometimes is diffused throughout a large part or even the whole of a lobe.

A part of the lung which is entirely deprived of its blood supply undergoes coagulation necrosis. Being exposed to the action of innumerable bacteria, the devitalized tissues speedily exhibit the characteristics of moist gangrene. They become a dark brown, dirty mass, which liquefies, and appears in the expectoration as a greenish-black, extremely fetid fluid, containing organic germs, shreds of tissue, pus corpuscles, oil globules, pigment granules, and various products of chemical decomposition. Circumscribed gangrene is surrounded by a line of hyperæmic demarcation not present in the diffuse form. The discharge of the ichorous slough leaves an irregular cavity, intersected by vessels more or less occluded by thrombi. The walls, at first ragged, may granulate, and by contraction finally obliterate the space, or a chronic abscess may result. The process, at first limited, may become diffuse; in this form perforation of the pleura not infrequently occurs. From the

local thrombi in the pulmonary and bronchial vessels, metastatic septic emboli may establish secondary abscesses, in distant organs.

ETIOLOGY.—Gangrene may develop in the course of bronchitis, pneumonia, phthisis, cancer, or other pulmonary diseases, and may follow severe penetrating wounds or the entrance of foreign bodies into the larger bronchi. It may complicate pyæmia, septicæmia, or certain of the prolonged debilitating fevers.

SYMPTOMATOLOGY.—The principal symptoms are great prostration, pallor, emaciation, rapid pulse, rapid and oppressed respiration, hæmoptysis, and cough, with abundant greenish, brownish, or blackish purulent sputum of a sickening gangrenous odor, and containing fragments of the decomposing lung. The odor is not perceived in the breath constantly, but mainly after coughing.

The most prominent *signs* are: dulness on percussion, with large and small mucous râles; bronchial breathing or absence of the respiratory murmur; and, when the slough has been thrown off, gurgles and respiratory sounds indicative of a cavity. The disease at first presents the signs of consolidation, which are soon followed by breaking down of the lung tissue, and the production of vomicæ.

DIAGNOSIS.—Most of the symptoms and physical signs are not distinctive, as the same may be found in phthisis, bronchitis, or dilatation of the bronchial tubes. The diagnosis must therefore rest upon the character and the odor of the expectoration, which may be considered pathognomonic.

Small, circumscribed patches of gangrene, which occasionally occur in *bronchitis* or around tubercular deposits, cause fetid breath and fetid expectoration. The odor in these cases is only temporary, whereas in diffuse gangrene the fetor is persistent, though most marked after each act of cough and expectoration.

In bronchial dilatation or *bronchiectasis* the sputum is abundant and fetid, but not brownish in color, and the breath has not that peculiar, sickening odor of gangrene, which, once impressed on the olfactory sense, is not easily forgotten.

PROGNOSIS.—This depends largely upon the cause of the gangrene, and upon the extent of lung involved. In the diffuse form, death is inevitable, usually within a few days. In the circumscribed form, recovery may occur, but in either case there is great danger from pyæmia and sepsis. Death may result from acute hemorrhage or exhaustion.

TREATMENT.—Quinine, tincture of iron, alcoholics, and nourishing diet are the chief remedies in this affection. Inhalations of thymol, carbolic acid, creasote, eucalyptol, or turpentine may be useful in modifying the offensive odor and in limiting the amount of discharge. Anodynes should be used to soothe pain. Cases of cure are reported from external incision and drainage, conjoined with internal medication.

PULMONARY CANCER.

Pulmonary cancer is fortunately a rare disease. It is usually of the medullary variety, though scirrhus, epithelioma, and other varieties also occur.

Anatomical and Pathological Characteristics.—Cancer may occur in miliary bodies scattered throughout the entire lung, or in nodules ranging from two to ten or twelve pounds in weight; or the lung tissue may be almost supplanted by the malignant deposit.

Whether primary or secondary, single or multiple, the ultimate result of pulmonary cancer is destruction of the lung immediately involved, by pressure, atrophy, or by infiltration with the cancer cells and the products of their degeneration. Extension occurs chiefly along the lymphatic spaces. While growth proceeds at the periphery of the cancer, disorganization takes place at its centre, where a cavity is usually formed after a time. About the cancerous nodules not infrequently the lung becomes congested, inflamed, œdematous, collapsed, or emphysematous. There is always enlargement of the bronchial glands, and usually pleuritis, with extensive thickening and adhesions, and effusion of bloody serum into the pleural sac.

Etiology.—Pulmonary cancer rarely develops before the twentieth year, and more frequently affects men than women. Heredity can usually be traced. It may spring primarily from the epithelial or connective tissue of the lung, according to its type.

More frequently it is secondary to cancer in other parts, which penetrates the lungs by direct growth or by embolic cells through the circulation.

Symptomatology.—The most marked symptoms are pain and emaciation, with some dyspnœa and cough, and often bloody expectoration which resembles currant jelly.

The *signs* vary with the conditions. If only the bronchial mucous membrane is affected by the cancerous deposit, we obtain simply the signs of bronchitis. If the air vesicles are filled, we obtain the signs of pulmonary consolidation, as in pneumonia. When softening and ulceration have occurred, cavernous signs are sometimes obtained. If part of the air vesicles are filled, and others remain open, we obtain bronchovesicular respiration and other signs similar to those of phthisis.

The occurrence of the nodular variety of cancer in the lung gives rise to signs which are often distinctive. We generally notice the following:

Inspection reveals more or less loss of motion and retraction or bulging of the thoracic walls on the affected side; the former when the lung has collapsed, the latter when the growth is peculiarly large or when considerable pleuritic effusion is present.

On palpation, vocal fremitus will be feeble or suppressed, according to the proximity of the tumor to the chest walls.

Percussion, most frequently near the middle or the upper part of the chest, will show dulness or flatness over the tumor, according to its nearness to the chest walls. In many instances, over one or more places resonance remains normal, surrounded by areas of flatness, owing to the presence of a small portion of healthy lung surrounded by a cancerous mass.

On auscultation, the respiratory sounds may be feeble or entirely suppressed over the tumor. Occasionally the cancer rests upon a large bronchial tube, in such a position that the sounds from the latter are transmitted to the surface, giving rise to bronchial breathing and bronchophony.

If the pleura is involved, there will be an exudation of serum into its cavity, yielding the signs of chronic or of subacute pleurisy. Upon exploratory aspiration, the fluid is often found more or less sanguinolent.

DIAGNOSIS.—When the disease is primary, it is very difficult to detect. When secondary to cancer in other portions of the body, the occurrence and persistence of bronchial or other pulmonary signs should lead us to suspect its true nature.

Pulmonary cancer is most likely to be mistaken for chronic or subacute pleurisy with effusion. It bears some resemblance to phthisis, and also to aortic aneurism.

If the cancer is attended with effusions into the pleural sac, an accurate diagnosis cannot be made by the ordinary methods, but the character of the fluid obtained by aspiration will usually enable us to make a correct diagnosis.

The differential points between the nodular variety of pulmonary cancer and *chronic pleurisy* will be seen in the following table:

PULMONARY CANCER.	CHRONIC PLEURISY.
Symptoms.	
Nearly constant pain, and often currant-jelly expectoration.	Little, if any, pain; the expectoration, if any, only purulent.
Percussion.	
Dulness does not begin at the base of the lung; usually one or more isolated spots of resonance within the area of dulness or flatness.	Flatness beginning at the base of the lung, uniform to its upper limit.
Auscultation.	
Usually some respiratory signs, due to isolated portions of normal lung, or to only partial consolidation of the pulmonary parenchyma.	Absence of the respiratory murmur, and usually of the bronchial sounds; the latter when heard are diffused and distant.
Aspiration.	
Sometimes a sanguinolent fluid. The fluid, when serous, coagulates much more slowly than in pleurisy.	Serous or purulent fluid is obtained.

Cancer of the lung is not likely to be mistaken for *phthisis*, though such an error might be made. The cancerous growth does not often begin in the apex of the lung, and it may become very extensive without causing bronchial râles. The reverse is true in phthisis.

The history of *aortic aneurism* is different, as intra-thoracic cancer is nearly always secondary to external manifestations. The symptoms due to pressure, viz., pain, dyspnœa, dysphagia, and venous congestion and pulsation, are less persistent in aneurism than in cancer.

Aneurisms usually have a distinct expansile pulsation, and when they cause a murmur, it is likely to be double, that is, systolic and diastolic. Cancers have no pulsation excepting that communicated from the aorta, and this is feeble and simply lifting. If a cancerous growth, by pressure on the artery, causes a murmur, it is always systolic, no second sound being produced.

Prognosis.—The prognosis is always hopeless. Death usually results within a year.

Treatment.—Anodynes to relieve pain are the only remedies that can be recommended. None of the remedies which have, from time to time, been recommended for the cure of cancer have borne the test of experience.

PULMONARY TUMORS.

Tumors or morbid growths in the lungs may result from hydatids, syphilis, enlargement of glands, abscesses, and malignant disease.

HYDATID CYSTS OF THE LUNGS.

Hydatid cysts in the lungs constitute a rare affection, which presents symptoms and signs similar to those of phthisis. The cyst most frequently occupies the lower lobe of the right lung, and is generally secondary to hydatids of the liver.

Anatomical and Pathological Characteristics.—The wall of a hydatid cyst is composed of an outer and an inner layer, and the cyst contains a clear fluid non-coagulable by heat or acid. From the inner membrane develop young echinococci with characteristic hooklets; these cysts may in turn develop within themselves others of similar form.

The growth after attaining a variable size may by fatty degeneration of its contents undergo evolution and largely disappear, or it may remain permanently as the seat of calcification. Suppuration may occur within the cyst, and its subsequent course may be that of an abscess. Again, by gradual increase in size, it may produce great disturbance by its pressure, by exciting inflammation, or by rupture into the surrounding lung or pleural cavity.

Etiology.—The ova of the tænia echinococcus, which commonly inhabits the intestinal tract of dogs and other animals, upon entering the

human stomach are freed from their capsules by the digestive fluids. Thence the parasites burrow to the viscera, chiefly the liver, and become hydatid cysts. The disease is rare in this country, and is seldom found excepting among people who mingle freely with the lower animals.

SYMPTOMATOLOGY.—The symptoms are like those of phthisis, viz., emaciation, night-sweats, cough, dyspnœa, and expectoration of bloody and purulent sputa. Finally, hydatid cysts, or portions of them, and the hooklets of the echinococci may be thrown off through the bronchi.

Symptoms of pyrexia are due to the secondary inflammation, not to any specific action.

The principal *signs*, if the tumor be large, are: bulging and loss of motion of the side, nodular prominences in the intercostal spaces; and, when the cysts approach the surface of the lung, dulness or flatness on percussion, with suppressed respiration or tubular breathing. A positive diagnosis can seldom be made until the hooklets of the echinococcus are discovered in the sputum. This does not occur until late in the disease, when, after death of the *entozoon*, it begins to be ejected from the body.

According to Bird, the diagnosis may be made with a fair degree of certainty early in the disease if the cyst is of any considerable size and impinges against the chest wall. In such cases the following signs have been noticed:

Inspection reveals decubitus always on the sound side. The respiratory movements of the affected side are deficient, and there may be slight bulging in one or more places along the intercostal spaces, over the cysts.

Vocal fremitus may be absent, and fluctuation can sometimes be detected over the cyst by palpation.

On percussion, flatness is found over a limited area corresponding to the cyst. In order to be of value in diagnosis, this area of flatness should not be less than three or four inches in diameter. It should have a rounded outline, and it must be clearly separated by a line of demarcation from the surrounding resonance. It does not change with the position of the patient.

In auscultation there is absence of the respiratory murmur over the area of flatness, and normal respiration around it, immediately beyond the line of demarcation. The compressed lung close about the cyst may cause a more or less tubular sound.

DIAGNOSIS.—The affection is liable to be mistaken for phthisis or circumscribed pleurisy. Attention to the differential characters noted in the following table will aid in making the diagnosis:

HYDATID CYSTS OF THE LUNGS.	PHTHISIS.
Inspection.	
Prominence of the intercostal spaces.	No prominence of the intercostal spaces.

Hydatid cysts of the lungs.	Phthisis.
Palpation.	
Absence of fremitus, and perhaps fluctuation over the cyst.	Exaggerated vocal fremitus; no fluctuation over the consolidated lung.
Percussion.	
Flatness over the cyst sharply defined by a line of demarcation from the resonance of the surrounding healthy structure.	Dulness over consolidated lung, gradually fading off into normal resonance.
Microscopic.	
No tubercle bacilli in simple cases.	Tubercle bacilli commonly present in the sputum.
Auscultation.	
Absence of respiratory murmur over cyst (flat area).	Broncho-vesicular respiration, or cavernous signs over dull area.

The distinctive features between hydatid cysts of the lungs and circumscribed pleurisy are as follows:

Hydatid cysts of the lungs.	Circumscribed pleurisy.
History.	
Usually located in the infra-clavicular or axillary regions.	Usually located at the base of the chest.
Symptoms and Signs.	
Gradual accession of the local and constitutional symptoms.	Usually ushered in with acute febrile symptoms.
Inspection.	
Nodular prominence of intercostal spaces.	Uniform prominence of intercostal spaces.
Percussion and Auscultation.	
Signs usually in the upper part of the chest.	Signs generally in the lower part of the chest.

Treatment.—As the disease can seldom be distinguished from phthisis, the treatment must generally be the same as for the latter. In those cases where the disease can be positively diagnosticated, aspiration of the cyst and injection with iodine (Form. 11) is the most rational treatment.

DISTOMA PULMONALE.

The people in some parts of China, Corea, and Japan, by the use of surface or ditch water in the preparation of uncooked food, and for drinking purposes, are liable to a peculiar form of pulmonary disease due to entrance into the lung of the distoma pulmonale, which infests these waters. It is an animal parasite somewhat resembling an ordinary leech in miniature, being eight or ten millimetres long, with oval and ventral suckers by which it effects locomotion.

By burrowing in the walls of the bronchi it causes sacular bronchiectatic cavities, surrounded by irregular zones of congestion and induration and containing débris, mucus, and the parasites with their ova.

SYMPTOMATOLOGY.—The *symptoms* and *signs* are those of chronic bronchitis of increasing severity associated with frequent, and often severe hemorrhages.

The presence of the characteristic organism in the expectoration, the history of the case, and the geographical locality of its occurrence establish *the diagnosis*.

Some patients recover with or without treatment, but the affection is of long duration and no specific medication avails. Prophylaxis is the most important part of treatment (Annual of Universal Medical Sciences, 1888).

SYPHILITIC DISEASE OF THE LUNGS.

It is a well-recognized fact that syphilis causes a morbid condition of the lungs, the signs of which in no way differ from those of ordinary phthisis. Cases are occasionally observed in which a specific form of bronchitis or gummata occurs as a result of the venereal taint.

The signs of syphilitic bronchitis are the same as those of the non-specific affection. A distinction between the two can only be made by attention to the history and the attendant symptoms.

DIAGNOSIS.—The differential diagnosis between syphilitic disease of the pulmonary parenchyma and phthisis is extremely difficult, and often impossible. But when uncomplicated, pulmonary syphilis usually differs from phthisis, as shown in the following table:

SYPHILITIC DISEASE OF THE LUNGS.	PHTHISIS.
History and Symptoms.	
The history of syphilis; thickening of the periosteum and perichondrium over the inner end of the clavicles, and one or more of the cartilages of the upper ribs, with sub-sternal tenderness on pressure over the upper part of the sternum. Usually neither fever nor decided emaciation, and no hæmoptysis.	No history of syphilis; no thickening of the periosteum or perichondrium over the clavicles or cartilages of the upper ribs, and no sub-sternal tenderness. Hectic fever and marked emaciation always present, with usually hæmoptysis.
Physical Signs.	
Dulness over the nodules, usually confined to one lung, and found at its base or at the lower part of the upper lobe. The dulness remaining circumscribed for a long time. Viscid subcrepitant râles, or several mucous clicks, diffused over a considerable portion of the lung, are believed to be one of the earliest indications of the syphilitic affection; later the auscultatory signs are the same as those of phthisis.	Dulness usually at the apex, and gradually extending over the surrounding lung.

PROGNOSIS.—The prognosis is favorable in uncomplicated cases when discovered early.

TREATMENT.—Anti-syphilitic constitutional remedies as iodine, potassium iodide, and the compounds of mercury are indicated. If these were oftener tried in cases of so-called phthisis, probably more would be cured. We should also employ tonic and supporting measures, similar to those recommended in pulmonary phthisis.

ENLARGED BRONCHIAL GLANDS.

As an independent affection, this is of rare occurrence. It deserves attention here from its close resemblance in some particulars to phthisis.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—The chief bronchial glands lie at the bifurcation of the trachea and about the two main bronchi, where they are numerous and in relation in front with the aorta, pulmonary artery, and pericardium; behind with the aorta, œsophagus, vena azygos, and sympathetic plexus. Those about the bronchi are also adjacent to the large venous and arterial branches and pneumogastric and recurrent laryngeal nerves.

Enlargement of these glands occurs from engorgement and increase of interstitial connective tissue with thickening of the capsule. When acute, suppuration may occur.

ETIOLOGY.—Some enlargement of the bronchial glands usually accompanies inflammation of the lung or bronchitis; it is marked in phthisis, syphilis, and malignant disease of these organs. It also occurs to some extent in typhoid fever, measles, whooping-cough, and other infectious diseases.

SYMPTOMATOLOGY.—The prominent symptoms are: a dry, ringing, and paroxysmal cough like that of pertussis but without the whoop; with dyspnœa, and more or less pain and tenderness on pressure near the fourth or the fifth vertebra, associated with emaciation, hectic flush, and night-sweats.

The symptoms vary greatly according to the size and position of the enlargement. Compression of the bronchi and lungs gives rise to cough, expectoration, and dyspnœa.

Pressure upon the recurrent laryngeal nerve produces dyspnœa, occasionally of a spasmodic type, and may also cause hoarseness or aphonia.

Crowding of the tumor upon the œsophagus produces dysphagia; pain and tenderness result from implication of the sympathetic plexus. Compression of the pneumogastric accounts for the palpitation, rapid pulse, and the nausea and vomiting that sometimes occur.

On inspection, we find as *signs* frequently, distention of the cervical veins and sometimes cyanosis, rarely deficiency or absence of respiratory movements of one side due to occlusion of the main bronchus.

By palpation and percussion, tenderness may usually be detected over

the bronchial glands in the interscapular region near the fourth and fifth dorsal vertebræ. Circumscribed dulness over the enlarged glands is sometimes found. Compression of a bronchus may cause collapse of the lung, with consequent uniform dulness.

By auscultation, we usually hear numerous râles and feeble or harsh respiration, or in other words the signs of consumption. Sometimes arterial murmurs may be detected. Again, pressure on a bronchus may cause localized râles and feeble respiration; or it may prevent respiratory sounds in the portion of lung supplied by that bronchus. In these cases a deep breath will frequently bring out the respiratory sound, where it could not be heard in ordinary respiration.

DIAGNOSIS.—Enlargement of the bronchial glands cannot usually be distinguished from phthisis, but in some instances a reasonably certain differentiation can be made by remembering that the disease under consideration usually occurs at an earlier age than phthisis, and that the pain, tenderness, and dulness which it induces are first found in the region of the bronchial glands, instead of over the apex of one lung.

PROGNOSIS.—The prognosis must be based upon the evidences of the structures involved, the size of the enlargement, and its rate of growth. A simple inflammatory enlargement may be arrested, but if terminating in suppuration it is frequently fatal. Syphilitic adenitis rapidly yields to appropriate remedies. Malignant disease in this locality is always fatal.

Tuberculosis of these glands is likewise unfavorable.

TREATMENT.—Treatment is usually of little avail in this disease, but the remedies which are most beneficial in scrofulous enlargement of the superficial glands should be tried. Iodine, potassium iodide, calcium chloride and cod-liver oil may be used, with quinine to relieve fever, or iron when fever is not present.

The diet should be plain but nutritious, and all the surroundings of the patient should be made as healthful as possible.

PERTUSSIS, OR WHOOPING-COUGH.

Pertussis is an infectious, contagious disease, often epidemic, and characterized by paroxysmal, spasmodic cough terminating in a prolonged inspiratory crowing or whooping sound. It is most common in children under ten years of age; it is rare before the third month; it seldom affects adults but is occasionally observed even in advanced life. One attack usually gives immunity from later ones.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—The only morbid condition, found in fatal cases of pertussis, which is due to the disease specifically, is a more or less marked catarrhal inflammation of the upper air passages, larynx, trachea, and large bronchi. Other pathological conditions present are secondary and due largely to the severity of

the cough. Pulmonary vesicular emphysema is commonly present, and sometimes bronchiectasis, chiefly in the upper lobes. Pneumonia and atelectasis are not infrequent complications. There may be congestion of the meninges and apoplectic extravasation into the brain, associated with effusion of serum into the cerebral cavities. Prolapsus ani and hernia are occasionally observed as results of the cough, and more rarely, rupture of the membrana tympani.

ETIOLOGY.—It is highly contagious and is said to affect even the lower animals. Infection is usually conveyed directly from one person to another, though a third person may be the medium of communication. Recent evidence favors the germ theory of its production, but as yet no one micro-organism has been discovered as the sole cause.

A stage of incubation of from two to fourteen days precedes the appearance of catarrhal symptoms.

SYMPTOMATOLOGY.—The disease is conveniently divided into a catarrhal, a paroxysmal, and a declining stage. Sneezing, coryza, epiphora, and some cough characterize the first period, which commonly lasts from one to two weeks, and in no way differs from an ordinary cold.

The more severe the affection, the shorter the first stage. In the second period, the cough becomes a series of short expiratory efforts ending in a prolonged inspiration with a stridulous whooping sound caused by spasmodic contraction of the glottis.

Generally several of these series occur in succession, terminating with the expectoration of a small amount of viscid secretion, and with some of a frothy nature, and often vomiting of a large amount of thick, glairy mucus. These paroxysms last from half a minute to a minute or longer, and recur during the height of the attack, every two or three hours, or sometimes three or four times an hour. The longer the intervals, the more severe the paroxysms. They are more frequent at night.

Conjunctival hemorrhage, œdema of the eyelids, and epistaxis are frequently caused by the venous congestion which occurs during the cough. In some cases there is marked cyanosis, followed by great exhaustion. Three or four weeks is the average duration of the second stage. In mild cases the characteristic cough may be entirely absent. In some cases it may persist as a habit for many months even after convalescence. The symptoms of the third stage are those of a declining catarrhal inflammation of the air passages, which usually lasts about two weeks.

DIAGNOSIS.—The diagnosis rests upon the history, the peculiar character of the cough, and the expectoration or vomiting of large quantities of viscid mucus. Affections of the bronchial mucous membrane, or of the pulmonary parenchyma, which are frequently developed during the course of pertussis, yield the same signs as when they occur independently.

PROGNOSIS.—Whooping-cough is a serious disease among infants. The

prognosis improves with increasing age, and larger children seldom succumb to the affection, excepting when it is complicated by other disease. The indications are good if the patient is fairly well between the paroxysms, but evidence of illness is significant of some complication. Intercurrent attacks of measles or other diseases are unfavorable. Bronchitis and broncho-pneumonia, especially the latter, frequently cause a fatal termination. Cerebral congestion, apoplexy and convulsions, or more rarely, hemorrhage from a mucous surface may be the cause of death.

The patient may die from emaciation and exhaustion due to frequent vomiting. The affection is frequently preceded or followed by measles.

TREATMENT.—Many "specifics" have been recommended for this disease, but none have proved effectual.

Morphine and chloral may be given in doses suited to the age of the patient, especially to adults (Form. 2). For children I like better potassium and ammonium bromide or hydrobromic acid with syrup of lactucarium, with or without syrup of hydriodic acid.

Sulphate of quinine in large doses, given in solution so as to make the strongest possible impression on the sense of taste, has been highly recommended, and, according to reports in the current medical literature, it will cure the majority of cases in a few days; but my own experience with it has been unsatisfactory.

My experience with the preparations of anemone pratensis, thymus vulgaris and œnothera biennis has been very limited, but never satisfactory. Antipyrine in doses of gr. ij. every three to five hours for a child twelve years of age, to be discontinued as soon as any cyanosis appears, is highly recommended by many; and bromoform in doses of ♏ ss.–i. for a child of the same age, has been extolled by others.

CHAPTER X.

PULMONARY DISEASES.—*Continued.*

PULMONARY PHTHISIS.

UNDER pulmonary phthisis may be grouped several affections, differing somewhat in their anatomical characteristics, but closely resembling each other in their physical signs. From this latter fact, it is especially appropriate, in the matter of diagnosis, to consider them together. The term phthisis will then include all those wasting pulmonary affections which are attended with exudation or infiltration into the pulmonary parenchyma, causing consolidation, and are attended or followed by more or less induration and contraction and subsequent breaking down of lung tissue, whether these diseases be the result of a simple inflammatory affection, or the cause or the result of tubercular infiltration. The term pulmonary phthisis will therefore include fibroid phthisis and the ordinary acute and chronic forms of pulmonary tuberculosis. Any special symptoms or signs which are of value in differentiating between these various conditions will be separately considered.

Fibroid phthisis is also known as cirrhosis, induration, or fibroid degeneration of the lung; sometimes as chronic catarrhal pneumonia, and occasionally as bronchiectasis.

The ordinary forms of phthisis have various names, as, chronic croupous pneumonia, caseous pneumonia, cheesy or tuberculous infiltration of the lung, chronic tuberculosis, and pneumonic phthisis.

PULMONARY TUBERCULOSIS.

Pulmonary tuberculosis may be more or less acute or chronic; running its course within a period of six months or a year, or being prolonged in exceptional cases for many years. The term acute tubercular phthisis is properly applied to miliary tuberculosis of the lung as a part of a generally disseminated disease.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—Upon post-mortem examination usually both lungs are found to be affected. A lung which is the seat of ordinary tuberculosis may appear superficially normal or mottled, with grayish-yellow areas over which minute tubercles may be seen in the pleura. This membrane may also be covered with an inflammatory exudate. The organ is heavier, more solid, and less crepitant than normal. Section usually reveals at the apex one or more ragged cavities, and yellow, cheesy masses, some of which may be semi-

fluid. About these are miliary foci of caseation, a line in diameter, sharply defined to the naked eye, rounded, firm, translucent, and gray or yellowish in color. Throughout the rest of the affected lobe or the entire organ may be scattered miliary tubercles, and larger areas the size of a pea, more yellow in color. There is accompanying bronchitis, and from the severed tubes, some of which are dilated, pus may be pressed. The non-tubercular parts of the lung may be the seat of emphysema or congestion and œdema, and the bronchial glands are infiltrated and enlarged.

In acute tuberculosis, tubercle bacilli commonly find lodgment on the mucous membrane of the bronchioles or alveoli, having entered the bronchi with the inspired air, or occasionally by rupture into the

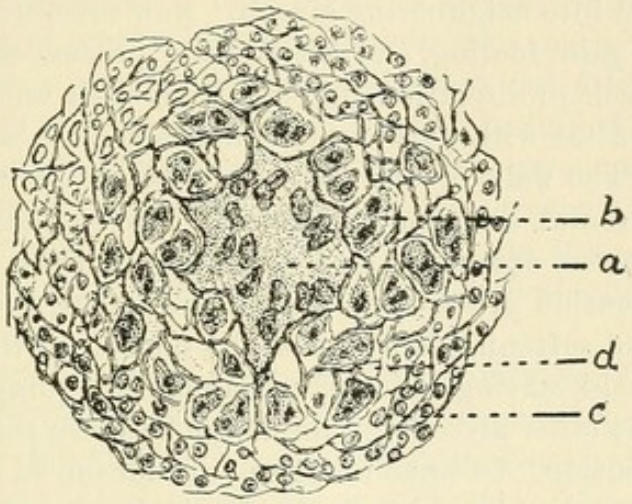

FIG. 28.—TUBERCLE. *a*, Giant cell; *b*, epithelioid cells; *c*, round lymphoid cells; *d*, fibrous reticulum.

passages of a tubercular gland. They may, however, reach the lung through the circulation by one or more emboli from a distant tubercular involvement of a vein or the thoracic duct. Whether they primarily gain footing on the epithelium of the air passages or on the endothelium within the vessels, under favoring conditions they effect the formation of a tubercle.

The tubercle has no constant form, but consists of one or more multinuclear giant cells, surrounded by an aggregation of smaller epithelioid cells, about which is a zone of round lymphoid cells the size of leucocytes and smaller than epithelioid cells. Between these, and continuous with the irregular processes of the giant cells, is a fibrous reticulum more or less prominent.

Tubercle bacilli are present in and about these elements.

Epithelioid and giant cells, though not peculiar to the tubercle, are more frequently found in it than elsewhere.

The many oval nuclei of the giant cells are arranged at its circumference or at opposite poles. The epithelioid cells may have one or two nuclei; the lymphoid cells, which are smaller than the epithelioid, have each a single relatively large nucleus. A prominent feature of the

tubercle is its non-vascularity, with a tendency to undergo early coagulation necrosis, with coalescence of its cells into a homogeneous, firm, gray mass, which later becomes softer, cheesy, and yellow.

This caseation invariably begins at the centre of the nodule, and is probably the result of the lack of nourishment and the specific action of the bacilli. This tubercle formation is the same when occurring in the lungs as elsewhere; its subsequent course is, however, very different and varies in these organs according to the mode of infection, the resistance of the tissues, the number of bacilli and possibly their virulence. From the primary focus, the migrating leucocytes and round cells carry the tubercle bacilli into the surrounding intercellular and perivascular lymph spaces and into neighboring alveoli. New tubercle develops wherever the germs gain footing, and, either as a process of inflammatory exudation or of cell proliferation starting from their walls, the adjacent air cells become filled with fibrin and cellular elements bearing the noxious principle. The walls of the alveoli and associated bronchi become infiltrated with round cells and thickened. The capillary plexus is destroyed as the process extends and the tubercles coalesce, forming larger foci. While extension proceeds at the circumference, the centre undergoes caseation and softening, and eventually may be partially discharged through the bronchi, leaving an irregular, rapidly sloughing cavity behind. By aspiration into other alveoli this discharge becomes the means of further lobular extension. In some instances, in addition to these evidences of acute inflammation, breaking down of the lung, and wide dissemination of caseous foci, and more or less extensive fibroid thickening or cirrhosis of the peribronchial and interlobular tissues will be observed. Such are cases either of chronic inflammation of the lung upon which tuberculosis has supervened, or of primary pulmonary tuberculosis in which the partially successful efforts of nature to limit the disease have resulted in connective-tissue hyperplasia.

Etiology.—The predisposing causes of the disease are those influences which depreciate the general health of the individual or which, by diminishing local tissue resistance, afford fitting soil for growth of the bacilli. Though the essential cause, the tubercle bacillus, is probably rarely transmitted from mother to child, it is reasonable to suppose that the weakness of constitution which tuberculosis engenders in the parent may be inherited by the offspring. In so far, the latter is a more suitable field for infection. As reported by James T. Whittaker, of Cincinnati, observations by Csokor (*Deutsche Medizinal-Zeitung*, Berlin, Jan., 1892) and F. V. Birch-Hirschfeld (*Deutsche medicinische Wochenschrift*, Leipzig, March, 1892) seem to prove that the bacilli may be transmitted directly from the mother to the fœtus. Children of those who are debilitated by other diseases, by vicious habits, or by age receive a similar heritage. The predisposition to tuberculosis may also be acquired by those who are habitually subjected to improper hygienic influences.

Poor or insufficient food, scanty clothing, want of cleanliness, impure or damp and chilly air, and lack of sunshine, variously combined, may reduce the most robust constitution to a condition as favorable to phthisis as is the inherited, so-called scrofulous diathesis. Prolonged lactation, frequent child-bearing, alcoholism, and chronic malaria, by enfeebling the constitution, also prepare the way for tubercular infection. Bronchitis, pneumonia, and other pulmonary affections frequently prepare the soil locally for the growth of the specific germ.

It is now generally conceded that the ultimate cause of tuberculosis is the tubercle bacillus, as first determined by Koch in 1882. This is a slender rod varying in length from one-quarter to one-half the diameter of a red blood-corpuscle; it is straight or curved, occurring singly, in chains, or in groups, and is incapable of voluntary motion. When properly stained, it has a peculiar beaded appearance, and if highly magnified, small bright spots may be seen within the rod, having the appearance of spores. The bacilli are relatively enduring, but grow outside the body only under the most careful regulation of temperature, nutrient media, and other conditions. Tubercle bacilli enter the lung chiefly through the air passages, conveyed by particles of dried phthisical sputum or dust.

Entrance may take place through the circulation from a primary focus elsewhere. Such a focus may in rare instances be established by the ingestion of tuberculous meat or of milk from a diseased animal. Chickens that are allowed to eat the sputum from tuberculous patients often contract the disease and may become a source of infection. There can be no doubt that in a small percentage of cases the disease is contracted by direct contagion, as in case of those who have nursed consumptives long and closely. However, notwithstanding the vast multitudes who yearly die of consumption, very few well-authenticated cases of direct contagion, or infection from ingestion of tuberculous substances, can be adduced. The investigations of Henry P. Loomis, of New York (Researches of the Loomis Laboratory, No. 1, p. 75), show that forty per cent of the bodies of persons dying suddenly in general good health, apparently perfectly free from tuberculosis, have the bacilli in the bronchial glands. Therefore, while it may be admitted that Koch's bacillus is the ultimate cause of the disease, it appears impotent excepting in the presence of a favorable soil as furnished by those of depraved constitution.

SYMPTOMATOLOGY.—The chief symptoms of ordinary pulmonary tuberculosis are only too well known, even by the laity. Few there are who have not noticed among their immediate friends the bright and suffused eye, hacking cough, progressive emaciation, hæmoptysis or purulent sputum, the hectic flush, and the night-sweats of this dread disease.

The affection often comes on insidiously, with a slight hacking cough, which does not attract attention till the patient takes a severe cold, or is

taken down with some acute disease from which he does not convalesce at the proper time; he is then discovered to have symptoms of consumption. Sometimes, however, there may have been no hacking cough in the beginning; we are often told that the disease started with a severe cold, whooping-cough, measles, influenza, typhoid fever, intermittent fever, parturition, or chronic affection of the throat or bronchial tubes. In quite a large percentage of cases the patient has been apparently in perfect health until hæmoptysis has occurred; from this he may have perfectly recovered, but not infrequently the symptoms of a grave disease have steadily progressed. Often there is a history of prolonged overwork and exhaustion culminating in fever, supposed to be malarious or typhoid, during which the evidences of pulmonary disease are discovered. In most instances loss of weight occurs early in the affection, depending generally upon loss of appetite or imperfect digestion. Daily fever of two or three degrees is common, and a nearly uniform symptom is rapidity of the pulse; even while other symptoms may not be pronounced, the pulse frequently runs from one hundred to one hundred and thirty per minute. The cough is at first hacking, with little or no expectoration; subsequently the sputum may become mucous and later muco-purulent. Hæmoptysis occurs in a considerable number of cases, but not in all; in many, early in the attack; in others, not until the close of the disease. A simple streaking of the sputum with blood should not be considered as evidence of tuberculosis. In many cases these symptoms gradually increase for six or eight weeks, and then slowly subside until the disease is arrested, and it may not again become active; but in the majority who are less fortunate, as the disease progresses there are only periods of comparative health between the attacks of great depression, and each of these latter is likely to leave the patient weaker than when it began, so that he grows worse, although at times, not only the patient, but his friends are encouraged to believe that he is improving.

Disorders of the digestive tract are prominent accompaniments of the pulmonary trouble. Anorexia, commonly an early symptom, may be associated with nausea and vomiting; the latter may be due to the severity of the cough. Gastric pains, which are often present, may be reflex or may be dependent upon an inflamed condition of the mucous membrane of the stomach. Diarrhœa is frequently very troublesome in advanced cases, and is not uncommon at any period of the disease. Rapid emaciation, proportioned to the acuteness of the affection, is a natural consequent of continued fever and anorexia, and attendant malnutrition may be aggravated by hæmoptysis or a chronic colliquative diarrhœa. In many instances tubercular patients are hopeful to the end, though this is less common than is generally supposed. In the later stages of the disease, cerebral anæmia or possibly tubercular changes in the brain itself, or the sympathetic effects of imperfect digestion affect the mental condition, causing irritability, fretfulness, cerebral

fatigue upon mental exertion, and finally, in some cases, hallucinations or fixed delirium; though commonly the mind remains clear to the last.

The *signs* differ in various stages of the affection, the most important being: diminished movement and sinking in of the chest walls in the infra-clavicular region, with dulness on percussion; and at an early stage, feeble respiration, or subcrepitant râles confined to one apex, followed by broncho-vesicular respiration, exaggerated vocal resonance, metallic râles, and the signs of cavities.

Phthisis is generally described as having three stages, but these run imperceptibly into each other, so that the signs of two or of all of them are likely to be combined at one time in the same individual. The stages, therefore, cannot be sharply delineated, and I think an attempt to describe the signs of each separately would only lead to confusion.

The stages of phthisis consist of: first, the incipient stage; second, the stage of more complete deposition, occasioning consolidation and retraction; and third, the stage of softening with breaking down of lung tissue and the formation of cavities. The pulmonary lesions occur with about equal frequency on the right and on the left side of the chest, and almost always they are to be found at the apex of the lung.

Inspection and mensuration yield no signs in the early stage of this disease, except increased rapidity of the respiratory movements. After a few weeks, in the second stage, in addition to the rapid respirations, we observe more or less loss of motion, with sinking in of the chest wall over the affected organ, especially during deep inspiration. In the last stage of the disease, there is marked emaciation, with prominence of the clavicles due to the sinking in of the tissues above and below them; loss of motion becomes more distinct, and there is depression of the chest walls, usually in the infra-clavicular region.

Exceptional.—In exceptional cases, cavities may exist in the apices of the lungs without any considerable depression of the chest walls or diminution in their movements.

Early, palpation furnishes no signs. As soon as any considerable amount of consolidation has taken place, the vocal fremitus is apt to be increased, but this sign is variable, and therefore unreliable. Sometimes gurgling fremitus is detected over superficial cavities.

Exceptional.—Shrinking of the affected lung may drag the heart a short distance from its normal position, as indicated by the site of its apex beat. The formation of a large cavity occasionally causes bulging of the portion of the chest which was formerly depressed.

On percussion in the *first stage* of this disease, there is slight dulness if the superficial portions of the lung be affected; but if only the deeper structures are involved, this sign may be absent.

Dulness, when slight, is best obtained with the patient's mouth open, and the difference in the resonance of the two sides can be most easily recognized at the end of a full inspiration.

The late H. A. Johnson, of Chicago, told me that he sometimes obtained excellent results, in obscure cases, by listening with the ordinary binaural stethoscope, the chest piece of which was held by the patient about two inches in front of his open mouth while percussion was being made on the chest.

In this connection, it must be constantly borne in mind that moderate dulness is frequently a normal sign over the right apex, and that other diseases than phthisis, as, for example, bronchitis and circumscribed pneumonia, not infrequently cause temporary dulness in the infra-clavicular region.

Dulness over the left apex, even though slight, is always abnormal, and, when persistent, is nearly always a sign of phthisis. Marked dulness, if persistent, has the same significance when found over the right apex. This sign is sometimes found behind when it cannot be detected in front. It is frequently present in the supra-clavicular or clavicular region when it cannot be obtained below the clavicle.

Exceptional.—In the first stage of phthisis the resonance is sometimes vesiculo-tympanitic, on account of secondary circumscribed emphysema.

Consolidation of the deeper portions of the lung may cause no dulness upon ordinary percussion if healthy lung tissue intervene between it and the surface. In forcible percussion a small amount of consolidation at the surface of the lung may be overlooked in consequence of the intense resonance from the deeper tissues.

It should be remembered, in estimating the amount of phthisical consolidation, that the degree of dulness and its area may be due to the temporary consolidation of circumscribed pneumonia. The extent of phthisical consolidation in such cases can only be ascertained after the inflammatory product has been absorbed.

In the *second stage* of phthisis, dulness becomes very marked, and gradually extends over a wider area, owing to progressive pulmonary consolidation; up to this time, dulness is almost universally confined to one side. At the same time, tubular—or, according to Flint, tympanitic—resonance may be caused by the bronchial tubes or the trachea, especially when percussion is made near the borders of the upper part of the sternum.

Exceptional.—In this, as in the first stage, vesiculo-tympanitic resonance may be obtained in rare instances.

In the *third stage*, dulness is obtained over the affected lung, unless cavities of considerable size exist near the surface. In this case, resonance over a limited portion, surrounded by dulness and corresponding to the cavity, may be tympanitic, amphoric, or cracked-pot in character. Sometimes early in the morning, dulness or flatness may be obtained over a cavity, owing to its being filled with secretions, which will give place, after free expectoration, to the signs of a vomica. In this stage, or in the latter part of the second stage, dulness nearly always

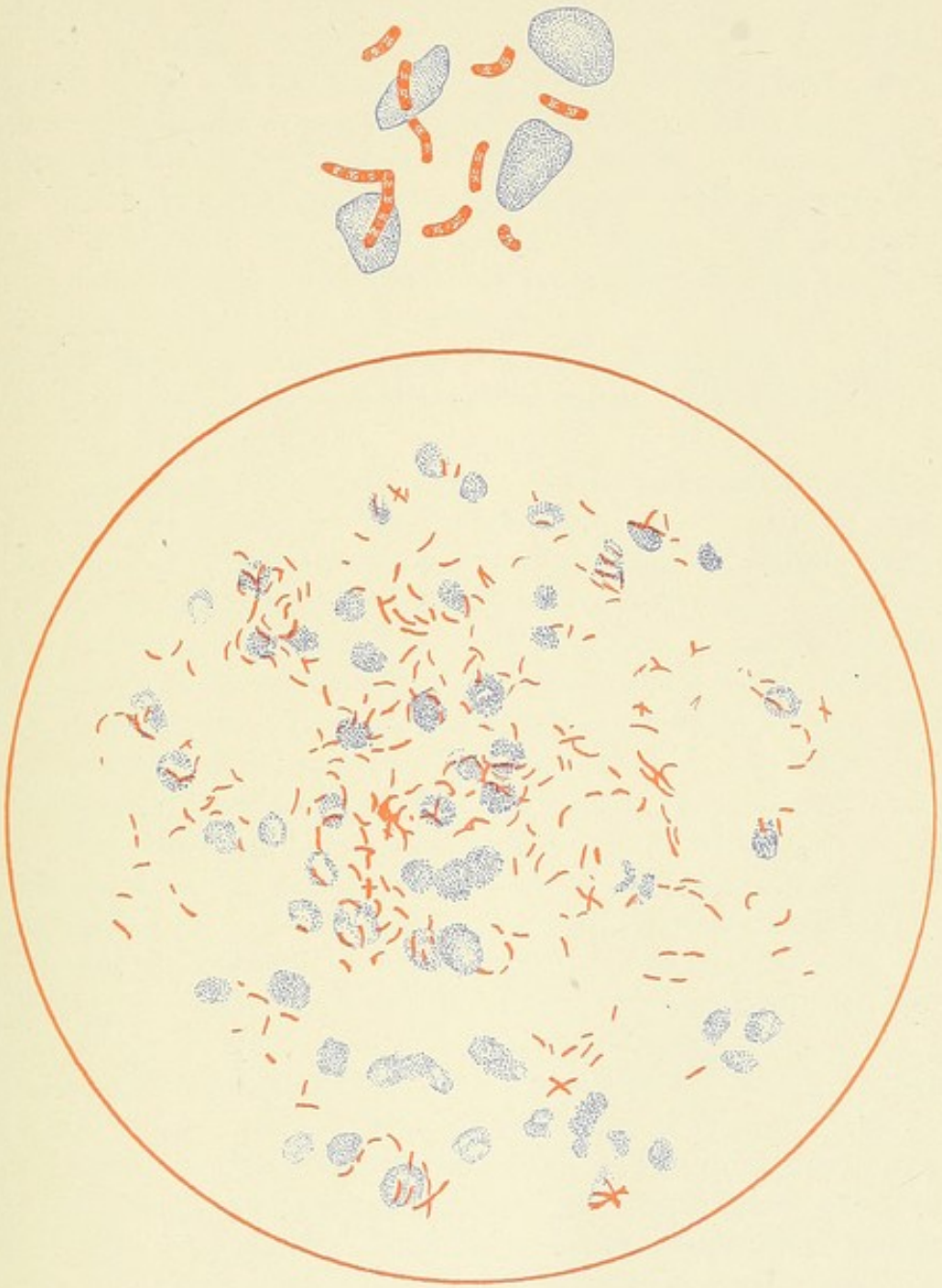

FIG. 29.—TUBERCLE BACILLI IN SPUTUM.

The bacilli, stained red, in the larger plate, are magnified about 1,000 diameters; those in the smaller plate about 3,500 diameters.

appears at the apex of the opposite lung, where it can be detected by comparing the resonance over the diseased structure with that below the second or third rib.

Among the early signs of this disease to be detected by auscultation are feeble or cog-wheel respiration, with subcrepitant râles, limited to a small portion of the apex of one lung. Occasionally the mucous click or a few crepitant or sibilant râles, or crumpling or friction sounds, may be heard in the same locality. Broncho-vesicular respiration is obtained a little later. The heart-sounds are heard with abnormal intensity over the affected lung; if the consolidation be upon the right side, the first sound of the heart will be most distinct; if upon the left, the second sound is more intense than the first.

In the first stage, the exaggerated bronchial whisper is a sign of considerable value, and exaggerated vocal resonance can usually be obtained.

At a later period, in the second stage, broncho-vesicular respiration becomes distinct, the respiratory sounds are harsh and tubular in quality, and the expiratory murmur is prolonged and high-pitched. There are also large and small, moist, crackling, or metallic râles, which are often sticky in character, and not affected by coughing. Friction sounds are often present, due to circumscribed pleuritis, caused by the tubercular deposit in the pleura. In a few cases, subcrepitant or sibilant, and occasionally sonorous, râles may still be heard in the second stage, limited to a small space over the affected tissue. Râles are generally most abundant in the morning, before free expectoration has taken place. Vocal resonance, with the whispered or the loud voice, is now exaggerated or bronchophonic. In some cases, when the consolidated lung immediately surrounds a large bronchial tube, pectoriloquy may be obtained. During the latter part of this stage, the signs of incipient phthisis usually appear at the apex of the opposite lung.

In the third stage, when cavities have formed in the lungs, if they are empty and are connected with a bronchial tube, cavernous or broncho-cavernous respiration will be detected. True cavernous respiration, of a soft blowing or puffing character, and of low pitch, is one of the very rare signs of phthisis. Broncho-cavernous respiration, having much of the bronchial element, still with a hollow quality strongly suggestive of a cavity, is heard in nearly every case. Amphoric respiration is found in exceptional instances only. Associated with these signs we usually hear numerous râles and gurgles with bronchophony, pectoriloquy, or cavernous voice, and occasionally metallic tinkling and amphoric voice. The signs of the second stage also are generally present.

If cavities are filled with fluid, none of the ordinary signs of the third stage may be obtained. Small cavities located in the deeper portions of the lungs are not easily detected.

In advanced phthisis, we may reasonably conclude that a cavity ex-

ists whenever the respiratory and vocal sounds over a small space, and limited to it, are peculiarly intense and bronchial in character, and associated with metallic râles.

Diagnosis.—Pulmonary tuberculosis is to be distinguished from chronic laryngitis, chronic bronchitis, pleurisy, chronic pneumonia, syphilis of the lung, cancer of the lung, and other intra-thoracic tumors. Its differential diagnosis from these affections will be found under their respective titles. The diagnosis will depend upon the history, symptoms, and physical signs just mentioned, and upon the discovery of tubercle bacilli in the sputum. The presence of these bacilli in any number is always indicative of tuberculosis, and in most cases their abundance is in proportion to the severity of the disease (Clinical Diagnosis, Jaksch); their absence from the sputum is not in every case positive evidence that the disease does not exist.

Elastic fibres in the sputum, though not peculiar to tuberculosis, are indicative of pulmonary ulceration.

To Stain Tubercle Bacilli in Sputum.—Many modifications of the Koch-Ehrlich method for staining tubercle bacilli have been suggested.

Ziehl's solution, which remains good for many months, is now commonly employed instead of the aniline preparations. It consists of distilled water one hundred parts, alcohol ten, carbolic acid five, fuchsin one part. The procedure which I have found most convenient is as follows:

(1) Examine the sputum on a plate of glass against a black background.

(2) Pick out a very small quantity of nummulated purulent sputum.

A platinum needle fixed in a glass rod is most suitable for this purpose; it should be sterilized in the flame of an alcohol lamp or Bunsen burner before using.

(3) Spread the selected sputum, in a *thin* layer, evenly between two glass slides, by drawing them successively one upon the other.

(4) Dry in the air or high above the flame of an alcohol lamp or Bunsen burner.

(5) Fix the albumin by passing the slide several times through the flame with the film upward.

(6) Pour about twenty minims of Ziehl's solution upon the slide thus prepared, and heat over the flame till it steams.

(7) Let it stand for thirty seconds, or longer; then wash in clean water.

(8) Decolorize to a faint pink color with a two or three per cent solution of sulphuric or any of the mineral acids.

This can be done best by dipping the slide for a few seconds in the

acid solution, washing directly in water, and holding it up to the light for inspection, repeating the operation until the faint pink color is obtained.

(9) Counterstain with a two or three per cent watery solution of methylene blue, which is merely poured upon the slide and left from thirty to sixty seconds with or without heating. Methylene blue, if a good article, is readily soluble in water. Two or three grains of chloral may be added to the ounce of methylene solution to prevent decomposition.

(10) Wash in clean water.

(11) Dry, and mount with cover-glass in glycerine or permanently in balsam, and examine; or dry and examine directly without a cover-glass, with a one-twelfth oil immersion lens. This lens with a No. 4 eyepiece (Zeiss) magnifies about a thousand diameters and shows the bacilli as represented in Fig. 29, which was drawn for me by Heneage Gibbes, of the University of Michigan.

The bacilli may be seen distinctly with lower powers, but their detection is much more easily and speedily accomplished by this lens.

Thus prepared, the small beaded bacilli appear red, while all other micro-organisms, cells, albumin, and fibres are stained blue. The only other micro-organism yet discovered which closely resembles the tubercle bacillus in form, size, and manner of staining is the bacillus of leprosy, which differs from the tubercle bacillus in taking the watery anilin stains equally as well as other bacteria (Linsley's translation of Fraenkel's Bacteriology, page 231).

Discovery of the bacilli may sometimes be facilitated by thoroughly stirring, and boiling in a large test-tube, about 3 i. of the sputum with 3 vi. of a solution of caustic soda, 3 parts to 1,000, until it forms a thin mass. This should be allowed to settle twenty-four hours, when the sediment, which carries down the bacilli, should be examined.

ACUTE MILIARY TUBERCULOSIS.

Miliary tuberculosis of the lungs is a part of a general disease; though all the viscera, and especially the peritoneum, pleura, and meninges, may be involved, the lungs are the chief seat of deposit.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—Small nodules the size of a pin-head are observed scattered over the pleura and disseminated throughout the affected lungs, which are usually congested and œdematous. To the unaided eye these tubercles appear sharply defined. Microscopically the outer zone of lymphoid cells is seen to merge gradually into the surrounding lung. The air cells contain to some degree the elements of exudation.

ETIOLOGY.—The immediate focus of general infection may be in any

organ, bones, joints, or in the urinary tract, but usually it is in the lungs or lymphatic glands.

Ulceration into a lymphatic trunk is followed by entrance of bacilli into the circulation and more or less extensive infection of other parts.

SYMPTOMATOLOGY.—The general symptoms are very like those of typhoid fever, though the temperature is frequently highest in the morning, ranging between 103° and 105° F., and occasionally going up to 107° F. Prostration is very early and marked. Involvement of the meninges gives intense headache, vomiting, opisthotonos, delirium, and ocular disturbance. The pulmonary symptoms are not characteristic, but cough is usually present and expectoration, if present, is frothy instead of muco-purulent. No tubercle bacilli are present in the sputum, unless a localized tuberculosis of the lung has existed before occurrence of the miliary form of the disease.

Acute miliary tuberculosis is attended by no physical *signs* unless the mucous membrane lining the air passages is involved, and then there are no signs except those of bronchitis. The diagnosis in such cases must rest upon the history and symptoms, and the exclusion of other pulmonary affections.

DIAGNOSIS.—Discrimination between the various forms of phthisis is often attended with more or less uncertainty. The principal features of value in distinguishing between them may be seen in the following table:

FIBROID AND OTHER VARIETIES OF SIMPLE INFLAMMATORY PHTHISIS.	CHRONIC TUBERCULOSIS OR THE ORDINARY FORM OF PHTHISIS.	ACUTE MILIARY TUBERCULOSIS.
	History.	
The constitutional symptoms come on slowly, and are less severe than would naturally be expected from the condition of the lung, as indicated by physical signs.	The constitutional symptoms come on more rapidly, and are graver than would be expected from the physical signs.	The disease is ushered in with chills and fever without complete remissions, and there is rapid accession of grave constitutional symptoms, which cannot be accounted for by the bronchitis, signs of which are the only ones to be obtained.
	Symptoms.	
The fever is intermittent, with an afternoon or evening elevation in temperature of from one to two degrees.	The fever more continuous, with nearly constant elevation of temperature, but less marked exacerbations.	Fever remittent, temperature often highest in the morning, varying from 103° to 105° or even 107° F.
Diarrhœa not common.	Diarrhœa usual.	

FIBROID AND OTHER VARIETIES OF SIMPLE INFLAMMATORY PHTHISIS.	CHRONIC TUBERCULOSIS OR THE ORDINARY FORM OF PHTHISIS.	ACUTE MILIARY TUBERCULOSIS.
	Signs.	
Rapid respiration, and signs of consolidation upon palpation, percussion, and auscultation, usually extending over a large part of the lung.	Rapid respiration, physical signs of consolidation less marked and limited to a smaller area than in the preceding variety.	Rapid respiration, with usually the signs of bronchitis, and ordinarily no signs of consolidation, but occasionally slight dulness.
No tubercle bacilli in sputum.	Tubercle bacilli in sputum.	Usually no tubercle bacilli in sputum.

FIBROID PHTHISIS.

Synonyms.—Fibroid degeneration of the lungs; fibrosis; chronic pneumonia; interstitial pneumonia; cirrhosis, or scirrhus of the lungs; induration of the lungs.

Fibroid phthisis is a chronic inflammatory affection characterized by comparatively slow progress, though in the majority of cases it finally terminates in tuberculosis. As compared with the ordinary form of consumption, the symptoms are slight in proportion to the amount of lung tissue involved.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—The chief anatomical changes consist of hyperplasia of the interalveolar, interlobular, and peribronchial structures, which encroach upon the air passages and blood-vessels, correspondingly diminishing their capacity; this encroachment is subsequently increased by the contraction of the newly formed elements. There is little or no exudation into the air cells. The disease may involve a part or the whole of one lung, or both lungs may be affected, though commonly it is confined to one side of the chest throughout the greater portion of its course.

Inspection of the affected organ reveals in most cases more or less extensive adhesions of the overlying pleura, and often extensive thickening of the latter membrane, especially when the disease has resulted from pleurisy.

Occasionally fluid is found in circumscribed pockets of the partially obliterated pleural cavity. The thickened pleura may present very much the appearance and density of fibro-cartilage. When the process is general, an entire lung may be found shrunken to one-tenth of its normal size. The color varies from a dark red to a bluish-gray, marbled with black and streaked with lighter lines.

When localized, the shrunken, cirrhosed area contrasts strongly with the adjacent normal or emphysematous lung tissue. This part is abnormally heavy, and sinks readily in water, and when pressed yields but little fluid from its cut surface. In advanced cases, the tissue is

so firm that upon section the knife grates as in cutting cartilage. The cut surface is of a dark gray or blackish color, intersected by yellowish-white bands, and mottled with lighter circles marking the position of obliterated vessels and tubes.

As the process advances, and contraction of the new tissue occurs, many of the air cells become destroyed, although here and there islets of normal or emphysematous vesicles may still remain. During the process, many of the bronchial arteries, together with numerous branches of the pulmonary artery are obliterated; and as a result of the process of contraction, here and there dilatation occurs in the bronchial tubes; and bronchiectatic cavities are found, lined by dark red, thickened mucous membrane, and containing purulent fluid, or cheesy débris. These cavities may also be the seat of ulceration or gangrene and vary from half an inch to two inches in diameter. The bronchial glands are frequently enlarged, and ultimately these and the cirrhotic lung tissue, in many cases, become the seat of tuberculosis.

When the affection is confined to one lung, the opposite organ may be functionally enlarged or may become emphysematous, and not infrequently at the autopsy this lung will be found the seat of bronchitis or acute croupous pneumonia which has been the immediate cause of death. In marked cases the heart is displaced toward the affected organ by traction of the contracting tissues, and its right cavities are usually dilated, while their walls are hypertrophied as the result of obstruction to the passage of venous blood through the lung.

Etiology.—The disease occurs most commonly in males between fifteen and forty years of age, and is generally the result of local causes having little or no dependence upon diathesis. Catarrhal pneumonia and pleurisy are among the most frequent causes of the disease, but it may result from chronic bronchitis or acute croupous pneumonia; circumscribed induration is also a common result of arrested pulmonary tuberculosis.

Symptomatology.—The progress of fibroid phthisis is not so rapid as that of the common form of consumption; but its symptoms and signs are usually much the same excepting that the symptoms do not appear commensurate with the pulmonary lesions, as indicated by the physical signs.

As a rule, the disease is chronic from its inception, although its development may date from an attack of pleurisy, pneumonia, or bronchitis. The origin is often obscure, and the history is similar to that of chronic bronchitis, with frequent exacerbations. Dyspnœa, though often absent or moderate, increases with the advance of the disease, and is subject to exacerbations, during which the difficulty of breathing may be experienced for several days. During the latter portion of the disease dyspnœa is constant upon any exertion, and eventually becomes very great, even though the patient is quiet. Cough is a common symptom,

though it varies much in different cases, and different periods of the same case. It is increased by recurrent attacks of bronchitis, and is generally worse during the winter months. When bronchiectasis exists, the cough is likely to be paroxysmal, especially severe in the morning, and accompanied by a profuse, fetid expectoration, after which relief may be experienced for several hours. Vomiting often follows these paroxysms of coughing. The sputa may be scanty, and viscid, but when dilatation of the bronchial tubes has taken place, it is generally copious, sometimes amounting to two or three pints in the twenty-four hours. It may consist of mucus or muco-pus, and is usually of a yellowish or greenish-yellow color.

Hæmoptysis is not uncommon, even in the absence of tuberculosis. During the greater portion of the disease the appetite usually remains good, and consequently the strength may be fair and emaciation gradual unless tuberculosis supervenes. In well-marked cases the signs are tolerably distinctive.

Inspection shows flattening of the chest wall over the affected part, and depression of the shoulder may be observed.

On palpation, vocal fremitus is exaggerated. The heart is dislocated more or less toward the affected side, as shown by the position of the apex-beat.

Percussion gives dulness over the affected side and exaggerated resonance on the sound side, which sometimes extends, in consequence of the distention of the healthy lung, from two to four inches beyond the median line toward the affected side.

Auscultation gives bronchial breathing and bronchophony, with or without bronchial râles. Subcrepitant râles are, however, commonly present. The vesicular murmur is feeble or absent.

The diagnosis, prognosis, and treatment of fibroid phthisis will be considered with pulmonary tuberculosis, though we may here state that, during the earlier part of the disease, the treatment indicated is essentially the same as that for chronic bronchitis.

PROGNOSIS IN THE VARIOUS FORMS OF PULMONARY PHTHISIS.—Acute miliary tuberculosis frequently runs its course within three to six weeks, and seldom extends over three months. Chronic tuberculosis may terminate fatally within five or six months, but it often lasts for two or three years, the average duration being about eighteen months. The records of autopsies show that about twenty-five per cent of the patients dying in hospitals as a result of accidents and acute disease, have cicatrices in the apices of the lungs resulting from old inflammations, probably of tubercular origin; and experience has shown that quite a large percentage of patients suffering from well-marked though not extensive tuberculosis recover. While I am not able to fortify my impression by statistics, I believe that, all told, about thirty-three per cent

recover under ordinary conditions, and I think that patients sent early to high altitudes and a dry atmosphere have their chances of recovery increased fully fifty per cent. Where the disease is so extensive at the apex of one lung that the signs may be recognized below the second rib, perfect recovery, so that no signs whatever can be detected, seldom occurs, but the disease not infrequently becomes arrested, the cough and all other symptoms disappearing, the evidence given by a scar in the lung being all that can be detected on careful physical examination. When the disease has extended as low as the fourth rib, there are a few cases in whom it may be arrested, provided they have the best hygienic surroundings; but after the whole of the upper lobe of one lung and possibly a small part of the lower lobe, together with the apex of the opposite lung, have become involved, it is very rare that much improvement takes place, though even when these conditions exist and after cavities of considerable size have been formed, we occasionally find the disease arrested, so that the patient may live for many years.

Usually fibroid phthisis continues four or five years, sometimes longer, but finally it eventuates in tuberculosis, terminating in much the same way as the ordinary form of this disease. Usually death results from asthenia, occasionally from heart failure, and in a small percentage of cases from hemorrhage. Out of over six hundred private cases of which I have records, but five are known to have died from hemorrhage. Generally the approach of death is indicated by rapid extension of the disease and speedy failure of the vital powers.

After decided swelling of the feet occurs, patients seldom live more than five or six weeks; they usually succumb in from three to eight weeks, when the strength has so far failed that they are unable to leave the bed, though sometimes life is more prolonged. Two or three days before the fatal issue, many consumptives become so feeble that the sputum is raised with great difficulty; cough becomes less and less frequent, and may finally cease a few hours before death.

Treatment of the Various Forms of Pulmonary Phthisis.—Having considered some of the special forms which pulmonary phthisis assumes, we may discuss more fully the general treatment.

As a matter of prophylaxis, healthy persons should not occupy the same apartment with consumptives, and great care should be exercised to prevent the drying of tubercular sputum, and to thoroughly disinfect or destroy it. The treatment of acute tuberculosis can seldom if ever be more than palliative, though it is proper to use the same remedies that are recommended for more protracted forms of the disease.

For chronic tuberculosis the most important remedies are alcohol, malt preparations, cod-liver oil, calcium chloride, quinine, iron, iodine, guaiacol, and oil of cloves, with proper climate.

Alcohol should be used in large quantities, as much as can be borne without affecting the head, providing it does not derange digestion or cause elevation of temperature.

Cod-liver oil should be given to those patients who can take it without disturbing their digestion, in doses of a teaspoonful to a tablespoonful three times a day, always commencing with small doses. Whenever cod-liver oil cannot be borne, it may be substituted by cream or preparations of malt. The latter are usually preferable to oil during warm weather.

Calcium chloride is a remedy of undoubted value in many cases. I have found it more serviceable than the calcium or sodium hypophosphites. The dose is from ten to twenty or even thirty grains three times a day. It may be dissolved in a small quantity of water, and combined with the cod-liver oil. By shaking the bottle before the medicine is poured out, the two can be sufficiently mixed. It may be added to an emulsion of cod-liver oil prepared as directed (Form. 3).

Quinine is the best remedy for relieving hectic fever. It will usually prove efficient when given in the same manner as for intermittent fever. It acts most promptly when given in one or two large doses a couple of hours before the fever is expected. It should be continued in this manner until the temperature falls or cinchonism appears; even though it fails to check the fever the patient is generally benefited by it.

Iron is a valuable remedy in this disease, but it must not be given when there is much fever, for it aggravates this symptom.

Belladonna is the best remedy for checking the night-sweats. Six minims of the tincture of belladonna, or the one-hundred-and-twentieth of a grain of atropine, at bed-time, is sufficient in many cases, but the dose may be increased to twice this amount, and repeated two or three times daily if necessary. For the same purpose, aromatic sulphuric acid, ℥x. to xx. properly diluted; minute doses of aconite; of agaricin, gr. $\frac{1}{8}$; of zinc oxide, gr. iij.; of ergotin, gr. ij.; or of black oxide of manganese, gr. ij.; may be given three times daily with success in some cases, but any or all may fail. I have known obstinate night-sweats checked occasionally by rubbing into the skin a powder of four per cent of salicylic acid triturated with magnesium salicylate; by placing a large pan of cold water under the bed at night, by sleeping in light blankets, or by drinking a preparation made by steeping for two or three hours two heaping tablespoonfuls of sago in one and one-half pints of water, reduced by evaporation to about one-half pint.

Tonic doses of mercury bichloride gr. $\frac{1}{36}$ to $\frac{1}{24}$, or gold and sodium chloride gr. $\frac{1}{20}$ to $\frac{1}{10}$ will be found beneficial in some cases, especially those of a chronic catarrhal or fibroid character. The same may be said of arsenious acid, but this must not be given when there is much fever.

When there is a suspicion of syphilitic origin of the disease, potassium iodide should be tried.

As a result of numerous experiments on Guinea-pigs and monkeys, E. L. Shurly, of Detroit, and Heneage Gibbes, of Ann Arbor, Mich., have demonstrated that animals may be rendered immune to tubercular virus by hypodermic injections of aqueous solutions of chemically pure iodine, prepared by J. E. Clark, of Detroit, or of gold and sodium chloride; and they have recommended, for the cure of consumption, hypodermic injections of these remedies with inhalations of chlorine gas. The injections should be made with an absolutely clean syringe, which should always be washed with pure alcohol before and after using. The treatment should be commenced with small doses, which may be gradually increased until some constitutional effects are observed or until the largest dose recommended is reached. It is usually best, excepting in advanced cases, to begin with the iodine (though it is apt to cause considerable smarting), and it should be continued ten to fourteen days, and then may be given alternately with the gold and sodium chloride solution, and later, after four or five weeks, the gold solution may be used alone if everything is going well. In some patients the gold and sodium chloride answers best, but I think most benefit will be derived from the iodine. The dose of iodine is from one-twentieth to one-sixth of a grain, and of the gold and sodium chloride from one-twenty-fourth to one-eighth of a grain.

When symptoms of iodism appear or there is loss of appetite, disturbance of the bowels, or complaint of unusual fatigue, gold preparation may be substituted for a day or two, when the iodine may be given again in diminished doses, which may subsequently be gradually increased. Sometimes, while patients are receiving the gold and sodium chloride in large doses, pains are experienced in the bowels, and in some instances there are uncomfortable sensations in the head; occasionally, also, profuse sweating has been noticed. If any of these symptoms develop, the dose should be at once diminished, or the remedy substituted by the iodine. The most favorable place for the injection is beneath the loose skin in the gluteal region. As it is difficult to get at this point on account of the clothing, the injections are given to women just below the inferior angle of the scapula or between this and the spinal column. Injections are advised daily for about two weeks, every second day for the two following weeks, and subsequently once in three, four, five, six, or seven days, gradually diminishing the frequency according to the result. When these remedies are acting well, the appetite and strength gradually improve, the weight increases, and the cough and expectoration gradually diminish. The chlorine inhalations may be given either by means of some of the common or specially devised inhalers, or in a room filled with chlorine gas. The latter is applicable to hospitals where small rooms can be arranged, or even to small bedrooms, where it is readily carried out in the following manner: first, a steam-atomizer is made to throw into the atmosphere of the room a solution of sodium

chloride, about fifteen grains to the ounce; this is continued until the atmosphere is so permeated by the spray that a person on the opposite side of the room can taste the salt. One or two teaspoonfuls of chlorinated lime are then placed upon a saucer and wet with a mixture of hydrochloric acid one part and water two parts, which causes the rapid liberation of chlorine gas. This is then held directly under the spray of salt solution, and the gas is carried by it into the atmosphere of the room, where the patient sits for ten or fifteen minutes—as long as he can well tolerate the inhalation.

I have employed this treatment in over a hundred cases of phthisis during the last few months, and found it very beneficial in the first stage, helpful in some cases during the second stage but of only little value in the third stage, though occasionally even then some appear benefited by it.

Among other remedies in phthisis, creasote has been very highly recommended, in doses of one to five minims, or even as much as half a drachm, several times a day. It has appeared to me most beneficial in moderate or small doses (Form. 7). Morsen's creasote is seemingly less irritating than other preparations. Guaiacol, one of the chief constituents of creasote, has been quite extensively tried in the treatment of pulmonary tuberculosis. Although I have had but little experience with it, general report, and especially the apparently good results obtained from its use in surgical tuberculosis by Nicholas Senn and W. T. Belfield, of Chicago, induce me to recommend its thorough trial in pulmonary phthisis. It may be administered in essentially the same doses and manner as creasote, but I prefer the carbonate of guaiacol, which has but little taste or odor, causes little irritation, and is apparently quite as efficient when given in corresponding doses.

Oil of cloves given five times a day, in doses of two to twelve minims, or oil of cassia, in doses of one to five minims, in conjunction with other remedies, has been of great benefit in some cases. The medicine should be dropped in capsules just before it is taken and administered with each meal and in the middle of the forenoon and afternoon, the patient taking, when possible, a glass of milk with each dose—never taking it on an empty stomach lest it cause irritation. The dose should be small at first and increased, one-half to one minim each day until the maximum dose is attained unless it disturbs the digestive organs.

The therapeutic value of tuberculin is still uncertain, but the majority of those who have tried it believe that it is more potent for harm than for good.

Sedative troches (Forms. 25, 26, 30, 33, and 35) and sedative inhalations of benzoin, opium, or chloroform are useful in allaying the cough (Forms. 53 to 60). Stimulant inhalations are frequently serviceable in the early stages of the disease. They are most conveniently administered with the Globe nebulizer shown in Fig. 30. For this purpose, iodine, carbolic acid, creasote, or oil of white pine are most

frequently used (Forms. 62, 68, 69, and 72 to 74). Cough mixtures are necessary, especially late in the disease, but they should be given as sparingly as possible. Sedative troches and inhalations are preferable when they will answer the purpose. The neuralgic pains which often trouble phthisical patients are best prevented by regular and vigorous frictions of the surface with a coarse towel; when severe, they are usually promptly relieved by hot applications to the surface. These applications should be as hot as can be borne, and should be frequently repeated until pain subsides.

Counterirritation is useful, especially in cases of an inflammatory character, as those growing out of pneumonia, bronchitis, or pleuritis, before tubercles have been deposited.

I sometimes employ for this purpose an ointment composed of tartar

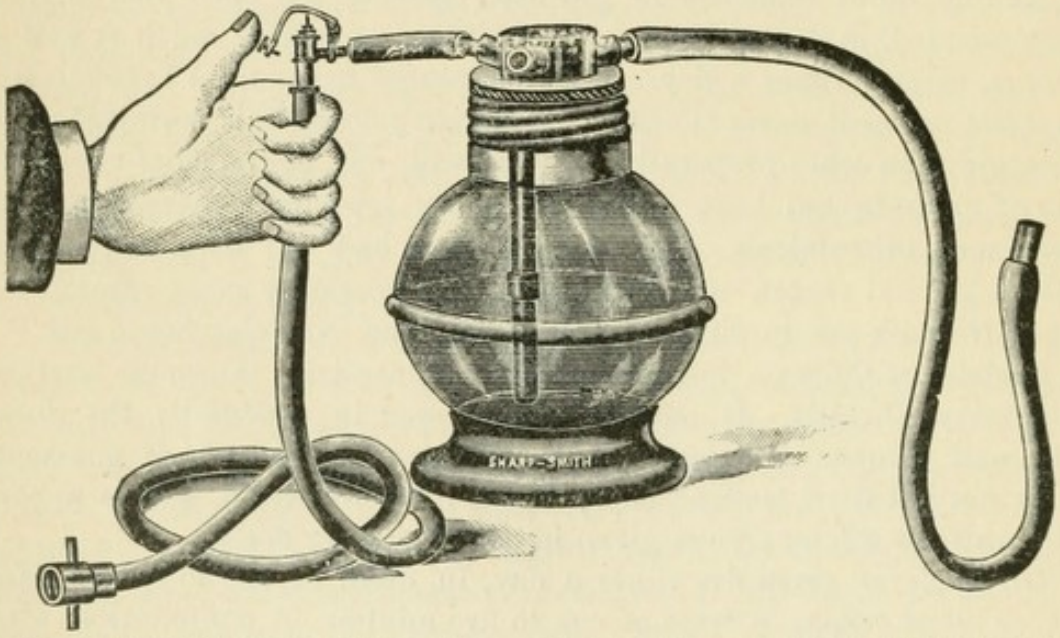

Fig. 30.—Globe Nebulizer, ⅓ Size. Best used with an air pressure of ten or fifteen pounds only. It may also be used by the hand ball.

emetic, croton oil, cantharides, stramonium, and camphor (Form. 10). It is an effectual and almost painless counterirritant. Burgundy pitch plasters, croton oil, iodine, or blisters may be used for the same purpose.

The digestive functions must receive careful attention. Nutritious and easily digestible diet of varied character should be ordered.

Climatic Treatment.—Many consumptives will be greatly benefited by suitable climatic influences. In the first stage of phthisis, I believe that the patient's chances of recovery are improved from fifty to seventy-five per cent by residence in a suitable climate; in the second stage, from fifteen to thirty per cent; in the third stage, a small percentage will be permanently benefited; and in a large proportion of others life may be considerably prolonged.

There is no climate to which consumptives may be sent indiscriminately, but suitable places should be selected for each patient. Some

patients feel better in cold weather, but the majority are better in summer. It will be found that those who feel best in winter are likely to be benefited by a comparatively cool climate, the others in a warm climate. As a rule, a warm, dry climate and high altitude are most salutary. It is always desirable, when there are no contra-indications, that the patient in the early stages of the disease should be sent to an altitude of from six to seven thousand feet; but this is not suitable for those who are nervous to a marked degree, or who have a high temperature, pronounced cardiac disease, emphysema, or laryngeal complications. Hæmoptysis is not, as is often supposed, a contra-indication to a sojourn in a high altitude; on the contrary, bleeding is often promptly checked by this change, and those who seldom or never have hemorrhages in a high altitude frequently experience them quickly upon a return to a lower level. In the second stage of the disease, a high altitude is often beneficial, but we cannot feel so certain of its results; therefore it is best to send the patients to an altitude of not more than two or three thousand feet, and, if they do well, subsequently advise a higher altitude.

In the earlier stages, warmth is not so important, providing an abundance of sunshine and dry atmosphere can be obtained, though it is usually best to recommend for such patients a southern latitude in winter.

In this country in summer the high altitude of Colorado, Wyoming, Montana, and Utah affords a typical climate for these cases, whereas in winter they generally do better in New Mexico, western Texas, or Arizona.

Those for whom an altitude of two or three thousand feet is preferable often do well in summer in some portions of Dakota, Nebraska, and Minnesota; in the Adirondacks, or the mountains of Virginia, North Carolina, or Tennessee. In winter, more suitable climates are found in warmer latitudes; many cases will do well in eastern Tennessee or western North Carolina or in Georgia at from fifteen to eighteen hundred feet above the sea. The typical climate for these cases in the winter months is found in Arizona or southern California, in the latter among the foot-hills as far as possible removed from the ocean. Southern New Mexico and the western portion of Texas are favored by a similar climate. In many parts of Mexico, patients in the first and second stages of consumption do remarkably well during the winter months.

In the Old World, the mountainous regions of southern Germany, of Switzerland, Austria, Spain, France, Algiers, and Egypt, according to their temperature, offer advantageous resorts for summer or winter.

In the advanced stage of the disease, patients, if sent anywhere, should be recommended to a warm climate and usually to a comparatively low altitude, of not more than one or two thousand feet above the sea. For these, a typical climate is found in Arizona or southern California, and many of them do well in Florida, South Carolina, Georgia, and Texas.

In the Old World, these patients also find a suitable climate in southern Spain or France and in Algiers or Egypt, but usually persons who have passed to this stage of the disease are much better off at home, where they are surrounded by friends and the comforts that cannot be obtained elsewhere. No patients should be advised to go from home except those whose financial condition will enable them to secure easily the comforts as well as the necessaries of life, and usually to surround themselves with agreeable companions and friends.

CHAPTER XI.

THE HEART.

ANATOMY AND PHYSIOLOGY.

A KNOWLEDGE of the anatomy and physiology of the heart is so essential to a correct diagnosis, that we shall give them brief consideration before proceeding to the means for detecting cardiac diseases.

The heart is a hollow, muscular organ of conical form, which as the centre of circulation distributes blood throughout the entire body. Located near the central portion of the chest, it is held in place above by the large blood-vessels springing from its base, and below by the attachment to the diaphragm of the fibro-serous sac which envelops it. In front it is sheltered by the sternum; posteriorly by the thick chest walls, and spinal column; and laterally it is cushioned by the lungs.

Its long axis is oblique to the perpendicular axis of the chest; its base is directed upward, outward, and backward toward the right shoulder; its apex downward and forward.

The pericardium, the fibro-serous sac which envelops this organ, is composed of an external, fibrous layer and an internal, serous layer. The external layer incloses the arteries for about two inches from the base of the heart, and is continuous with their external covering; below, it is attached to the diaphragm. The internal, serous layer completely envelops the heart, and covers the blood-vessels springing from its base for about two inches. It is then reflected upon the inner surface of the fibrous layer, and passing downward covers the upper surface of the diaphragm, beneath the heart, thus forming a closed sac similar to the pleura. The two serous surfaces of the pericardium, constantly in apposition during health, are moistened by serum, and glide upon each other without friction during the action of the heart. The pericardium extends from the level of the second to that of the seventh left costal cartilage. It is farther from the chest walls superiorly than inferiorly.

The heart, with its pericardium, is in relation: anteriorly, with the anterior borders of the lungs and a small portion of the thoracic walls, from which it is separated by a small amount of areolar tissue; laterally, with the lungs covered by the pleuræ; posteriorly, upon each side, with the lungs and pleuræ. In the middle line posteriorly, it lies near the spinal column, from which it is separated by cellular tissue and the aorta and œsophagus.

The heart is about the size of its owner's fist, its weight ranging in women from eight to ten ounces, in men from ten to twelve. The anterior surface is convex; the posterior surface flattened; the right border is long, thin, and sharp; the left border is short, thick, and rounded. Running longitudinally about the heart is a well-defined fissure, found upon the anterior surface within half or three-quarters of an inch of the left border, and on the posterior surface a similar distance from the right border. This fissure lodges the coronary arteries, which supply the heart with blood; and it indicates the position of the septum, which divides the right side of the heart from the left. Near the base of the heart is a transverse fissure, interrupted in front by the origin of the pulmonary artery. This fissure indicates the position of the septum between the cavities at the base of the heart and those at the apex.

By these septa, the heart is divided into four cavities: two above at the base, known as the right and left *auricles;* two below at the apex, known as the right and left *ventricles.* Each of these cavities is capable of containing about two fluid ounces. The walls of the cavities upon the right side are thinner than those upon the left, and the walls of the auricles are much thinner than those of the ventricles.

The right auricle receives the blood from the venous system, through the ascending and descending venæ cavæ, and transmits it through the auriculo-ventricular opening, into the right ventricle, which, contracting, forces the blood onward through the pulmonary artery into the lungs. The left auricle, receiving the blood from the lungs through the pulmonary veins, transmits it to the left ventricle, whence it is distributed, by the aorta and its branches, throughout the body.

The internal surface of the heart is lined by a glistening membrane, known as the *endocardium,* folds of which at the various orifices constitute the valves. At the orifice between the right auricle and the right ventricle, we find three of these folds, which are named the tricuspid valves. At the orifice of the pulmonary artery are three similar folds, known as the pulmonary semi-lunar valves. At the aortic orifice are a similar number, called the aortic semi-lunar valves. At the orifice between the left auricle and ventricle are two folds, known as the mitral valves.

The greater portion of the heart lies beneath the lower part of the sternum, but the right auricle, and a small part of the right ventricle, extend from one-half to three-fourths of an inch to the right of the sternum; the ventricles extend about two inches to the left (Fig. 1).

The auricles are on a line with the third ribs, the right auricle extending considerably beyond the sternum into the third interspace upon the right side, the left being located beneath the third left costal cartilage and intercostal space upon the left. The left ventricle lies mainly behind the right; that part of it which is superficial is found entirely to the left of the sternum. Most of the right ventricle lies behind the lower part of the sternum; but a small part of it, at the base, extends to

the right of the sternum, and its apex is found to the left of this bone in the triangular space between the sternum and the margin of the left lung. The base of the heart extends to the upper margin of the third rib, corresponding behind to the sixth and seventh dorsal vertebræ; its apex lies at the fifth costal interspace from an inch and a half to two inches below the nipple, about half an inch to the right of the mammillary line, and two or two and a half inches to the left of the sternum. The position of the apex changes slightly with the respiratory movements, the position of the patient, or with the distention of the stomach.

It is said that the apex may move as much as an inch and a half from left to right, or *vice versa*, when the patient lies on the right or the left side; a few cases have been reported in which prolonged decubitus on one side seems to have caused permanent dislocation of the heart.

From the base to the apex of the heart, in a vertical line, the distance is about five inches. Measuring from the mesosternal line to the left over the third rib, the heart extends from two and one-half to three inches, over the fourth rib three and one-half to four inches, and in the fifth interspace from three to three and one-half inches.

Position of the Valves.—The relation of the valves to the surface of the chest may be ascertained by passing needles through the chest walls of the cadaver before the thorax is opened. In this manner it has been ascertained that the pulmonary valves lie beneath the junction of the third costal cartilage of the left side with the sternum. The mitral valves lie close to the left border of the sternum in the third intercostal space. The tricuspid valves lie in front of the mitral, near the middle of the sternum, on a line with the fourth ribs. The aortic valves lie beneath the sternum, just below the level of the third ribs, and a little to the left of the median line (Fig. 1). As indicated in treating of the chest regions, a very small circle, with its centre at the left edge of the sternum in the third intercostal space, will include the greater part of all of these valves.

The discrepancy noticeable in the descriptions, by different authors, of the position of the valves is probably due, in the main, to their being located after the thorax has been opened, when the collapse of the lungs has more or less displaced the heart.

The aorta springs from the base of the left ventricle, and passes upward, forward, and to the right, to the second intercostal space, where it is more superficial than in any other part of its course. In this situation, it is within the pericardial sac; thence it passes backward, upward, and to the left, and finally passes downward, bending completely upon itself, so as to rest along the left side of the fifth and sixth dorsal vertebræ. The highest portion of the arch is on a line with the first costo-sternal articulation.

The pulmonary artery rises from the base of the right ventricle, beneath the third costal cartilage at its junction with the sternum, and passes upward and outward, about two inches, to the second costal cartilage, where it bifurcates, one of the branches going to each lung. It will be seen that the aorta may be found close to the margin of the sternum in the second intercostal space upon the right side, and the pulmonary artery in a similar position on the left.

PHYSIOLOGICAL ACTION OF THE HEART.

In health, the heart acts as a perfect automatic engine, the strokes of which follow each other in regular succession, continuing from fœtal life until the moment of death.

The pulsations of the heart consist of, first, contraction, then dilatation of its walls; this is followed by a short period of rest. These pulsations occur in the adult from seventy to eighty times per minute. During their occurrence the blood is flowing from the auricles into the ventricles, and from these on into the arteries, and the valves guarding the orifices of the heart are opening and closing synchronously with its contraction and dilatation (Figs. 34 and 35). The contraction of the heart is known as its *systole;* the dilatation, as its *diastole.*

The cardiac pulsation begins with auricular systole, which occupies about one-eighth of the period of a complete pulsation. While this is taking place, the blood is flowing through the auriculo-ventricular openings into the ventricles, and the mitral and tricuspid valves float out upon the current, causing no obstruction (Figs. 34 and 35).

The systole of the auricles is followed immediately by their diastole, a passive movement which continues from the end of the systole to the beginning of the next pulsation. This occupies seven-eighths of the time of a complete cardiac pulsation. During the diastole of the auricles, the blood is again filling them from the venæ cavæ and pulmonary veins. The contraction of the cardiac muscular fibres passes with a wavy motion from the auricles to the ventricles, so that the ventricular systole immediately follows that of the auricles.

During the systole of the ventricles, the vertical diameter of the heart is shortened; the apex approximates more nearly to the base; at the same time it describes a spiral motion from left to right and from behind forward, striking against the chest wall between the fifth and sixth ribs, where its impulse may usually be seen and felt.

With this contraction there is sudden closure of the mitral and tricuspid valves. The semi-lunar valves being thrown open by the current, the blood is carried onward into the aorta and the pulmonary artery (Fig. 35). The time occupied by the *systole of the ventricles* is about *three-eighths of a complete pulsation.* With the closure of the mitral and tricuspid valves, we may hear the first sound of the heart.

The ventricular diastole follows immediately after their systole. The elastic tissue of the arteries contracts, forcing a portion of the blood backward toward the heart, which it is prevented from entering by the abrupt closure of the semi-lunar valves that guard the aortic and pulmonary orifices.

With *diastole of the ventricles* the heart assumes its former shape and position, the auriculo-ventricular valves open, and blood flows passively into the ventricles. This occupies about *one-fourth* of the period of a complete cardiac pulsation.

Closure of the semi-lunar valves, which is caused by the contraction of the arteries, produces the second sound of the heart.

The diastole of the ventricles is followed by a period of *rest*, which occupies about *one-fourth* of the time for a complete pulsation.

During this period, the blood continues to flow from the auricles into the ventricles, so that, at the instant just preceding another pulsation, all of the cavities of the heart are full, but not distended. With the contraction of the auricles, the ventricles are distended by an additional amount of blood, but probably the auricles are not completely emptied. The distention of the ventricles, caused by the systole of the auricles, excites their contraction, and the blood is forced onward into the arteries. If the cycle of time taken up by a cardiac pulsation were divided into five equal parts, about one-fifth would be occupied by the systole of the auricles, two-fifths by the systole of the ventricles, and two-fifths by the diastole of the ventricles and the period of repose. The physiological action of the heart is graphically represented by a modification of Gairdner's diagram (Fig. 31).

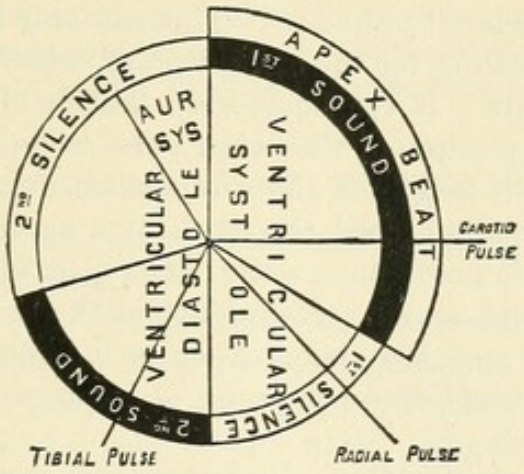

FIG. 31.—PHYSIOLOGICAL ACTION OF THE HEART (altered slightly from Gairdner).

In the diagram, the inner circle represents the physiological action of the heart, apart from any manifest signs.

The outer circle represents the external manifestations of the heart's action: the ring between the circles illustrates the sounds and periods of silence; out side of the outer circle represents the impulse of the apex against the chest wall. Lines radiating from the centre represent the pulse in the neck, wrist, and ankle.

As seen by the diagram, the systole of the *auricles* gives rise to no external manifestations, but with the beginning of the *ventricular systole* we appreciate the first sound of the heart and, at the same time, we may feel the beat of the apex against the chest wall, and the carotid pulse.

The long, first sound, as indicated in the diagram, is followed by a short period of silence, known as the first silence, during which the radial pulse may usually be felt.

Immediately following the first silence the ventricular diastole begins, and with it occurs the second sound of the heart, which, as indicated in the diagram, is shorter than the first, and is followed by the second or long silence, extending through the period of rest and the time occupied by the auricular systole.

In some cases only one sound of the heart can be heard, either at the apex or at the base. In such instances, in order to determine which is the first and which the second, it is absolutely necessary to associate the sound with the arterial pulsation. This can only be done, in the majority of cases, by feeling for the carotid pulse, which occurs with the first sound of the heart. If the heart were beating slowly, it might be easy to recognize the position of the radial pulse between the first and second sounds; but as the length of the first silence, during which this may be felt, does not usually exceed the tenth of a second, it is difficult to be certain whether it accompanies the latter part of the first or the first part of the second sound. Knowledge of the instant when the carotid pulsation or the apex beat takes place is indispensable in ascertaining whether an abnormal sound precedes or accompanies the systole of the ventricles.

The regular contraction, dilatation, and rest of the heart constitute what is known as its rhythm. In health, each pulsation is similar in every respect to those which precede and follow it. In disease of the heart, alterations in the rhythm are among the most constant signs; and in all the affections giving rise to abnormal sounds produced at the valvular orifices, the signs occur with either contraction or dilatation of the organ. It therefore becomes necessary in the physical diagnosis of cardiac disease to ascertain the rhythm of the heart. When the pulsations are of normal frequency this is an easy matter, if we recollect that the first sound is dull, heavy, and prolonged, while the second sound is comparatively short and clacking, and that the period of rest, or long silence, follows the second and precedes the first, and also the first sound is coincident with the carotid pulse and the impulse of the apex beat. If the heart is beating more than a hundred times per minute, it is always difficult, and frequently impossible, by auscultation alone, to distinguish between the two sounds.

If we divide the entire period of the cardiac pulsations into two parts, one of motion and the other of rest, it at once becomes evident that the more rapid the

pulsations the shorter must be the period of repose, and consequently the shorter will be the silence between the two sounds of the heart. This is well illustrated by a series of circles of increasing size (Fig. 32).

In the first or smallest circle, which indicates the most rapid pulsation of the heart, the intervals between the first and second, and the second and first,

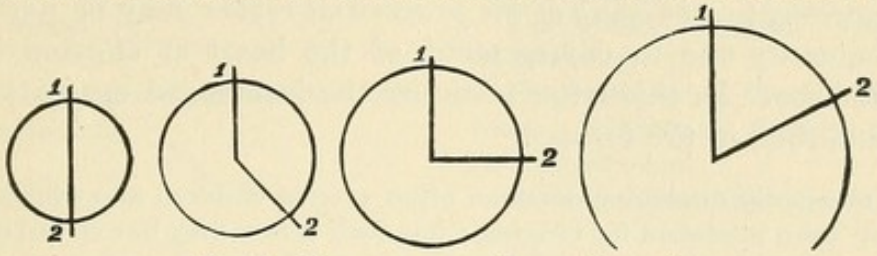

FIG. 32.—RHYTHM OF THE HEART (LOOMIS).

sounds are equal; whereas in the largest circle, in which the interval between the first and second sounds is represented by the same distance upon the circumference as in the small circle, the time between the second and the first sound is greatly increased, as indicated by the greater distance on the circumference. In the small circle the time between the first and the second sound is equal to that between the second and the first, while in the large circle the time between the first and the second sound, which corresponds to the period of motion, is only about one-fourth as great as that which includes the period of rest between the second and the first.

PHYSICAL EXAMINATION OF THE HEART.

The methods employed in examination of the heart are those already described, except succussion.

Upon inspection of a patient suffering from cardiac advanced disease, we often observe a peculiar sodden expression, with puffiness of the lower eyelids. In many instances there is marked pulsation of the veins and arteries at the base of the neck. Slight pulsation of the jugular vein is not a sign of cardiac disease, for it may be caused normally by the auricular contraction. Distinct systolic jugular pulsation in this position is always associated with more or less dilatation of the right side of the heart, which may result from protracted emphysema, mitral disease, or obstruction of the pulmonary artery by embolism or thrombosis. When very marked, especially on the right side, it is always associated with dilatation of the right ventricle and regurgitation of blood through the tricuspid valves, by which the impulse is transmitted directly to the jugular veins, as there are no valves guarding the opening of the descending vena cava into the right auricle. Pulsation in the veins is always most distinct when the patient is lying down, and may be rendered still more noticeable by pressing the blood upward in the vein with the finger, and allowing the vessel to refill from below.

Visible pulsation in the superficial arteries is not uncommon in conditions of health; but when this is excessive and symmetrical in the carotid, subclavian, and brachial arteries, it is always due to hypertrophy and dilatation of the left ventricle, with regurgitation through the aortic valves. Marked pulsation confined to one subclavian or carotid artery

usually indicates dilatation of the vessel, and the commencement of an aneurism.

By inspecting the chest, we obtain information regarding the *form of the cardiac region* and the position and character of the *apex beat.*

Enlargement or bulging of the præcordial region may be normal, but it is frequently due to enlargement of the heart or effusion into the pericardial sac. In this latter instance, the intercostal spaces are more prominent than in the former.

The unusually distinct pulsations often seen in children and emaciated persons have been mistaken for bulging; but such errors may be avoided by careful inspection and palpation.

Rachitis may cause bulging of the præcordial region, but in such instances a corresponding depression is usually found on the posterior aspect of the chest, immediately to the left of the spine, and the spine is generally curved.

Prominence anteriorly caused by aneurism of the aorta is found only above the fourth rib.

Depression in the præcordial region, of a permanent character, usually indicates previous pericarditis with adhesion of the two surfaces of the pericardium to each other, and of the pericardium to the costal pleura.

Care must be taken not to confound with this condition those rhythmical depressions which may occur independent of adhesions, as the result of atmospheric pressure. These take place when the heart is enlarged and the left lung contracted, provided the person has thin and elastic chest walls.

Inspection reveals any alteration in the position, character, and force of the *apex beat.* The apex is crowded upward and outward by hypertrophy of the left lobe of the liver or by abdominal tumors. It may be carried directly upward to a point above the fifth rib by pericardial effusions; it is raised by contraction of the left lung, as in fibroid phthisis. It is crowded downward and to the right, when the left lung is enlarged by emphysema, or it may be drawn in the same direction by contraction of the right lung. It is crowded to the right by collections of fluid or of air in the left pleural sac, or by large tumors occupying that side of the chest; to the left, by corresponding conditions upon the right side. It is forced downward by aneurisms or by other mediastinal tumors and is drawn downward and inward by hypertrophy of the right ventricle. It is carried downward and to the left by hypertrophy of both ventricles, but in uncomplicated hypertrophy the apex seldom extends more than an inch to the left of its normal position. It is also carried downward, and often far to the left, by enlargement of the heart, as the result of dilatation or of dilatation and hypertrophy combined. The significance of alterations in the position of the apex beat is shown at a glance in the following table:

Displacements of the Apex.	*Significance.*
Apex crowded to the right or left.	Fluid, air, or tumors in opposite side of chest, or contraction of the corresponding lung.
Apex raised.	Pericardial effusions. Contraction of left lung.
Apex more or less upward and outward (to the left).	Hypertrophy of the left lobe of the liver. Abdominal tumors and pericardial effusion.
Apex depressed.	Aneurism or other mediastinal tumors.
Apex more or less downward and to the right.	Pulmonary emphysema. Contraction of the right lung or hypertrophy of the right ventricle.
Apex more or less downward and to the left.	Hypertrophy of the left or both ventricles. Dilatation of the heart. Hypertrophy with dilatation.

The *area* over which the cardiac impulse can be seen is increased in all those diseases which cause enlargement of the heart.

Feeble pulsations above the fourth rib are usually due to auricular contraction, but they may be caused by an aneurism of the aorta. These two conditions can be distinguished from each other by noting the time of their occurrence. Pulsation of the auricles always precedes the apex beat, while that of an aneurism must necessarily follow or accompany it. If the heart is acting slowly, this distinction can be made easily by ordinary inspection, but this is not the case if it is beating rapidly. Under such circumstances the differentiation is facilitated by attaching, by means of wax, two bristles, each carrying a paper flag, to the two pulsating points, one over the apex and the other above the fourth rib. By watching their movements, it will be easy to determine which is first and which second.

When there is dilatation of the ventricles, or when agglutination of the two surfaces of the pericardium has taken place, the *character* of the impulse is wavy or undulating; it may sometimes be seen over the entire præcordial region.

Alterations in the force of the impulse may be recognized ordinarily upon inspection, but can be better appreciated by palpation.

Before examining the heart by palpation, it is always desirable to ascertain the condition of the *pulse*, the signs furnished by which are sometimes sufficient to establish the diagnosis.

If the radial pulse is of unequal force upon the two sides, it is probably caused by an aneurism, though it may depend upon an abnormal distribution of the arteries. In the latter case pulsations in the brachial arteries are alike on the two sides; whereas, in case of aortic aneurism, they vary in force.

If the pulse is small and weak when the arm is hanging in the natural position, and if it disappears upon raising the arm, general anæmia is present, and it may be the only cause for this sign. When the arm is in the natural position, if the pulse is small and weak, and if it maintains the same characteristics when the arm is elevated, there is likely to be disease at the mitral valves; if the pulse is also very irregular, it is probably caused by mitral stenosis.

If the pulse is small and irregular, but distinct, and upon elevation of the arm becomes still more distinct, two lesions are present, one at the mitral valves, and the other at the aortic.

If the pulse is full and distinct with the arm in its natural position, and becomes much more distinct and assumes the characteristics known as hammer pulse when the arm is elevated, there is probably regurgitation through the aortic valves, with more or less hypertrophy and dilatation of the left ventricle.

Upon examining the chest by palpation, we obtain evidence concerning the force, frequency, and regularity of the heart's action, and we may detect abnormal pulsations or thrills.

By pressing firmly upon the sternum with one hand, while the other is pressed upon the back, we are sometimes able to detect pulsations in a slightly dilated aorta which could not be felt in the ordinary manner.

The position of the impulse is to be noted. Forcible pulsation above the fourth rib may be due to an aneurism; but if observed to the left of the sternum, it is ordinarily caused by hypertrophy and dilatation of the left auricle. The two conditions may be differentiated by observing whether the pulsation precedes or follows the apex beat.

When the left lung is retracted from the base of the heart, pulsation of the pulmonary artery may be frequently seen in the second intercostal space. It can be distinguished from pulsations of the auricle by the time of its occurrence.

Abnormal pulsations along the course of the aorta are nearly always aneurismal; but in very rare instances they are caused by displacement of the artery, as in rachitis. If the pulsations are feeble, they can be most distinctly felt during expiration.

Pulsation beneath the lower portion of the sternum, and in the epigastric region, with disappearance of the apex beat, is a sign of enlargement of the right ventricle.

The *force* of the heart may be increased or diminished.

The force is *increased* in simple hypertrophy, and in hypertrophy with dilatation, whenever the former more than compensates for the latter. It is slightly increased in the early stages of endocarditis, and of pericarditis; and it is increased by simple irritability of the heart, as in hysterical palpitation.

Occasionally a double shock is felt in case of extensive hypertrophy and dilatation, due to the rebound of the heart after its systole.

The force is *diminished* when the chest walls are very thick, in consequence of a large amount of adipose tissue; when the heart is abnormally separated from the chest walls, as in pulmonary emphysema; and when there is effusion into the pericardial sac. It is also diminished when the heart is enfeebled by atrophy, fatty degeneration and softening, or general muscular debility resulting from protracted or low forms of fever or other disease.

The position of the apex beat can often be detected by palpation when it is not perceptible upon inspection. It is altered by the diseases mentioned in speaking of inspection.

The *frequency* of the heart's action is increased in such a great variety of diseases that it is not a sign of much importance in the diagnosis of cardiac affections.

Irregularity of the heart's action is often a sign of disease in this organ.

When the pericardial surfaces are roughened by exudation, *friction fremitus* may be obtained. This is usually most distinct in the fourth intercostal space, near the left margin of the sternum.

Regurgitation through the valvular orifices gives rise to a peculiar vibration known as the *purring tremor* or thrill, which may be felt by the fingers. This is sometimes detected by simply touching the surface, but in other instances firm pressure must be made.

Exceptional.—The same sensation is occasionally communicated from the larger arteries.

Feeble *epigastric pulsation* is frequently found in perfectly healthy individuals; but pulsation in this locality, associated with absence of the apex beat from its normal position, is generally the result of dilatation of the right ventricle, with or without hypertrophy. This is a common sign of dilatation of the right side of the heart caused by pulmonary emphysema. Epigastric pulsation may be due to the impulse of the abdominal aorta, especially in emaciated people who have formerly been of full habit. It occurs also when a tumor rests upon the aorta in such a manner as to be lifted with each pulsation; and it is one of the signs of aneurism of this artery.

Exceptional.—Sometimes epigastric pulsation is due to the action of the heart upon the left lobe of the liver.

Hepatic pulsation in a few rare instances is caused by venous regurgitation from a dilated right ventricle, through the tricuspid valves and the right auricle, into the ascending vena cava. It sometimes extends over the entire hypochondriac region of the right side, but in other instances it is limited to a portion of the liver. Similar pulsations are observed in very rare cases, as the result of an aneurism, the pulsations of which are transmitted through the liver.

Sometimes a peculiar pulsation is communicated to the epigastric region by

the systole of the heart, the apex of which draws the diaphragm upward in contraction instead of crowding it downward, in consequence of agglutination of the two surfaces of the pericardium. This pulsation is the reverse of that ordinarily observed, the expansion taking place with the dilatation instead of with the contraction of the heart.

By percussion, we learn the size of the heart, or detect collections of fluid or air in the pericardium. It is generally considered very difficult to map out this organ by percussion, but by attention to the following rules we find it comparatively easy. The patient should be in the recumbent posture when the examination is made, and the force of the blow should be proportionate to the depth of the part to be examined. To learn the extent of the cardiac area which is not covered by lung, we must percuss lightly; to learn the deeper outlines of the organ, a harder stroke must be made.

For clinical purposes, it is not necessary to find the exact limits of the heart in every direction, for our results will be equally good if we ascertain simply the upper, lower, and lateral lines of dulness, over its greater diameters.

To find the base of the heart, percussion should be performed on a line parallel to the sternum and about an inch to the left, so as to avoid the dulness occasioned by the aorta and the pulmonary artery, which in no way differs from that of the heart itself. On this line percussion should be made from above downward, until we reach the upper limit of cardiac dulness, ordinarily found at the third rib.

To locate the lateral boundaries, percussion should be made in the fourth intercostal spaces. Beginning in the right mammary region, where there is perfect resonance, the examination should be carried gradually toward the sternum, until the cardiac dulness is reached; which will usually be about half an inch to the right of this bone.

Upon the left side, the examination should be commenced left of the line of the nipple, and carried gradually toward the sternum, until cardiac dulness is obtained, usually about half an inch to the right of the mammillary line.

It is a difficult matter, by simple percussion, *to find the lower border of the heart,* since it lies immediately above the left lobe of the liver, and a distinction between the dull or flat sounds produced by these two organs is hardly practicable. If we find the apex of the heart either by palpation or by auscultation, and then the upper surface of the liver, in the right mammary region, by forcible percussion, and draw a straight line between these two points, it will correspond almost exactly with the inferior border of the heart.

Cardiac Dulness.—In a small triangular space at the inner part of the left mammary region, and at the lower part of the sternum, the heart lies close to the chest wall, not being covered by the anterior border of the lung (Fig. 1). This area, which is about two and one-half inches

in width, and nearly the same in height, is known as the *area of superficial cardiac dulness*. It might appropriately be called the *area of cardiac flatness*. The apex of this triangle is at the centre of the sternum, nearly on a line with the fourth rib; the base corresponds to the costal cartilage of the sixth rib.

This space is altered in extent by various diseases of the heart and the lungs. Its area is usually increased by all those affections which cause enlargement of the heart, as hypertrophy and dilatation, or simple hypertrophy.

In some cases of hypertrophy, an emphysematous condition of the lung more than counterbalances the enlargement of the heart, and thus the space, instead of being increased, is diminished.

This area is also increased by effusions of fluid into the pericardial sac.

Normally, the area is *increased* by forced expiration, and *diminished* by deep inspiration.

The area of superficial cardiac dulness is *diminished* by emphysema, which crowds the anterior border of the left lung over the heart, and by pneumothorax; it is *obliterated* in the rare disease known as pneumopericardium, in which air or gas collects in the pericardial sac, and the normal dulness is supplanted by tympanitic resonance.

The *area of deep-seated cardiac dulness* corresponds to the borders of the heart. It extends normally from the third rib above to the resonance of the stomach below; and laterally from about three-fourths of an inch to the right of the sternum to within half an inch of the left nipple. This area of dulness is increased in those affections which cause enlargement of the heart, as hypertrophy and dilatation, and by pericardial effusions.

When the dulness is first increased in the upper portion of the præcordial space above the third ribs, we may be almost certain that there is pericardial effusion, for an increase in the vertical diameter of this area is seldom found in disease of the heart itself.

The area of cardiac dulness is apparently increased by consolidation of the left lung.

The outlines of the heart may be traced a little more easily by auscultatory percussion than by the ordinary method of percussing. In practising this method, we may employ either the solid stethoscope made for this purpose, or the ordinary binaural stethoscope with the small chest-piece. In either case the chest-piece should be placed over the most superficial part of the heart, and percussion should be made from the resonant portion of the lungs toward the central portion of the heart, from above downward and laterally from without inward. By this method, as soon as the outer limits of the pericardium are reached, the change in the percussion note is at once perceptible to the listener.

In auscultation over the heart, accurate information cannot usually be obtained by the unaided ear; but by mediate auscultation, especially

if the small chest-piece of the stethoscope be used, most satisfactory results can be secured.

The patient should be in the recumbent position during at least a portion of the examination, which should be commenced while the individual is breathing naturally. Subsequently, the patient should be directed to take three or four deep inspirations, which will enable us more clearly to detect sounds that are produced by the lungs. Then he should hold his breath for a few seconds, which will enable us to eliminate pulmonary sounds, and will render the heart-signs more distinct.

The examination must not stop with the præcordial space, but should be carried over the entire chest, and the various points must be localized at which the heart sounds, both normal and abnormal, may be heard most distinctly. It is not the point at which the sound may be heard which is of diagnostic importance, but the *point at which it is loudest.*

CAUSE OF THE HEART SOUNDS.

Considerable difference of opinion exists regarding the *cause* of the heart sounds. All concede that the second sound is usually produced by closure of the semi-lunar valves; and it is generally admitted that several elements enter into the production of the first sound, though the importance of each of these is variously estimated by different authors.

The main factors in the production of the first sound are: first, the closure of the mitral and the tricuspid valves; second, the contraction of the muscular fibres of the heart; third, the impulse of the apex against the chest walls. Besides these elements, friction of the blood against the inner surface of the heart, and of the heart against the surrounding tissues, evidently plays some part in forming this sound. I believe that the contraction of the muscular fibers is a much more important factor in the production of the first sound than is generally supposed.

The influence of the contraction of the muscular fibres may be shown by the following simple experiment. Place the end of the stethoscope over the body of a muscle which can be contracted or relaxed without moving the integuments, as, for example, upon the ball of the thumb; flex and extend the terminal phalanx regularly about seventy times a minute and one will hear what almost seems to be the heart beating immediately beneath the instrument. Skoda states that the heart sounds may be produced by the arteries, and it appears to follow with tolerable certainty that both ventricles, the pulmonary artery, and the aorta are capable, each separately, of producing both the first and second sounds perceptible in the region of the heart.

In health, the *first sound* of the heart is dull, soft, and prolonged, as compared with the second, and is synchronous with the systole of the heart, the apex beat, and carotid pulse. Its point of maximum intensity corresponds to the apex beat.

The *second sound* of the heart, which is dependent upon closure of

the semi-lunar valves, caused by resilience of the arteries, is shorter, sharper, and more superficial than the first, and possesses none of that muscular element observed in the latter. It coincides with the diastole of the heart and follows the arterial pulse and apex beat. Its point of greatest intensity is at the articulation of the left third costal cartilage with the sternum. Immediately following the second sound is the period of silence, which varies in duration with the rapidity of the heart's action.

The extent of the area over which the cardiac sounds may be heard will vary with the adaptability of the surrounding organs for transmitting sounds. If the lungs are solidified, the sounds may be heard much farther than in the normal condition; but if the lungs are emphysematous, the sounds are not heard as far as in health.

Usually the sounds produced upon the right side are heard loudest over the corresponding portion of the heart, and toward the right side of the sternum; while those produced upon the left are heard loudest over the left side of the heart, and nearer the left nipple.

As a rule, the heart sounds are louder in children and in those with thin chest walls than in adults or in those with the parietes very muscular or thickened by adipose tissue. The intensity varies in different individuals with the changing force of the impulse and the conformation of the chest walls, and with peculiar idiosyncrasies, which we cannot well understand.

Hence, we recognize the necessity of studying a large number of healthy hearts, for no one individual can be taken as a standard.

MODIFICATIONS OF THE HEART SOUNDS BY DISEASE.

The heart sounds are modified by disease, in their *intensity*, *pitch*, *quality*, *seat*, and *rhythm*. They may be preceded, accompanied, or followed by abnormal sounds known as murmurs; or murmurs may entirely supplant them.

The *intensity* of the heart sounds is *increased* by hypertrophy of the ventricles, nervous irritability, cardiac palpitation, consolidation of adjacent lung tissue, and, exceptionally, by dilatation of the heart. The intensity of these sounds is *diminished* by simple dilatation of the ventricles, by fatty degeneration of the muscular fibres of the heart, or by deposition of fat between them or on the surface of the organ, by softening or debility of the muscular fibres as the result of protracted disease, for example, typhus or typhoid fever, and by pericardial effusions. It is also diminished by pulmonary emphysema. The heart sounds are sometimes masked by bronchial râles.

The *quality* of the heart sounds is considerably altered in a great variety of diseases. The sounds, instead of being clear and distinct, as in typical healthy cases, may be slightly muffled, or they may be associated with an indistinct and transient sound which closely resembles a

murmur. This impurity of the heart sounds, unless associated with other signs of cardiac disease, is of no diagnostic importance, because it very frequently occurs as the result of pulmonary disease when the heart is in no way involved, and it is often noticed in healthy individuals.

The first sound of the heart is rendered duller and lower in *pitch* than natural, by hypertrophy of the ventricles, with thickening of the tricuspid and mitral valves. The second sound is modified in the same way by thickening of the semi-lunar valves without regurgitation, and by loss of elasticity in the arterial walls.

The first sound of the heart is sharper and higher pitched than normal in dilatation of the ventricles without alteration of the auriculo-ventricular valves.

The second sound of the heart may be higher pitched than natural, or, in other words, accentuated, at either the aortic or the pulmonary orifice.

At the aortic orifice, this sound is somewhat intensified by hypertrophy of the left ventricle, due to obstruction in the artery. A ventricle thus hypertrophied propels the blood with increased force into the aorta, unduly distends this vessel, and thus causes sudden and more forcible contraction of the artery, with a sharper sound from the semi-lunar valves. Well-marked accentuation of the second sound in this position results from setting back, on the valves, of an increased volume of blood, and it is always caused by dilatation of the aorta.

Accentuation of the second sound at the pulmonary orifice occurs in a great variety of diseases. It is the most persistent of all the signs of cardiac disease, but it is also found in nearly every case of pulmonary congestion from whatever cause. Whenever there is obstruction or regurgitation at the mitral orifice, there must be increased tension of the blood in the left auricle and in the pulmonary veins, which will be transmitted through the short pulmonary circuit back to the pulmonary artery. This will cause a sudden and sharper closure of the valves which guard the outlet of the right ventricle. Obstruction in the pulmonary circuit from disease of the lungs, by inducing hypertrophy and dilatation of the right ventricle, causes extreme distention of the pulmonary artery with each pulsation, and consequent accentuation of the second sound in the pulmonary area.

The heart sounds become metallic or tinkling in quality in irritable conditions of the organ and when the stomach is distended with gas.

Exceptional.—The heart sounds are very metallic in character in the rare disease known as pneumo-pericardium. They are sometimes metallic in left-sided pneumothorax. The same character is sometimes noticed with the second sound, at the aortic orifice, when there is atheroma of this vessel limited to its initial portion.

The *seat* of the heart sounds is a limited space in which they can be

heard most distinctly. It may be altered by several diseases. The sounds obtainable over the apex are heard above their normal position, whenever the abdominal organs are so enlarged as to encroach upon the thoracic cavity, as in distention of the stomach, or enlargement of the liver, or ascites, or large ovarian tumors. They are also heard above their normal position when effusion is present in the pericardial sac.

These sounds are heard below their usual seat when the apex is depressed by mediastinal tumors, or by hypertrophy with dilatation of the auricles. They are displaced laterally by pleuritic effusions, pneumothorax, and by deformities of the chest. They are displaced to the left whenever the heart is enlarged, whether by hypertrophy or by dilatation, or when it is drawn from its position by contracting adhesions.

The *rhythm* of the heart sounds is altered by many diseases.

Frequently the heart acts regularly for some time, and then drops one or more beats to go on again with its regular pulsations. This is known as an *intermittent rhythm.*

If the intermittent rhythm includes the period of one pulsation only, it is of no special importance, as such phenomena occur under a variety of circumstances, independent of cardiac disease; it is a curious fact that intermission in the heart's action often occurs in some people just preceding a thunder-storm. But if this intermission occupies the time of two or three pulsations, and if the heart's action is irregular—that is, beating rapidly, then slowly, finally intermitting, and then starting up with rapid pulsations, as if to make up for lost time—it is a sign of cardiac disease.

The first sound of the heart is *prolonged* by hypertrophy of the ventricles, and by agglutination of the surfaces of the pericardium. It is *shortened* in dilatation of the ventricles, and both sounds are shortened by fatty degeneration and softening of the heart walls.

The period of repose is sometimes *prolonged* by obstruction to the onward flow of the blood into the left ventricle, owing to stenosis of the mitral orifice.

REDUPLICATION OF SOUNDS, another alteration of the rhythm, consists of a repetition of one or both of the heart sounds during a single pulsation, so that three or four sounds may be heard with each contraction of the heart. Ordinarily, the right and left sides of the heart contract at exactly the same time, and consequently the sounds which are produced in the two cannot be distinguished; but occasionally there is a slight interval between the closure of the valves at the auriculo-ventricular or at the arterial orifices of the two sides, so that the sounds do not occur simultaneously, and thus the first sound may be doubled, the second sound remaining natural; or the second sound may be doubled, the first remaining single; or both may be doubled.

This phenomenon occurs in diseases of the heart, but may often be discovered in health, if searched for with the differential stethoscope

(Fig. 15). When occurring in disease, reduplication is usually caused by stenosis of the mitral orifice or incompetence of its valves. This gives rise to increased tension in the pulmonary circuit and to abrupt closure of the pulmonary semilunar valves, which thus slightly anticipates the closure of the aortic valves, and causes reduplication of the second sound.

Reduplication of the first sound is due to tardy closure of the mitral valves. Some care will be necessary to avoid mistaking reduplication for endocardial murmurs which precede or follow the normal sounds. Intermission is a characteristic of reduplication (Loomis' Physical Diagnosis). In some cases reduplication is influenced by the acts of respiration. In forced or laborious respiration, the first sound may be reduplicated at the end of inspiration and at the beginning of expiration; the second sound may be reduplicated at the end of expiration and at the beginning of inspiration.

CHAPTER XII.

THE HEART.—*Continued.*

ABNORMAL SOUNDS—CARDIAC MURMURS.

THE abnormal sounds heard over the præcordial region are denominated murmurs. Sometimes these are produced upon the surface of the heart, between the two layers of the pericardium, but most of them originate within the heart. The latter are known as endocardial and the former as exocardial murmurs.

THE EXOCARDIAL OR PERICARDIAL FRICTION SOUNDS OR MURMURS are produced by the rubbing together of the roughened surfaces of the pericardium, in the same manner that friction sounds are produced within the pleura. These murmurs vary greatly in their intensity and quality. Sometimes they are very indistinct, at others loud. In quality, they may be grazing, grating, rubbing, creaking, or crackling, like pleuritic friction sounds.

The quality of an exocardial murmur yields no information regarding the peculiar condition of the surface which produces it, though, in the dry stage of pericarditis, the grazing sound is the one most likely to be heard.

These murmurs may be either single or double; that is, they may occur with the systole or with the diastole of the heart, or with both. They sometimes accompany the valvular sounds; at other times they are independent of them. They are always superficial in character and they seem to be produced immediately beneath the chest walls. The area over which they can be heard is restricted to the præcordial space. They are generally heard loudest at the junction of the fourth left costal cartilage with the sternum. They generally last for only a few hours, seldom longer than one or two days, and then disappear in consequence of the exudation of serum into the pericardium. As the serous effusion becomes absorbed in the later stage of pericarditis, the friction murmur may reappear.

Pericardial friction sounds are distinguished from endocardial murmurs: first, by their superficial character; second, by being limited to the præcordial space, *i.e.*, never being transmitted to the left of the apex, or above the base of the heart; third, by their being independent of valvular sounds; and fourth, by the variation in their intensity with changes in the position of the patient. When the patient is in the erect or in the

recumbent posture, the heart does not approach so near to the surface of the chest as when he is leaning well forward, and therefore the sounds are not as distinct. In general, the intensity is greater during expiration than during inspiration.

Pericardial friction sounds are distinguished from pleuritic friction sounds by their confinement to the præcordia, by their synchronism with the cardiac movements instead of the respiration, and by continuance during temporary suspension of the respiratory act.

Exceptional.—It should be remembered that, in some cases of pleurisy, rubbing of the fibrous layer of the pericardium against an inflamed pleura gives rise to a friction sound having the same rhythm as the heart, and continuing while respiration is suspended. Such a sign is called a *cardio-pleuritic friction murmur*. It is easily mistaken for the pericardial murmur, but its cause should always be suspected when other signs of pleurisy exist, especially if the pleurisy be associated with pneumonia. This sound differs from the pericardial murmur in the uniformity in intensity of the successive sounds, in its limitation to the border of the heart, and, in some cases to the end of inspiration, and in generally being affected to a greater or less degree by the movements of inspiration.

Endocardial murmurs vary in their intensity, pitch, and quality; but these elements are of very little importance from a diagnostic point of view, as the intensity and the pitch of the sounds yield us no information whatever, and the quality is never characteristic, except in the presystolic murmur due to stenosis of the mitral orifice.

These sounds are produced by changes in the physical condition of the heart, in which case they are known as organic murmurs; or by changes in the condition of the blood, when they are termed inorganic, anæmic, or hæmic murmurs.

Organic murmurs are usually permanent, though not infrequently they cease for a considerable length of time, and in some cases they may entirely disappear. The inorganic murmurs are transitory—present for a few hours or days and then disappearing permanently, or to recur after a short interval. Sometimes they come and go while the examination is being made.

A murmur in the præcordial space indicates nothing except a disturbance of the normal relations of the heart to the blood, and may be due to a change in the physical condition of the heart itself or in the normal composition of the blood, or it may result from irregular contractions of the cardiac muscle.

The important things to note regarding a murmur are: first, the seat; second, the rhythm; and third, the quality. The direction in which the sound is most clearly transmitted is also an essential feature in diagnosis.

In noting the rhythm, we observe the relation of the murmur to the systole and the diastole of the ventricles, and we ascertain whether it precedes, accompanies, or follows the first or second sound of the heart.

In a few instances, the peculiar quality of the sound itself is important. Some murmurs are grating, others blowing or rushing in quality, and others are harsh, or soft, or musical. A murmur may have many of these characteristics at different times without any appreciable change in the conditions which produce it.

Whenever we hear an abnormal sound in the præcordial space, we should ascertain, by careful examination, its point of maximum intensity, whether it is synchronous with either the contraction or the dilatation of the cardiac cavities and depends upon the current of blood through the valvular orifices, or whether it is produced outside the heart. As the majority of abnormal cardiac sounds are produced within the heart, the presumption is always that a murmur is endocardial; if we should find it comparatively deep seated, and synchronous with the systole or the diastole of the ventricles, and transmitted to the left of the apex, or above the base of the heart, we may safely conclude that it belongs to this class.

When we remember that nearly all endocardial murmurs are produced at one of the valvular orifices, and that these approximate so closely to each other that a circle half an inch in diameter may include a portion of each, it is at once apparent that it must be impossible to distinguish between different endocardial sounds by listening for them directly over their point of origin.

Sound loses its intensity by passing from one medium to another, as will occur in the passage of sound from one cavity of the heart to another, and any sound produced by fluid in motion is transmitted in the direction of the current which causes it. A knowledge of these two facts will aid us greatly in differentiating between endocardial sounds. We shall find that, as a rule, sounds produced in any of the cavities of the heart, or transmitted into them, are best heard over the space where that cavity is most superficial. For example, the only point at which the left ventricle impinges directly on the chest wall is where the apex beat is felt; murmurs produced at its auricular orifice are best heard at this spot, while those at the tricuspid orifice are most distinct over that portion of the right ventricle which is superficial. The murmurs at the aortic and pulmonary orifices are respectively heard with the greatest distinctness where these arteries approach nearest the chest wall.

Some of the endocardial murmurs, however, are produced by blood flowing in an abnormal direction. Therefore, the areas in which murmurs produced at the various orifices are most distinct will not always exactly correspond to the positions in which the normal sounds are loudest.

Before examining the heart by auscultation, we should ascertain its superior and lateral limits by percussion or by auscultatory percussion, and, either by these methods or by palpation, determine the position of the apex.

The *mitral area*, as the space is named where the mitral sounds may be heard with maximum intensity, corresponds to a circle two inches in diameter, which includes the apex of the heart (*A*, Fig. 33). If this organ is in its normal position, the circle, as shown in the diagram, will have its centre near the normal position of the apex beat; but if, from enlargement or other causes, the heart is displaced to the left, the position of this circle is correspondingly changed.

Mitral murmurs, if caused by regurgitation, are also heard diffused for a distance varying from one to three inches to the left of the apex. Often they may be heard behind, along the left side of the sixth and seventh dorsal vertebræ, with nearly the same intensity as in front;

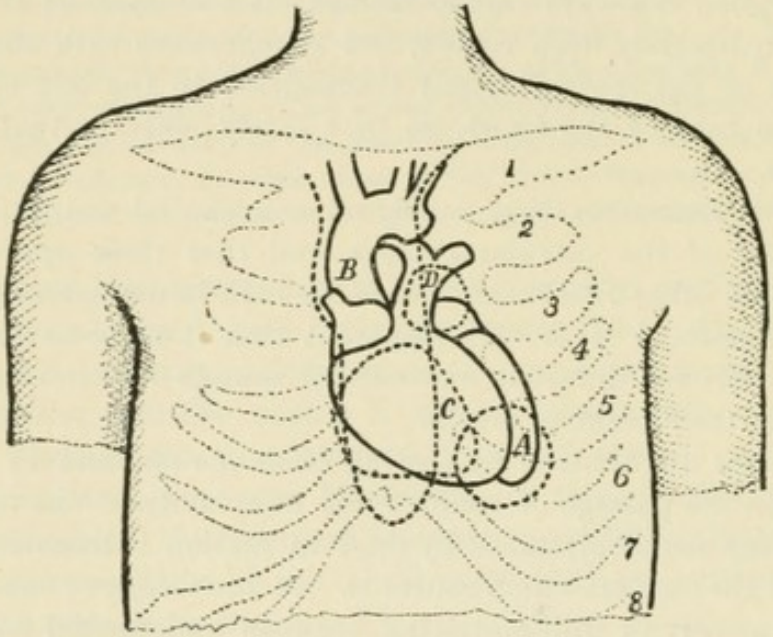

FIG. 33.—AREAS OF ENDOCARDIAL MURMURS. *A*, Mitral area; *B*, aortic area; *C*, tricuspid area; *D*, pulmonary area.

sometimes they may be heard in this position when they are not distinct in front.

Care must be taken not to confound mitral murmurs with aortic regurgitant murmurs, which are occasionally heard at the lower angle of the left scapula, and in the left axillary region; or with aneurismal murmurs, which may also be heard along the left side of the spinal column, in the same position as the mitral regurgitant murmur.

A mitral regurgitant murmur differs from an aneurismal murmur in being heard behind only between the fifth and the eighth dorsal vertebræ. The aneurismal murmur may be heard above the fifth vertebra, and, with diminished intensity, below the eighth as well as between the two.

An aortic direct murmur, heard behind, should not be mistaken for a mitral regurgitant murmur, since it is heard loudest above the lower border of the fifth dorsal vertebra.

Mitral regurgitant murmurs may sometimes disappear, even though due to organic lesions. In such cases, accentuation of the second sound at the pulmonary orifice may be the only abnormal sign remaining.

If a mitral murmur is obstructive, or direct, *i.e.*, due to stenosis of the mitral orifice, it will be heard at the apex, but will not be distinctly transmitted to the left, and will not be heard behind.

It is to be borne in mind that, in speaking of the areas of murmurs, we refer only to the positions at which they may be heard with the *greatest intensity*. Sometimes a mitral murmur may be heard over the whole præcordial region, or even over the entire chest, but its point of maximum intensity will correspond to the area which we have just described.

The *tricuspid area* of murmurs is limited to the triangular space (*C*, Fig. 33) where the right ventricle is superficial. These murmurs are ordinarily loudest over the xiphoid cartilage, or along the left border of the sternum, at the junction of the sixth or seventh costal cartilage, and are seldom audible above the third rib. This latter feature distinguishes them from aortic and pulmonic murmurs. When the heart is hypertrophied or dilated, their intensity will sometimes be greatest at the junction of the fourth costal cartilage with the sternum. These murmurs are superficial in character as compared with those occurring upon the left side of the heart. If transmitted in any direction, they will be heard more distinctly to the right than to the left of the parasternal line.

The *pulmonary area* of murmurs corresponds to a small circle about an inch in diameter, located just above the third costal cartilage at the left border of the sternum, and covering the pulmonary artery (*D*, Fig. 33). Pulmonic murmurs are heard most distinctly directly over the pulmonary artery. These sounds are never heard in the carotid and subclavian arteries. If due to regurgitation through the pulmonary valves into the right ventricle, they may be most intense, an inch or an inch and a half below this area, near the left margin of the sternum. They are not heard at the apex, and this distinguishes them from some aortic murmurs. These, like the tricuspid murmurs, are comparatively superficial.

The *aortic area* of murmurs cannot be so sharply defined as the areas of the murmurs we have just described. Aortic murmurs are usually loudest in the second intercostal space of the right side, where the artery approaches most closely to the thoracic walls; or along the right margin of the sternum from the second to the fourth rib; but they are often heard over the whole sternum (*B*, Fig. 33).

Aortic murmurs are propagated to the carotid or subclavian arteries, and are frequently heard best in these localities. Occasionally they are louder in the pulmonary area than at any other point. In such instances they are distinguished from pulmonary murmurs by being heard also in the arteries at the base of the neck. Aortic murmurs are often heard behind, along the left side of the third and fourth dorsal vertebræ, and with diminishing intensity for a considerable distance down the spine. They are frequently very distinct at the apex of the heart.

Aortic regurgitant murmurs are often loudest over the lower part of the sternum, though we expect to find them most distinct a short distance below the aortic valves. These murmurs are frequently audible in the left axillary region, and at the lower angle of the scapula. The patient may often hear them himself, especially when lying down.

Exceptional.—Aortic murmurs may sometimes be heard over the arteries when they are not distinct at the base of the heart. At other times they are audible at the base of the heart only; and still again, they may be distinct over the entire præcordial region.

Regurgitant aortic murmurs are frequently heard in all the arteries which are accessible to auscultation. It should be remembered that the aortic murmurs are the only ones that may be heard above the clavicles.

Both the obstructive and the regurgitant aortic murmurs vary much in intensity. Sometimes it is necessary to listen intently in order to hear them at all. In other cases they are so loud that they may be heard at some distance from the patient.

The *rhythm* of a murmur refers to the relation which it bears to the cardiac pulsation, and consequently to the first and second sounds of the heart. In determining the rhythm of a murmur, we must first ascertain which is the first and which the second sound of the heart. This will not be a difficult task if the heart is pulsating slowly and both sounds are distinct; for we know that the first sound is the louder and longer, and that it is associated with the impulse of the apex against the chest wall. In some instances only one of the valvular sounds can be heard at the apex or at the base, and in such cases a murmur would very naturally be mistaken for the other sound. In every case of doubt we must feel for the carotid pulse, which is always synchronous with the first sound of the heart, and will therefore enable us to determine the rhythm of the murmur.

The *quality* of endocardial murmurs gives us no information regarding their place of origin or the conditions which produce them, excepting in cases of presystolic mitral murmurs, which will be presently described, and anæmic murmurs, which are always soft in character.

Causes of Endocardial Murmurs.—Presystolic mitral and tricuspid murmurs, preceding as they do the first sound of the heart, must occur while the blood is passing from the auricles into the ventricles, and while the valves are thrown out upon the current (Fig. 34). They are always caused by narrowing (stenosis) of the auriculo-ventricular orifice, which obstructs the onward flow of blood. Such a murmur, if produced upon the left side, will be loudest at the apex, but will not be transmitted to the left of the apex, and cannot be heard behind. It is called a mitral presystolic or obstructive murmur. This is perhaps the only murmur where the quality of the sound is of any special diagnostic value. According to Balfour, the quality of these murmurs is characteristic, though not exactly the same in all cases. It may be quite ac-

curately represented by vocalizing the symbols R r r b or V o o t. If a murmur which precedes the first sound of the heart is produced upon the right side—which is extremely uncommon—it is called a tricuspid obstructive murmur, and its area is limited to the triangular space *C*, at the lower portion of the sternum (Fig. 33).

Systolic murmurs, or murmurs accompanying or following the first sound of the heart, must occur with the contraction of the ventricles, the closure of the auriculo-ventricular valves, and the propulsion of the blood from the ventricles into the arteries. They may be due to lesions at any of the valvular orifices.

The mitral, systolic or regurgitant, murmur is produced at the mitral orifice, and is due to thickening, corrugation, or adhesion of the valves, which prevents them from perfectly closing the orifice, and thus allows

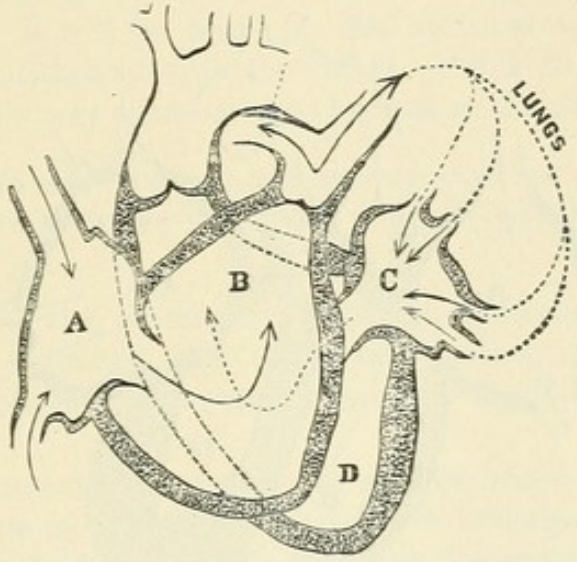

FIG. 34.—AURICULAR SYSTOLE. *A*, *C*, Contracted auricles; *B*, *D*, dilated ventricles. Mitral and tricuspid valves open; semilunar valves closed.

the blood to regurgitate into the left auricle. It may also result from rupture or undue shortening or stretching of the columnæ carneæ or their tendons, which normally keep the valves from giving way before the column of blood. This murmur is generally soft and blowing, and may be musical in quality; it will be loudest in the mitral area. It will be transmitted to the left of the apex, and may be heard posteriorly along the left side of the spinal column from the fifth to the eighth dorsal vertebra. It is seldom heard in this situation with the same intensity as at the apex, but occasionally it is distinct behind when it is not audible in front. If a mitral murmur is caused simply by roughening of the ventricular surface of the valves, it will not be heard beside the sixth or seventh dorsal vertebra, though it may be heard about the inferior angle of the scapula, and in the left axillary region.

Sometimes endocardial murmurs are produced by dilatation of the ventricles, which prevents perfect closure of the mitral valves. Such

murmurs have been termed *curable mitral regurgitant murmurs,* as they disappear when the tonicity of the muscular fibre has become sufficiently restored to contract the cavities to their original size. These murmurs are probably caused by dilatation of the ventricles without a corresponding elongation of the musculi papillares in consequence of which the chordæ tendineæ are too short to allow the valves to close. The tricuspid systolic, or regurgitant, murmur will be heard in the tricuspid area, and if transmitted in either direction will be louder to the right than to the left. It will not be heard at the apex distinctly, and never to the left of the apex or behind. This murmur has generally a blowing quality.

If the aortic systolic, obstructive or direct, murmur is of organic origin, it will be caused by constriction of the aortic semilunar valves,

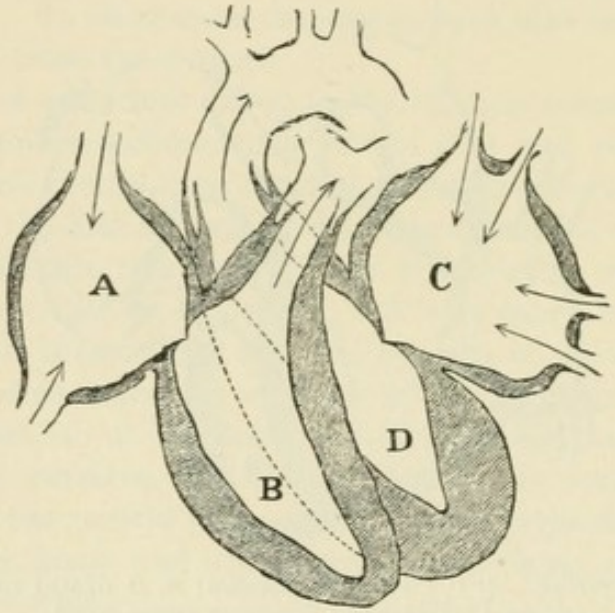

FIG. 35.—SYSTOLE OF THE VENTRICLES. *A, C,* Auricles dilating; *B, D,* ventricles contracting. Semilunar valves open ; mitral and tricuspid valves closed.

or by roughening of their ventricular surfaces, or possibly by disease of the artery. It will be produced while the blood is passing from the ventricles into the arteries (Fig. 35), and will be heard in the aortic area over the second intercostal space of the right side, or over other portions of the sternum as shown by the space *B* (Fig. 33). It will also be heard in the arteries of the neck, and frequently at the left of the third and fourth dorsal vertebræ posteriorly and possibly with diminished intensity farther down the spine.

If this murmur is loudest over the pulmonary artery, as occasionally happens, it may be distinguished, from murmurs produced at the pulmonary orifice, by the fact that it is transmitted to the carotid and subclavian arteries.

A systolic murmur produced at the pulmonary orifice is likely to be a hæmic murmur; but if of organic origin, it is usually due to obstruc-

tion similar to that just described as occurring at the aortic valves. These murmurs are sometimes caused by pressure on the artery from enlarged glands; or by constriction of the artery from pleuritic adhesions, or fibroid phthisis with contraction of the lung. Such a murmur will be heard most distinctly in the pulmonary area (*D*, Fig. 33), and will not be audible in the arteries at the base of the neck.

Diastolic Murmurs.—A murmur accompanying or following the second sound of the heart occurs with the diastole of the ventricles, and must be due to regurgitation of blood from the arteries through the semilunar valves, on either the right or the left side.

If a murmur, accompanying or following the second sound of the heart, occurs at the aortic orifice, it will be due to regurgitation of blood from the artery into the left ventricle, and may be called aortic diastolic or regurgitant. It will generally be soft and blowing in character, though it may be harsh. It will be heard in the aortic area, but usually most distinctly a short distance below the valves; it will be propagated down the sternum and it may sometimes be loudest at the ensiform appendix.

Exceptional.—In some instances such murmurs are very distinct at the apex, in the axillary region about the lower angle of the left scapula, or over all large superficial arteries.

If produced at the pulmonary orifice, a diastolic murmur is due to regurgitation through the pulmonary valves, and is called a pulmonary diastolic or regurgitant murmur. Such murmurs are extremely rare.

When such a murmur does occur, it will be heard in the pulmonary area, or an inch or an inch and a half below this space, and it will not be transmitted to the large arteries or to the lower part of the sternum. By this latter fact it may easily be distinguished from a similar murmur at the aortic orifice.

Thus, we may have eight distinct valvular murmurs, four of which are obstructive and four regurgitant. Two of these, viz., the regurgitant pulmonary and the obstructive tricuspid murmurs, are so very rare that their existence is doubted by many skilled diagnosticians. Regurgitant tricuspid murmurs are rare except as the consequence of disease of the left side of the heart, which gives rise to such dilatation of the right ventricle that the auriculo-ventricular orifice becomes too large to be closed by the tricuspid valves.

We may have two or more of these sounds combined in any case; thus, it is not uncommon to obtain a mitral regurgitant murmur associated with an aortic obstructive, and perhaps also with an aortic regurgitant murmur; or we may have both the mitral obstructive and regurgitant, with the aortic obstructive and regurgitant murmur.

Murmurs are common in the left side of the heart, but rare in the right side.

According to my observation, the various murmurs occur in the following order of frequency: mitral regurgitant, aortic regurgitant, aortic obstructive, mitral obstructive or presystolic, and tricuspid regurgitant.

VENTRICULAR MURMURS.—There are certain murmurs occasionally heard in the præcordial region, which are neither of valvular nor of hæmic origin. They are most frequent during the acute stage of endocarditis, but they also occur in chronic endocarditis. They sometimes precede and sometimes follow endocarditis, and in some instances they are apparently induced by simple irritability of the heart. They accompany the first sound of the heart, and are loudest at the apex. These murmurs seem to be caused by roughening of the endocardium or of the chordæ tendineæ, or by irregular contraction of the muscular fibres of the ventricles. They are of comparatively rare occurrence, and then are usually mistaken for valvular murmurs. They may be distinguished from the latter by their rhythm and by their seat. These murmurs are most likely to be confounded with mitral regurgitant and aortic or pulmonary obstructive murmurs.

A ventricular murmur, though heard at the apex with the first sound of the heart, is *never transmitted to the left;* whereby it is distinguished from the mitral regurgitant murmur, which possesses the same rhythm. A ventricular murmur is *never heard above the base of the heart,* and thus is distinguished from aortic and pulmonary murmurs.

Frequently in examination of the heart, *impure sounds* are obtained, which closely resemble faint valvular murmurs. They are generally heard just at the end of inspiration, and usually cease when respiration is suspended. These are not constant, but may come and go during the examination.

Congenital murmurs arise from imperfect closure of the foramen ovale, which allows the blood to pass directly from the right into the left auricle. This occasions a murmur which is audible over the base of the heart. It is heard with the systole of the ventricles, and is not transmitted into the arteries, or to the left of the apex. It may thus be distinguished from aortic and mitral murmurs. This murmur always occurs in early life, and is associated with a cyanotic appearance of the countenance. When the child reaches the age of ten or twelve years, other endocardial murmurs usually supervene.

Hæmic murmurs form another variety of adventitious sounds due to changes in the composition of the blood instead of to anatomical changes in the heart. They are also termed anæmic, or inorganic murmurs. They are always systolic, generally most distinct over the aorta, and are diffused through the vessels of the neck. Sometimes they may be heard in the second intercostal space of the left side, about an inch and a half to the left of the pulmonary artery.

The hæmic murmurs which are produced in the aorta are due simply to change in the composition of the blood. Those heard to the left of

the pulmonary artery seem to be produced by slight dilatation of the left ventricle, with consequent imperfect closure of the mitral valve, and more or less regurgitation of blood into the auricle.

These murmurs are inconstant; they often come and go during the examination, and finally they permanently disappear as proper treatment removes the anæmic condition of the blood.

The following characteristics distinguish them from organic murmurs: they always accompany the first sound of the heart; they are soft and blowing in character; those which are arterial may be heard over many of the aortic branches and are often loudest over the carotids instead of over the aorta, where the aortic obstructive murmurs would be most distinct. Those which are mitral may be heard a variable distance to the left of the pulmonary artery. They are inconstant and likely to be present when the heart's action is rapid, but absent when it is slow. They are incapable of supplanting the normal heart sounds, or even of making them less distinct, and are usually associated with the venous hum.

These murmurs are also attended by the symptoms and signs of general anæmia. Except in complicated cases, they are not associated with the signs of other cardiac disease.

ANOMALOUS HEART SOUNDS.

In rare instances, sounds may be heard over the præcordial space, which are not endocardial or pericardial. These result from the action of the heart upon the lungs, and usually cease when the respirations are suspended.

With the systole of the ventricles, a loud blowing sound may be occasioned by a large pulmonary cavity situated near the heart. More or less distinct blowing sounds are frequently heard when the systole of the heart occurs just at the end of inspiration. These cease when the patient holds his breath.

Friction sounds may be produced by the action of the heart upon the overlying pleura. Generally these may be easily distinguished from pericardial friction sounds by their seat, and by their disappearance with the cessation of respiration. The pericardial friction sounds are heard most distinctly along the left border of the sternum; but sounds produced within the pleura by the action of the heart are heard most clearly over the outer portion of the mammary region. They are also usually associated with friction sounds over other portions of the left lung. Ordinary pleuritic friction sounds are sometimes observed in the præcordial region; but these disappear when the patient holds his breath.

The sounds caused by the action of the heart upon the lungs occasionally resemble bronchial râles; but as these are limited to the præcordial space, they are not likely to be mistaken for sounds due to pulmonary disease.

SUBCLAVIAN MURMURS.

Subclavian murmurs are often heard just beneath the clavicle, at the outer portion of the infra-clavicular region, more frequently upon the left than upon the right side. Most of these seem to me to be produced by the pressure of the stethoscope; but murmurs frequently occur in this locality, and over other parts of the subclavian artery, which are not due to external causes. They are supposed to result from pressure upon the artery, either by consolidated lung tissue or by cicatricial bands resulting from pleurisy; but their exact cause is not known. They are most frequently associated with consolidation of the apex of the lung.

VENOUS SIGNS.

TURGESCENCE of the superficial veins of the neck and upper part of the trunk is a sign of cardiac or pulmonary disease, and of aortic aneurism or other intra-thoracic tumors. The condition is caused by direct pressure on the veins, or by increase in the intra-thoracic pressure from pulmonary disease, and consequent interference with the return of blood to the heart. It is always most noticeable when the patient is in the recumbent position.

This turgescence may be either temporary or permanent. If the former, it is most marked in expiration or after attacks of coughing, and it will entirely disappear upon deep inspiration.

Temporary turgescence of these veins is generally due to congestion of the pulmonary circuit, resulting from disease of the lungs, which compresses the capillaries, and consequently causes distention of the pulmonary arteries and of the right side of the heart, and, through it, of the descending vena cava and its branches.

Permanent turgescence most commonly results from disease of the mitral valves, which either obstructs the onward current of blood into the left ventricle or allows free regurgitation into the auricle. This gives rise to engorgement of the pulmonary circuit, which cannot be relieved by deep inspiration. Permanent congestion may be due to obstruction of the descending vena cava by a thrombus, or more frequently by the pressure of an aneurism or other tumor.

Localized turgescence, confined to a single vein and its branches, is always the result of a thrombus, an embolus, or of pressure upon the blood-vessel.

VENOUS PULSATION with marked pulsation in the jugular veins is observed when there is permanent engorgement of the descending vena cava, which generally results from extreme emphysema or stenosis of the mitral valves with secondary tricuspid regurgitation.

Pulsation in the jugular veins is usually observed just above the

clavicles, though sometimes it extends over the whole course of the vessel. It is most marked in the dorsal decubitus, and is more distinct upon the right than upon the left side, because the current of blood from the right ventricle, through the auricle, finds its way more readily into the veins of that side.

Venous pulsation may precede the impulse of the apex and the first sound of the heart, or may follow it. In other words, it may be either presystolic or systolic.

Presystolic venous pulsation is due to regurgitation of blood into the veins during the contraction of the auricles.

Systolic venous pulsation is due to contraction of the right ventricle with regurgitation of blood through the tricuspid valves into the auricle and thence into the veins. When slight and temporary, this is termed relative venous pulsation; when permanent, it is known as absolute venous pulsation. In order to be of value in the diagnosis of tricuspid regurgitation, it must be visible during both inspiration and expiration.

Pulsation of the jugular veins may be simply the transmitted impulse from the carotids. In such cases, there will be simply a lifting impulse, instead of expansion of the blood-vessel, and the vein will not be tortuous as in true venous pulsation.

Pulsation in the veins on the back of the hands has been repeatedly noticed by Peter, of Paris, in advanced consumption, and occasionally in other affections. It is increased by compressing the wrist, and therefore must be propagated through the capillaries from the left side of the heart. It may be more readily seen than felt.

Peter thinks this phenomenon due to paralysis of the muscular fibres of the arteries, through excess of carbonic acid in the blood. This rare phenomenon, when seen, indicates the near approach of death.

COLLAPSE OF THE JUGULAR VEINS is said to occur with the systole of the ventricles, in some cases, where there is agglutination of the two surfaces of the pericardium.

THE VENOUS MURMUR, venous hum or *bruit de diable* is a constant humming sound frequently obtained over the jugular vein just above the clavicle, or in the inter-clavicular notch. It is generally associated with an arterial hæmic murmur. It occasionally occurs in healthy persons, but is most often found in those who are anæmic, especially in chlorotic women.

This sign is most apt to be heard when the patient is sitting or standing, and is usually soft and humming in character, but occasionally musical, hissing, or even loud and roaring

Intermittent venous murmurs synchronous with the pulsations of the heart, are among the rarest signs of cardiac disease. They may be presystolic, systolic, or diastolic. The presystolic murmurs are heard only when the patient is lying down, and must result from regurgitation of blood from the right auricle into the open veins. The systolic murmur

is usually heard most distinctly just above the clavicle on the right side. It is due to regurgitation from the right ventricle through the auricle and into the veins. The diastolic murmur is extremely rare. It is said to require, for its production, hypertrophy and dilatation of the heart, with aneurism. These murmurs may be mistaken for arterial murmurs. They may be distinguished from the latter by slightly pressing on the blood-vessel, which will prevent the venous hum, but will not so affect the arterial murmur.

THE SPHYGMOGRAPH.

By the use of the sphygmograph we are enabled to obtain a graphic statement of the condition of the circulatory system, written, as it were,

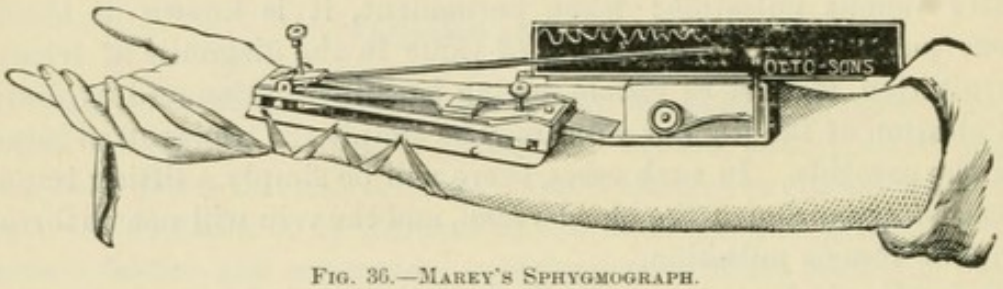

FIG. 36.—MAREY'S SPHYGMOGRAPH.

by the heart itself. When all the conditions are favorable, this statement furnishes interesting information to physiologists; but so much depends upon the adjustment of the instrument, its proper working,

FIG. 37.—NORMAL RADIAL PULSE (FOSTER)

and the pressure made upon the artery that up to the present time the instrument has been of little clinical value. When all the conditions are perfect, the tracings of the pulse may indicate: the time occupied by the systole and the diastole of the heart; the force of the heart's contraction; the resistance to the onward current of blood, or its regurgitation through the valves, and the tension of the arteries.

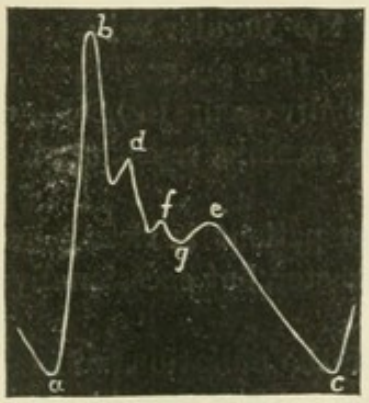

FIG. 38.—NORMAL RADIAL PULSE, SINGLE TRACE ENLARGED.

The tracing is composed of a series of curves, each of which represents a cardiac pulsation.

In the tracing of the normal radial pulse as shown (Figs. 37 and 38) each completed series consists of a line of ascent, a summit, and a line of descent. The line of ascent *a b* in the normal condition is perpendicular to the plane of the base. It is produced as the blood is propelled into the artery, and indicates the force of the heart by its height, and

the rapidity of the current of blood, by its direction. When the blood is retarded in its passage from the left ventricle into the aorta, as in

FIG. 39.—AORTIC OBSTRUCTION (HAYDEN).

constriction at the aortic orifice, this line will run more or less obliquely to the right, according to the amount of obstruction (Figs. 39 and 40).

FIG. 40.—AORTIC OBSTRUCTION (FOSTER).

When the pulsation is forcible, the altitude is much greater than when it is weak. The summit *b* (Fig. 37) in the normal condition a mere

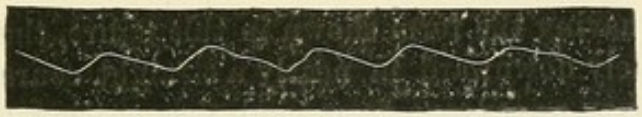

FIG. 41.—MITRAL REGURGITATION.

point, is reached at the instant when the artery is most fully distended, immediately after the systole of the left ventricle. When the vessel is

RIGHT ARM.

LEFT ARM.

FIG. 42.—ANEURISM OF ASCENDING AORTA (LOOMIS).

incompletely filled the summit is rounded, or the line of descent may run almost horizontally for a short distance. Examples of this are

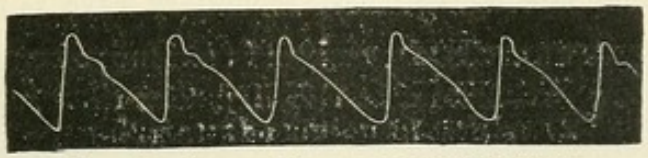

FIG. 43.—AORTIC REGURGITATION (BOILEAU).

found in mitral regurgitation (Fig. 41), or when the artery is partially occluded by an aneurism (Fig. 42), and when free regurgitation through

FIG. 44.—AORTIC OBSTRUCTION AND REGURGITATION (LOOMIS).

the aortic valves prevents full distention of the artery (Figs. 43 and 44). The line of descent *b c* (Fig. 37) corresponds to the period of arterial

systole and cardiac diastole. The length of the line indicates the rapidity of the heart's action. When the heart is beating rapidly, the line is short; when beating slowly, the line is correspondingly lengthened. The undulations in this line *d e f* (Fig. 37) are known as the first, second, and third secondary waves. The first secondary wave *d* is produced by the natural contraction of the artery. The second wave *e* corresponds to the impulse occasionally felt, which is termed *dicrotism*. The third

FIG. 45.—INCIPIENT HYPERTROPHY FROM OBSTRUCTION IN THE ARTERIOLES, DUE TO BRIGHT'S DISEASE OF THE KIDNEYS.

wave *f* is not often present. The depression *g* marks the complete closure of the aortic valves. A small notch in the line of descent is often seen near the summit.

Instead of having the form shown in this figure, the line of descent may run obliquely downward in nearly a straight course. It may have a generally convex or concave form, and the position of the secondary waves may vary in distance from the points *b* and *c*.

Convexity of the line of descent or small secondary waves (Fig. 45)

FIG. 46.—SENILE PULSE (FOSTER).

are due to increased arterial tension, as when there is incipient hypertrophy of the heart in consequence of contraction of the arterioles in Bright's disease.

Concavity of the line of descent is due to diminished arterial tension.

Sudden dropping of the line of descent indicates aortic regurgitation (Fig. 43).

In the normal tracing, the first secondary wave is found on a level

FIG. 47.—MITRAL CONSTRICTION (HAYDEN).

with the junction of the middle with the upper third of the line of ascent; but with loss of elasticity of the artery it occurs nearer the summit, as in the senile pulse (Fig. 46). The same condition of the artery is indicated by absence of dicrotism.

In mitral stenosis or constriction, the line of ascent is oblique, the summit rounded, the line of descent prolonged, and the secondary waves are absent or indistinct.

From what has been said, we learn that the sphygmographic tracing is not diagnostic of any disease, as will be at once apparent in looking over the tracings taken in different cases of the same disease (Figs. 39 and 40, 43 and 44); but the general appearance of the curve may indicate special conditions. The special points to notice in the tracing are:

FIG. 48.—MITRAL CONSTRICTION AND TRICUSPID REGURGITATION (HAYDEN).

the height and the obliquity of the line of ascent; the acuteness or rotundity of the summit; the length of the line of descent; the convexity of the line of descent; and the nearness to the summit of the secondary waves.

Sanderson considered this instrument principally useful in detecting

FIG. 49.—HYPERTROPHY AND DILATATION OF THE HEART (HAYDEN). High line of ascent; sudden falling of line of descent.

increased arterial tension consequent upon hypertrophy of the left ventricle (Fig. 45).

Francis E. Anstie thought that when the instrument worked perfectly, if accurately adjusted, it would be of value in the diagnosis, not only of commencing hypertrophy of the heart, but also of aortic regurgitation (Fig. 43), and especially of aneurism of the aorta (Fig. 44).

CHAPTER XIII.

CARDIAC AND ARTERIAL DISEASES.

PERICARDITIS.

PERICARDITIS is an inflammation of the pericardium, acute, subacute, or chronic, usually associated with myocarditis or endocarditis or both.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—Acute pericarditis, like inflammation of the pleura, is characterized by dryness and reddening from hyperæmia of the subserous vessels,and by infiltration and swelling of the serous and subserous tissues. This is followed by desquamation of the endothelium, loss of the normal glistening character, and the appearance of a highly albuminous exudate upon the surface of the membrane (pericarditis fibrinosa). This is usually localized at first, but becomes more widely spread by the cardiac motion, and later assumes a roughened, shaggy aspect (hairy heart). The inflammatory lymph may cover the entire surface of the pericardium, but is apt to be confined to the upper part.

In the acute form of the disease, serum is usually effused in small amount. It sometimes becomes enclosed in pockets formed by adhesions, but is sooner or later absorbed. The opposite walls may become permanently adherent by organization of the exudate into fibrous bands which connect the two surfaces, or the cavity may be obliterated by complete adhesion of the two surfaces. The pericardium itself is more or less thickened. In subacute inflammation, the effusion of serum becomes abundant (pericarditis serosa), its appearance and quality varying with the amount of serum, fibrin, red and white corpuscles present. The pericardial sac, when greatly distended, assumes a pyramidal form, its base downward, its apex at the base of the heart, and in enlarging it encroaches upon the lungs and diaphragm.

Milk Spots.—Frequently opaque, yellowish or gray raised and sharply defined patches termed milk spots are found on the surface of the pericardium, otherwise normal. They are due to hyperplasia and increased density of its fibrous elements, and probably arise from friction of an enlarged heart against neighboring parts (Hamilton, Text-Book of Pathology, page 558). Extravasation of blood into the sac, with the fibrin and serum, characterizes the hemorrhagic variety of pericarditis, commonly associated with cancer scorbutus or purpura.

In the purulent form, or pericarditis purosa, bacteria are found in the yellow or greenish fluid which may have been purulent from the first or have become so secondarily.

Chronic pericarditis is usually consecutive to the acute form, and often presents, in addition to the adhesion and fibrous bands, extensive thickening and calcareous deposits. Extension of the inflammation may result in myocarditis with weakening, atrophy or fatty degeneration of the heart muscle, followed by dilatation of the cavities. The walls may undergo compensatory hypertrophy; extreme dilatation of limited portions of the ventricular wall constitutes what is termed cardiac aneurism.

ETIOLOGY.—Acute rheumatism is the most common cause of pericarditis, as of endocarditis and myocarditis, hence their frequent coexistence.

Other not infrequent antecedent disorders are Bright's disease, alcoholism, syphilis, tuberculosis, typhoid fever, and acute infectious disease; also cancer, purpura, pernicious anæmia, and scorbutus, which produce the hemorrhagic form. In early life the exanthemata often cause this affection. It may also arise from penetrating wounds, severe contusions, and by extension of inflammation from neighboring parts; occasionally no cause can be detected.

SYMPTOMATOLOGY.—The affection may be divided into three stages similar to those of pleurisy—a dry stage, a stage of effusion, and a stage of absorption.

The most common symptoms are: pain in the præcordial and epigastric regions, shooting to the shoulder, and augmented by movements or by pressure; more or less fever, the temperature rising from one to four degrees; but in fatal cases sometimes falling again shortly before death; a small, wiry, irregular pulse, running from 90 to 120 beats per minute; œdema, dyspnœa, and occasionally dysphagia. Any or all of these symptoms may be absent; usually there is a history of coincident or preceding rheumatism.

The essential *signs* in the order of their occurrence are: irritable action of the heart; friction fremitus and murmur; increased cardiac dulness, ultimately obtained over a triangular area extending considerably to the left of the apex; feebleness of the heart's impulse and sounds, both of which are intensified when the patient leans well forward.

In the first stage, upon inspection and palpation, we discover nothing except an irritable action of the heart, with slightly increased force, and, in the latter part of the first stage, friction fremitus.

Upon auscultation, a grazing friction sound may sometimes be heard very early in the disease along the left border of the sternum, usually most distinct at the fourth costo-sternal junction. This sound may be distinguished from endocardial murmurs by its rhythm and seat, and by the fact that its intensity is increased by pressure and by a full inspiration. In the latter part of this stage, friction sounds of a harsher quality may be obtained. These may be either feeble or very intense.

In the second stage of the disease, the signs vary somewhat with the amount of effusion.

On inspection in children and young adults, with elastic chest walls, bulging of the præcordial region, extending from the second to the sixth rib, may be noticed. The respiratory movements of the left lung are somewhat impeded, and the apex beat is carried upward and to the left into the fourth intercostal space.

Palpation confirms the signs obtained by inspection. The impulse of the heart is feeble, especially when the patient is lying upon his back; but when he is leaning forward, it is much more forcible than in either the erect or the recumbent position. This is an important fact in the diagnosis. When the pericardium is greatly distended, the diaphragm may be forced downward, so as to cause bulging in the epigastric region. Undulation of the whole præcordial region, due to the action of the heart upon the surrounding fluid, may frequently be felt, and occasionally fluctuation can be detected.

Upon percussion, both the superficial and the deep-seated areas of dulness are increased. At first the latter is increased in its vertical diameter, and dulness is noticeable principally above the base of the heart in the second intercostal space, where the serum first collects. This is especially marked when the person is in the recumbent posture.

Von Stoffella, of Vienna, has noticed in these cases a dulness over the base of the heart in recumbency change to resonance when the patient sits up (*Internationale klinische Rundschau*, Feb., 1890).

When the effusion becomes somewhat greater, serum collects at the lower part of the pericardial sac; dulness is then increased in the transverse diameter at the level of the apex, and the area of dulness becomes triangular with its base downward, corresponding to the form of the pericardium. This triangular shape remains, however great the effusion may be. In extensive effusion, the dulness may extend from the first rib above to the resonance of the stomach below, and laterally from the right nipple to a point about two inches beyond the left nipple.

E. Pins, in well-marked cases, has frequently observed, when the patient is recumbent or sitting, a small area on the left side posteriorly, over which there is dulness with bronchial breathing and increased vocal resonance, but no râles or friction sounds (*Wiener medizinische Presse*, March, 1890).

This is most marked in a circular space the size of a silver dollar, extending from a point about three fingers' breadth below the angle of the scapula to within two of the lower margin of the lung. If the patient bends forward, and especially if he assumes the knee-elbow posture, dulness largely disappears, vesicular resonance taking the place of abnormal sounds. These phenomena are probably due to pressure upon the lung, which is relieved by a forward displacement incident to change in posture.

The position of the apex beat having been determined by palpation or auscultation, the existence of dulness to the left of this point and

below it becomes an important element in distinguishing pericarditis from enlargement of the heart; in the latter the apex beat corresponds very nearly to the limit of dulness on the left.

In the differential diagnosis of pericardial effusions, T. M. Rotch, of Boston, considers an area of flatness in the fifth intercostal space of the right side, about an inch from the border of the sternum, a very important sign.

The friction sounds usually heard on auscultation in the first stage generally disappear when effusion occurs, in consequence of the separation of the pericardial surfaces; yet they may remain at the base of the heart throughout the disease. In the second stage, the heart sounds are feeble and distant, but may be rendered more distinct by causing the patient to lean well forward; sometimes friction sounds may be reproduced by this means.

Pulmonary sounds are not heard over the area of flatness in the præcordial region.

In the third stage, the signs of the second stage disappear, the bulging gradually diminishes, the apex beat becomes more and more perceptible and returns to its normal position; there is a gradual diminution in the area of dulness; friction sounds may return and remain until resolution has taken place, or until the two surfaces of the pericardium have become adherent; the respiratory sounds may again be heard in the præcordia.

Exceptional.—Occasionally friction sounds continue long after apparent recovery.

We have no means of determining when adhesions of the pericardial surface have taken place unless the external layer of the sac has also adhered to the chest walls. When this has occurred, the intercostal spaces are seen to be depressed with each systole of the ventricles, and ultimately permanent depression of the præcordial region may take place. In some cases, when the heart is considerably hypertrophied and dilated, dragging in of the epigastric region is caused by each pulsation of the heart.

DIAGNOSIS.—Pericarditis is liable to be mistaken for pleurisy or endocarditis or for mediastinal tumors.

The first stage of *pleurisy* causes pain and friction sounds similar to those of pericarditis, and, if it happen to involve only the anterior portion of the left pleura, considerable care will be necessary to avoid an error in diagnosis. The distinctive features between the two affections are presented in the following table:

PERICARDITIS.	PLEURISY.
History.	
Commonly of rheumatic origin.	Non-rheumatic.

PERICARDITIS.	PLEURISY.
Symptoms.	
Pain usually in the præcordial region.	Pain usually in the infra-axillary region.
Signs.	
Friction sounds confined to the region of the heart and synchronous with its movements, and not affected by the respiratory movements.	Friction sounds, though they may be confined to the præcordial region, are generally heard farther to the left. They are not synchronous with the pulsations of the heart, but occur with the respiratory movements, and almost invariably cease when respiration is suspended.

Symptoms due to pressure by *mediastinal tumors* on vessels or nerves or bronchi are prominent; not so in pericarditis. There is also accompanying enlargement of the glands of the neck, and absence of some of the symptoms and signs of inflammation which characterize pericarditis. Malignant growths also cause marked and peculiar cachexia and have no history of rheumatism.

For the distinctive features between *endocarditis* and inflammation of the pericardium, see endocarditis.

PROGNOSIS.—Acute rheumatic pericarditis usually ends in resolution within three weeks, very rarely in death. It may, however, become subacute or chronic.

Adhesive obliteration of the pericardial sac tends to weaken the heart muscles, and, if associated with a crippled condition of the valves, is unfavorable; usually such adhesions result in cardiac hypertrophy. Slight adhesions always remain but are of little significance.

Fluid effusion is absorbed in most cases in ten to fifteen days, but large pericardial effusion may cause sudden death, or by long-continued embarrassment of the heart's activity give rise to atrophy or fatty degeneration and consequent danger of sudden death from pulmonary œdema or cardiac paralysis on slight over-exertion. Purulent and hemorrhagic pericarditis are always dangerous. Pericarditis accompanying nephritis is serious.

TREATMENT.—With the first symptoms of pericarditis, the patient should be put to bed, to remain absolutely quiet until convalescence has been established. Hot poultices should be kept constantly applied to the whole anterior surface of the chest. Opiates should be given in just sufficient quantity to control pain. Depressing measures of all kinds must be avoided.

If the cause of the disease can be ascertained, it should be removed. Rheumatism will call for alkalies, guaiacum, or small doses of colchicum. The latter must not be given in doses sufficient to derange digestion or cause depression. Salicylic acid should not be given on account

of its depressing effects on the heart, but the salicylates are less objectionable. If this affection follow depressing fevers, the supporting measures which are required for the latter should be more assiduously applied. If it result from Bright's disease, saline cathartics in moderate doses, diaphoretics, especially vapor or hot-air baths, dry cupping over the loins, and small doses of digitalis will be indicated. In most cases, iron is a necessary remedy, and quinine will usually be beneficial in maintaining strength.

The diet should be concentrated and nutritious, and, so far as possible, fluids should be avoided. If effusion takes place, its removal will be favored more by the means calculated to maintain the strength than by the various drastic cathartics so often prescribed. In many cases, good effects will follow the judicious use of hot-air baths, to promote diaphoresis; or of potassium iodide, bitartrate, or acetate, or fluid extract of scoparius, to cause diuresis; or of fluid extract of euonymus or small doses of elaterium, to induce catharsis.

If pressure on the heart from pericardial effusion becomes excessive, the question of aspiration will suggest itself. I would recommend this operation in cases where heart failure seems imminent, but it should be held as a last resort.

During convalescence from this disease, the greatest care is necessary for ten or twelve weeks to avoid exposure or active exercise. The heart is always weakened by such an attack, and there is a tendency to dilatation, which should be guarded against by small doses of digitalis, strychnine, and arsenious acid. To promote strength still further, we should make free use of iron and good diet. The patient should avoid everything which would cause the heart extra labor.

If acute inflammation of the pericardium does not terminate in recovery within three weeks, the disease is termed *chronic pericarditis.* This condition may be characterized by a collection of fluid in the pericardium or by adhesion of the two surfaces of this sac. If the fluid becomes purulent it is termed pyo-pericardium.

In the former case, counter-irritation, diuretics, and cathartics are indicated; but in both cases, iron and cardiac tonics must be constantly employed, and excessive action is to be avoided. If the effusion be purulent, or if a non-purulent accumulation be sufficient to cause great irregularity of the heart with muffling of its sounds, or to threaten collapse, aspiration should be performed, preferably in the fifth intercostal space, two and a quarter inches to the left of the meso-sternal line, *i.e.*, near the junction of the sixth costal cartilage with the rib. Some recommend a point between the left side of the ensiform cartilage and the adjacent border of the costal cartilages. In pyo-pericardium, aspiration may be repeated several times, but with small hope of permanent relief. Incision, followed by antiseptic irrigation and temporary drainage, has been recommended.

PNEUMO-HYDROPERICARDIUM.

Pneumo-hydropericardium is one of the rarest of cardiac diseases. As the name indicates, it is a condition in which air or gas and fluid occupy the pericardial sac.

Etiology.—Air or gas may enter the pericardial sac through a penetrating wound or fistulous tract communicating with the trachea, bronchi, œsophagus, stomach, or possibly the intestines; or gas may in rare instances result from decomposition of fluid within the sac (Da Costa, Medical Diagnosis; also Hamilton, Text-Book of Pathology).

Symptomatology.—The essential signs of the affection are tympanitic resonance over the air, and flatness over the fluid, changing as the patient shifts from recumbency to the sitting posture; and, on auscultation, a splashing sound synchronous with the pulsation of the heart and entirely independent of the respiratory movements. The heart sounds have a metallic quality. The symptoms are similar to those of pericarditis.

Diagnosis.—Pneumo-hydrothorax and certain conditions of the stomach might possibly be mistaken for pneumo-hydropericardium; but there is no danger of an error in diagnosis if we remember that the signs of pneumo-hydrothorax are found on the side and posteriorly; and that the splashing sounds sometimes produced within the stomach are heard below the præcordial region.

Prognosis and Treatment.—The cases are usually speedily fatal. When they are prolonged, the treatment must be expectant.

HYDROPERICARDIUM.

Hydropericardium is a transudation or non-inflammatory effusion into the pericardial sac similar to that of hydrothorax.

Anatomical and Pathological Characteristics.—The liquid is of a pale yellow or greenish color, alkaline reaction, saltish taste, is not spontaneously coagulable, and has a specific gravity of 1005 to 1024.

The quantity varies from a few ounces to several pounds; the pericardium in the latter case being markedly distended and presenting the appearance of an obtuse cone with base downward.

Long-continued or excessive pressure of this effusion greatly impedes cardiac action, and the heart muscle weakens and degenerates.

Etiology.—Hydropericardium usually accompanies dropsical effusion into the other closed cavities, dependent upon heart, renal, or pulmonary disease; rarely it is due to an altered condition of the blood accompanying the cancerous and other grave cachexiæ.

Symptomatology.—The symptoms and signs are similar to those attending the effusion of pericarditis, but without friction sounds or other symptoms of inflammation.

Diagnosis.—The diagnosis depends on the history and the manifesta-

tions of the causative disease, with increased disturbance of the heart, enlarged area of cardiac dulness, and signs peculiar to the presence of fluid in the pericardium. Exploratory aspiration may be employed if necessary.

PROGNOSIS.—If the effusion is large in amount and accompanies valvular lesions, it may cause sudden death from pressure upon an already embarrassed heart. Treatment should be chiefly directed to the causative disease, from which death usually occurs.

ACUTE ENDOCARDITIS.

Inflammation of the lining membrane of the heart may be acute or chronic. The former is usually a non-ulcerative affection the result of rheumatism, but an ulcerative form also occurs as the product of septic infection. It has been variously termed ulcerative, infectious, septic, and by Virchow, malignant endocarditis. Chronic endocarditis may be such from the beginning, but it usually follows the simple acute form of the disease.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—Normally the endocardium from within outward consists of a single layer of polygonal endothelial cells, a thin elastic basement membrane, and a layer of nucleated white fibrous tissue joined to the cardiac muscular structure by loose areolar tissue. The valves of the heart are reduplications of the endocardium, those at the auriculo-ventricular septum containing also a few striated muscular fibres. Blood-vessels ramify in the loose areolar tissue, but nowhere penetrate the three layers of the endocardium; these, like the cornea, receive nourishment from the lymphatic spaces.

A few vessels accompany the muscular fibres of the mitral and tricuspid valves.

In the early stage of *acute endocarditis*, the endocardium appears slightly opaque or distinctly cloudy; later it is roughened, but redness is rarely visible after death. The sub-endocardial capillary plexus is injected. The lymph spaces are crowded with inflammatory products. The fibrous layer, chiefly, but also the areolar tissue, becomes infiltrated with round cells; as these proliferate, cloudy swelling occurs in the native fibrous cells, which appear, as the disease advances (Hamilton, Text-Book of Pathology) to become homogeneous and to be in great part absorbed. The affected membrane becomes thickened; proliferation of cells and their irregular accumulation gradually forces the endothelium and basement structure before it, producing minute papillary projections. Swelling and consequent distention finally result in destruction of the basement layer, and endothelial desquamation at the summits of the projections; upon these fibrin is deposited from the blood current. As the growth thus increases by proliferation within and fibrinous accretion without, it takes an irregular verrucous form, spreading at its summit

and constricted at its base. These vegetations develop most luxuriantly upon the valvular margins where most friction occurs, especially along the ventricular margin of the aortic valve. They may attain the size of a pea. This process is attended by no vascularization until far advanced, when the vessels at the base extend for a short distance into the vegetation (Hamilton, *loc. cit.*).

ETIOLOGY.—Acute endocarditis occurs most frequently in those under thirty years of age, and is most often the result of acute rheumatism. It also occurs in those suffering from gout, diabetes, alcoholism, Bright's disease, scarlet fever, typhoid fever, diphtheria, pneumonia, syphilis, and tuberculosis; chorea appears to be an occasional cause.

SYMPTOMATOLOGY.—The usual symptoms are: a sense of uneasiness about the heart, fever, a short cough, dyspnœa, and an anxious countenance.

The temperature rarely reaches 103½° F. In some cases vertigo and other cerebral symptoms may occur, or gastric disturbance, but none of these are constant features.

Among the *signs*, inspection commonly reveals turgescence and an anxious expression of the face. The cardiac impulse may be visible over an enlarged area.

In the beginning, the pulsations are apt to be forcible and irregular, with a corresponding pulse. An endocardial thrill is sometimes detected by palpation.

Percussion gives no increase of dulness in uncomplicated cases.

Auscultation usually reveals a soft, systolic murmur, due to endocardial or valvular thickening or roughening; these, however, may be present without a murmur. Often the second sound at the base is doubled from inco-ordinated action of the two sides of the heart. Murmurs may occur from lesions at any of the valves, but are most frequently heard at the apex.

DIAGNOSIS.—When some of the above symptoms appear in the course of any of the causative diseases, and these signs are obtained over a heart the sounds of which were formerly normal, we may reasonably suspect inflammation of the endocardium.

Acute endocarditis, when occurring independent of *pericarditis*, is liable to be mistaken for the latter disease. Pericarditis may be distinguished from uncomplicated inflammation of the endocardium by the quality, rhythm and seat of the murmur.

ACUTE ENDOCARDITIS.	PERICARDITIS.
Quality of murmur.	
Murmur blowing.	Distinctly rubbing or friction sound, to-and-fro shuffling; increased in intensity on the patient's bending forward and taking a deep inspiration, also by pressure of stethoscope.

ACUTE ENDOCARDITIS.	PERICARDITIS.
Rhythm of murmur.	
Murmur synchronous with the first sound of the heart, and does not occur with the diastole unless regurgitation takes place through the aortic or pulmonary semilunar valves.	Murmur not exactly synchronous with the valvular sounds, and often occurs during both the systole and the diastole of the heart; is not constant.
Seat of murmur.	
Murmur loudest at apex of heart, and diffused beyond the præcordia.	Murmur heard loudest at border of sternum near the fourth or fifth left costal cartilage. Limited to præcordia.

PROGNOSIS.—Acute rheumatic endocarditis usually runs its course in two to four weeks, and is seldom fatal unless complicated with other disorders. One attack, however, renders the part more vulnerable to subsequent disease. In favorable cases, endocardial murmurs decrease or entirely disappear during convalescence, but permanent valvular lesions remain in about twenty-five per cent of all cases of acute mitral endocarditis (Loomis' Practical Medicine). These, especially in children, are usually rapidly compensated for by cardiac hypertrophy. These permanent lesions often cannot be detected until contraction of the inflammatory products takes place, some weeks or months after subsidence of the acute inflammation.

The prognosis is rendered correspondingly grave by marked antecedent depreciation of general health; by the coexistence of disease of the pericardium or heart muscle; by an intercurrence of pulmonary and other diseases; by the development of typhoid symptoms; or the presence of signs and symptoms indicative of cerebral, splenic, hepatic, or renal embolism.

TREATMENT.—Endocarditis is nearly always the result of rheumatism, chorea, pyæmia, or the acute exanthematous fevers. The proper treatment for these affections is that which should in the main be employed in the secondary heart disease.

Perfect quiet should be maintained, not only during the active stage, but also during the convalescence.

In the very inception of the attack, a full dose of quinine will occasionally cut it short. Later, this remedy and iron are very useful. During the treatment, the patient should be kept in a warm room at 70° to 75° F., and the chest should be specially guarded from exposure.

Sibson recommends a liniment of tincture of belladonna and chloroform sprinkled on cotton-wool and kept applied to the præcordial region. Great depression calls for alcoholic stimulants and nux vomica or digitalis. The latter in moderate doses, combined with quinine, arsenious acid, and iron, is needed during convalescence, but care should be taken not to overstimulate the heart.

Exceptional—Nearly all cases of endocarditis are associated with or follow

other diseases, and are attended by symptoms which demand supporting treatment; but now and then one occurs without apparent cause in a robust person of full habit. In such case, general bleeding would undoubtedly prove beneficial by relieving the over-burdened heart.

ULCERATIVE ENDOCARDITIS.

Ulcerative endocarditis is a destructive inflammation of the endocardium due to infection, usually running a rapid and fatal course. Either or both sides of the heart may be its seat, but most frequently the left is involved. On the surface of the endocardium, chiefly on the valves, may be found gray fleshy vegetations springing from the sub-serous tissue, frequently associated with greenish-colored clots and containing perhaps minute purulent cavities.

Micro-organisms are always present, pyogenic bacteria, pneumococci, or tubercle bacilli flourishing with others of a harmless nature. Ulcers may coexist with vegetations or they may mark the site of those which have disappeared; their edges are irregular and thickened, and their floors purulent; perforation of the valves is a common result. Not infrequently these ulcers are the source of septic embolism in distant organs.

Etiology.—Ulcerative endocarditis may be caused by various pathogenic bacteria which gain entrance to the circulation in the different specific affections mentioned when speaking of the etiology and treatment of acute endocarditis, but most often during pyæmia; occasionally it arises idiopathically.

Symptomatology.—The affection often has symptoms and signs similar to those of myocarditis.

The usual symptoms may be those of severe enteric fever, the attack being often ushered in by a chill, followed by prostration, delirium, or coma. The temperature usually ranges higher than normal, from two to four degrees F. The tongue is often dry and brown; vomiting and diarrhœa are common. The pulse is rapid and irregular, and sometimes there are præcordial pains and palpitation of the heart, with dyspnœa and occasionally articular pains.

The evidences of embolism are often seen.

Sometimes no *signs* whatever are present, in other instances auscultation reveals the signs of valvular disease, and repeated examination may show rapidly progressing valvular changes.

Diagnosis.—The absence of cardiac symptoms in many cases is likely to mislead the physician into the diagnosis of intermittent or typhoid fever, or of pyæmia; but if attention is directed to the heart, and it is known to have been previously healthy, the occurrence of a systolic mitral or tricuspid murmur, with the symptoms just mentioned, renders the diagnosis reasonably certain.

Prognosis.—The prognosis is always grave, the disease usually terminating in death from the primary septic condition or from secondary pyæmic involvement of the brain, kidneys, spleen, liver, or other organs,

evidenced by hemiplegia or albuminuria or sudden enlargement and tenderness of the spleen or liver.

TREATMENT.—Ulcerative endocarditis results from pyæmia or septicæmia, and consequently requires the most vigorous supporting measures. Large doses of quinine and alcoholic stimulants are indicated.

Sansom recommends sodium sulpho-carbolate in thirty-grain doses, with inunctions of carbolized oil (*London Practitioner*, Jan., 1889).

CHRONIC ENDOCARDITIS—VALVULAR DISEASE OF THE HEART.

In chronic endocarditis the non-ulcerative inflammation, which is less acute from the start than in the acute disease, becomes protracted, cell infiltration and hyperplasia being followed by organization and marked contraction, especially at the base of the vegetation. The thickened tissues commonly become atheromatous in patches, these in turn undergoing calcification, as seen in the yellow areas and nodules of concretion scattered over the surface. Frequently it is coincident with a like condition in the walls of the aorta. Indolent ulcers sometimes exist where calcareous scales have been detached or where an atheromatous patch has softened. These changes may occur on any part of the endocardium, but the local effects of chronic endocarditis are most distinctly recorded in the valves.

Following the slight thickening of the acute stage, there is greater hyperplasia of the areolar and white fibrous tissue, especially along the edges of the valves. Organization with inevitable retraction produces incompetence of the valves. Narrowing of the aortic orifice may also result from the occurrence of the same process in the fibrous ring which normally exists at the base of the valves at the cardio-aortic junction. Complete calcification of this ring is an occasional result.

Atheroma and calcareous deposits also occur in the valves.

Adhesions may form between the valves and the aortic wall. Vegetations often fringe their ventricular margin. Ulceration prone to follow fibrosis and atheroma may perforate the valve entirely, or from partial destruction give rise to valvular aneurism. The mitral valves are subject to similar changes, and, as the free edges of the valves are continuous, general contraction narrows the orifice in marked cases to a mere slit like a buttonhole.

The chordæ tendineæ are involved in the process of thickening and retraction, and may become agglutinated into one or more short, fibrous bands which draw down the contracted mitral margin, converting the valves into a funnel-shaped projection into the ventricle.

The tricuspid valve is seldom so affected. Aortic regurgitation or obstruction produces dilatation of the left ventricle followed in favorable cases by compensatory hypertrophy of its walls. Like conditions of the mitral orifice produce like effects in the left auricle.

Theoretically, similar affections at the tricuspid and pulmonary valves produce corresponding changes in the cavities and walls of the right heart; but practically tricuspid stenosis, and stenosis and regurgitation at the pulmonary valves, are exceedingly rare. Tricuspid regurgitation, with dilatation and hypertrophy of the right heart, is usually the result of serious lesions of the left heart.

Chronic valvular lesions, though sometimes occurring alone are apt to produce disease of other organs, by obstructing the circulation. In the lungs, we find congestion, œdema, bronchitis, apoplexy, brown induration, and lobar pneumonia. The kidneys may become congested and enlarged, and are not infrequently the seat of embolic infarcts or multiple abscesses. The same is true of the spleen. Continuous engorgement may cause parenchymatous, fatty, or atrophic degeneration of the liver, or chronic catarrh of the gastro-intestinal mucous membrane; and occasionally embolism or apoplectic extravasation may take place in the brain.

Endocarditis may produce at the orifices of the heart either obstruction or insufficiency of the valves.

Stenosis or stricture may be the result of thickening of the valves from the presence of calcareous deposit, atheromatous or fibroid tissue, or extensive vegetations; or of adhesions between the valves, or of induration, hyperplasia, and contraction of the margins of the openings. Rarely it is a congenital condition.

Incompetency may be due to perforation, tearing, or inflammatory retraction of the valves or to rigidity from calcareous deposit; to rupture or abnormal shortening or lengthening of the chordæ tendineæ, dilatation of the ventricle without compensatory lengthening of the chordæ and their muscles; and to spasm of the columnæ carneæ.

Etiology.—Chronic endocarditis is more frequent in men than in women. It usually follows the acute non-ulcerative form of the disease, but may be chronic from the beginning, especially when associated with chronic alcoholism, rheumatism, gout, or old age.

Symptomatology.—Chronic endocarditis sooner or later causes irregularity in the action of the heart, lividity of the lips, œdema, and dyspnœa on exertion. Dizziness and vertigo with facial pallor and sometimes syncope arise from cerebral anæmia; sudden loss of consciousness with subsequent paraplegia may arise from cerebral embolism or apoplexy. Headache, tinnitus aurium and muscæ volitantes are commonly due to cerebral congestion.

Often cardiac pains occur, frequently shooting to the left shoulder and down the arm. Sometimes there is true angina pectoris. Cardiac dyspnœa and palpitation are common. The pulse may be rapid, weak, irregular, intermittent, small, wiry, or full and compressible. The so-called water-hammer, collapsing, jerking or piston pulse is characteristic of aortic regurgitation. The pulse in other valvular lesions is

not diagnostic, but indicates the force of the heart, the tone of the vessels, and the condition of the circulation.

If the pulmonary circulation be embarrassed, cough, dyspnœa, oppression, and profuse expectoration are present, especially on exertion. Blood-stained sputum is common, and hæmoptysis not infrequent.

General venous engorgement is manifested by cyanosis, tenderness and enlargement of the liver and spleen, anorexia, nausea and vomiting, and sometimes jaundice; also by albuminuria with casts, scanty and occasionally bloodstained urine, increasing œdema commencing in the lower limbs, and effusion into the serous cavities.

The *signs* require careful discrimination. *Aortic obstruction*, commonly manifesting the symptoms of cerebral anæmia, is characterized by a hard, wiry, but regular pulse; enlargement of the left heart; a systolic murmur with the first sound usually harsh, loudest at the right second intercostal space, occasionally at the left or over the upper part of the sternum. This murmur is conveyed into the vessels of the neck, is heard behind, and toward the apex but with diminished intensity, and is not transmitted to the left of the apex. The pulmonic second sound is feeble.

Aortic regurgitation exhibits no peculiar early symptoms. It is characterized by a full, strong, but collapsing pulse. The left heart is enlarged; the carotids beat forcibly, and distinct capillary pulsation may sometimes be seen beneath the finger-nails and the mucous membrane of the lips, and at the fundus of the eye. It causes a diastolic murmur, soft and blowing, occurring with or following the second sound, which is most distinct over the lower part of the sternum, but is sometimes heard behind and in the arteries of the neck. It is more widely diffused than any other murmur.

Mitral obstruction causes marked pulmonary symptoms and signs, and is accompanied by a soft, small pulse and a purring thrill most distinct at the apex; by left auricular enlargement, sometimes but not usually elicited by percussion; and by the mitral presystolic murmur preceding the first sound already noted as represented by vocalizing the symbols, R r r b or V o o t. It is apt to be of longer duration than other murmurs. Its maximum intensity is about half an inch above the apex beat, it is louder when the patient is erect, is not transmitted to the left of the apex beat, is not heard behind, nor in the arteries of the neck.

Mitral regurgitation commonly produces the symptoms of pulmonary, hepatic, and renal congestion, and is accompanied by a compressible and irregular pulse and enlargement of the left heart. The murmur produced is soft and blowing; it is systolic, accompanying or replacing the first sound; and is heard loudest at the apex. It is transmitted to the left, and is often heard behind beside the sixth and seventh dorsal vertebræ opposite the mitral area in front. It is not propagated into the arteries of the neck. The pulmonic second sound is intensified.

Tricuspid regurgitation, usually secondary to lesions of the left heart or to pulmonary diseases, and when marked, producing symptoms of pas-

sive congestion of the brain, and of the liver and other abdominal organs, exhibits the following signs: pulsation of the jugulars, enlargement of the right heart, a comparatively feeble systolic murmur replacing the first sound, and loudest in the tricuspid area. It is transmitted to the right if at all, is not heard at the apex, behind, or over the carotids, and is seldom audible above the third rib. The pulmonic second sound is feeble.

Tricuspid obstruction and *pulmonic regurgitation* are so rare as hardly to merit mention. The former causes presystolic, the latter a diastolic murmur; the former harsh, the latter soft; the former heard most distinctly over the lower part of the sternum, the latter over the left second intercostal space, but propagated downward. The second pulmonic sound would probably be heard in tricuspid obstruction, but would be absent in pulmonic regurgitation.

Pulmonic obstruction causes enlargement of the right heart and a systolic murmur with the first sound, of maximum intensity at the left second intercostal space, occasionally transmitted toward the left shoulder, but never downward to the apex nor over the aorta and carotids. It is not heard over the lower part of the sternum or behind. There may be an attendant *bruit de diable* of the jugulars.

Diagnosis.—The differential diagnosis between different valvular lesions must be made from the foregoing symptoms and signs. In case of single, or clearly defined double valvular sounds, little confusion need arise in determining their diastolic or systolic character if their rhythm be referred to the carotid pulse. This in most cases can be felt on deep, digital pressure beneath the angle of the jaw, just in front of the anterior margin of the sterno-cleido-mastoid. Not infrequently an accurate diagnosis is impossible when the action of the heart is rapid, irregular, and tumultuous. In these cases better results may be obtained by auscultation after proper exhibition of digitalis. In the diagnosis of chronic endocarditis, too much significance must not be attached to the presence of valvular murmurs, as serious disease may exist without them. Such cases are indicated by the various symptoms already mentioned and by feeble or intermittent action of the heart, with increased area of cardiac dulness due to hypertrophy or dilatation.

Chronic endocarditis or organic disease of the heart may be confused with functional disease of the heart, pericarditis, anæmia, aneurism, fatty degeneration, cardiac dilatation, and with certain congenital deformities of the heart. The differential points are as follows:

Chronic endocarditis.	Functional heart disease.
History.	
Palpitation comes on gradually.	Palpitation paroxysmal, comes on suddenly, not constant.
Frequently history of rheumatism, gout, or syphilis.	History often points to indigestion, hysteria, the nervous diathesis or excessive use of tobacco or coffee.

CHRONIC ENDOCARDITIS.	FUNCTIONAL HEART DISEASE.
Symptoms.	
Anxiety not marked till late in disease. Palpitation usually brought on by exertion. Dyspnœa, cyanosis, or cough.	Anxiety, worry and nervousness prominent. Palpitation usually without exertion. No evidence of organic disturbance other than anæmia.
Signs.	
Enlargement of the heart, change in apex beat. Murmurs may be diastolic; they may replace heart sounds; they are usually constant.	No enlargement of heart. Murmurs if present are inconstant, always systolic. Are due to anæmia and disappear on treatment. Heart sounds present though feeble.

CHRONIC ENDOCARDITIS.	PERICARDITIS.
Signs.	
Usually cardiac enlargement. Murmurs constant and widely diffused; commonly synchronous with heart sounds which they may replace.	No enlargement till second stage. Murmurs confined to narrow limits; most distinct at left fourth costo-sternal articulation ; sometimes increased on pressure with stethoscope, on deep inspiration, and on forward inclination of patient. Murmurs inconstant and not synchronous with valvular sounds. Heart sounds not supplanted.

CHRONIC ENDOCARDITIS.	ANÆMIA.
Patient may appear robust. Pulse may be full and strong. Heart enlarged. Murmurs constant, widely diffused. No venous hum.	Pallor and lassitude. Pulse weak, compressible. Heart normal size. Murmur inconstant and often loudest over carotids. Venous hum.

CHRONIC ENDOCARDITIS.	THORACIC ANEURISM.
Symptoms.	
No marked symptoms at beginning.	Marked symptoms significant of pressure, as, boring pain, dysphagia, aphonia.
Signs.	
Heart enlarged. Pulse alike on both sides. No dilating impulse. Murmur frequently widely transmitted.	Heart of normal size. Pulse often different on two sides. Dilating impulse. Peculiar bruit localized. Never transmitted toward apex.

The diagnosis of *fatty heart* rests chiefly upon the history of the case, the absence of distinct signs of organic lesions, and the occurrence of Cheyne-Stokes respiration.

Congenital deformities of the heart may be distinguished by the history, the blueness of the surface, and the occurrence of a systolic murmur not transmitted to the left of the apex or to the arteries and heard only over the base of the heart.

Prognosis.—Organic valvular heart disease is rarely if ever curable, but in duration and fatality it varies widely in different cases according to the cause, extent, seat, and progressive or non-progressive tendency of the lesion; the degree and rapidity of compensation; the presence of complications; the age, sex, and condition of the patient and his willingness and capacity to follow a proper mode of life and treatment.

Infants and old people endure valvular disease poorly. In older children and adults, the heart tends to compensate more quickly. Women are oftener affected than men, but they have a better chance of prolonged life because of less exposure to severe strain and alcoholic and other excesses with the resulting arterio-sclerosis, and angina pectoris of organic origin. Arduous and exposing occupations and a reckless or passionate disposition influence the prognosis unfavorably. A progressive trend of the disease evidenced in the past and present history is unpropitious, especially when associated with or dependent upon renal disorder. Evidence of dilatation without compensation or of coexistent arterio-sclerosis is ominous. The gravity increases with the number of lesions, and is greatly augmented by the occurrence of diseases which weaken the heart. Heart disease dependent upon uncomplicated chorea is not usually serious. In any case prompt relief following the use of heart tonics is a good sign.

In *aortic stenosis*, compensatory hypertrophy is usually prompt and may be efficient for years. The danger lies in failure of compensation, or in cerebral embolism, which is more frequent from this than from any other valvular disease. Death may also result from sudden heart failure or from pulmonary œdema after secondary mitral insufficiency and left ventricular dilatation.

Aortic regurgitation, though frequently existing for years and without much discomfort, is the most apt of all valvular diseases to cause sudden death, mitral stenosis ranking close in this respect. It is most severe when suddenly developed (Loomis' Practical Medicine), and grave when followed by signs of mitral insufficiency, dilatation, heart failure, renal, or other visceral disease. Death may occur from these or from cerebral anæmia and syncope, from cerebral apoplexy or embolism, or from asphyxia due to pulmonary congestion and œdema.

Mitral stenosis renders the patient liable to pulmonary congestion, œdema, or apoplexy, and not infrequently ends in sudden cardiac failure.

In *mitral regurgitation*, the prognosis is fairly good as compensatory hypertrophy is usually equal to the necessity, at least for some time. Danger results from its failure and consequent general venous engorgement, giving rise to dropsy of the lungs, serous cavities, and limbs. Death from heart failure or from asphyxia naturally follows, but only about two per cent of patients with mitral disease die suddenly.

Tricuspid stenosis and *lesions of the pulmonary orifice* are seldom met with, but, when present, are necessarily grave conditions.

Tricuspid regurgitation is exceedingly grave, whether the result of

chronic pulmonary disease or secondary to lesions of the left heart. In this condition, sudden increase in the pulmonary engorgement and death from suffocation is a constant danger.

The symptoms usually indicative of a fatal issue in valvular disease of the heart are: great anxiety, with sense of oppression, followed by pallor, vertigo, syncope, and muscular debility, and irregular, weak, intermittent, and rapid pulse of 120 beats or more per minute, especially when accompanied, on palpation of the præcordia, by a purring tremor. Great anasarca and fluid effusion into the serous cavities, dyspnœa, hæmoptysis, and cyanosis are bad signs.

TREATMENT.—In the treatment of valvular lesions, three things are constantly to be borne in mind. The labor of the heart must be rendered as light as possible, the blood must be kept in a healthy condition, and the strength of the heart must be maintained.

With the first object in view, we interdict rapid walking, running, or heavy lifting, and enjoin the patient to avoid climbing stairs, and indeed every act or form of exercise, mental or physical, which causes dyspnœa and palpitation. We attempt also by proper treatment to remove all obstruction to the circulation; hence, pulmonary and other diseases must receive appropriate treatment. Even a simple bronchitis may be sufficient greatly to obstruct the pulmonary circulation. Portal congestion, or obstruction in the systemic capillaries which may be contracted as the result of nervous irritation caused by the retained excreta in Bright's disease must be relieved. Remembering that affections of the lungs, liver, alimentary canal, kidneys, or skin may have caused the cardiac disease, or may greatly aggravate it, we naturally look for these, and seek to combat them by appropriate treatment.

With the second object in view, we aim to maintain free elimination by the kidneys, bowels, and skin, and recommend vegetable tonics, iron, and nutritious diet, with regular habits.

To accomplish the third object, besides the means already suggested for relieving the heart of work and for furnishing it with proper nutrition, we prohibit the use of tobacco and of all other depressing agents and administer various heart tonics, chief among which are digitalis, arsenic, and cactus grandiflora; belladonna and squills have a tonic effect on the heart similar to these, though less potent. In many cases nux vomica is a most useful remedy.

Other heart tonics of value, alone or combined with digitalis, are: strophanthus, best given in tincture, ♏ v. to x.; sparteine sulphate, gr. $\frac{1}{8}$–i; caffeine citrate, gr. ij.–iij.; tincture of convallaria, ♏ x.–xx.; and nitroglycerin. The latter, in doses of gr. $\frac{1}{100}$ repeated within twenty minutes if necessary, is of special value when a prompt cardiac stimulant is needed. Amyl nitrite acts in a similar manner. Sparteine seems of most value, when given in full doses, in regulating the rhythm of the heart. Though the remedies directed to the heart itself are of the greatest service in the

treatment of valvular disease, they should not be used indiscriminately, for the apparent weakness may sometimes be much more effectually overcome by medicines which act upon some other organ. Moderate exercise is sometimes of great value in maintaining the strength of the heart muscle.

In aortic obstruction or regurgitation, it is especially important to avoid taxing the power of the heart, and to maintain its strength by cardiac tonics and a good supply of rich blood. Nature always attempts to compensate for the obstruction or regurgitation by hypertrophy of the left ventricle; but a time finally comes when the compensation fails, then digitalis should be given to strengthen the muscular walls. Ten minims of the tincture three times a day is the ordinary dose, but the amount may be gradually increased until the heart pulsates regularly and with normal force, providing the kidneys act freely and the stomach is not deranged. Twenty minims may be given as often as every two hours, without danger, if there is a free secretion of urine; but if the flow stops, the digitalis must be at once suspended.

When compensation is complete, so that the heart beats regularly and with normal force and frequency, good hygienic surroundings, with regulation of diet and exercise, are all that is needed. Exaggerated hypertrophy with too powerful systole demands cardiac sedatives.

In mitral obstruction or regurgitation, digitalis is usually most beneficial. It should be given as just recommended for aortic disease. When it loses its effect, arsenious acid or nux vomica should be tried, alone or with the digitalis. Other diuretics, vapor or hot-air baths, and cathartics will be required from time to time, to relieve pulmonary congestion and œdema or general dropsy.

From the experiments of Germain Sée (*La Tribune Médicale*, 1890) lactose, a well-known constituent of milk, appears to be diuretic. Calomel in small doses is also a stimulant of the renal function and is specially indicated when the liver is engorged.

It is important to continue the use of cardiac tonics in medium doses two or three times a day, for many months after the distressing symptoms, for which the physician was first called, have passed away; but the amount must always be carefully regulated, so as not to over-stimulate the organ.

Disease of the *pulmonary valves* requires similar treatment to that recommended for mitral affections.

In tricuspid regurgitation, the same general rules laid down for the treatment of other valvular lesions are to be followed; but unless mitral disease coexists, digitalis will do more harm than good, by increasing the venous congestion of the brain and of the abdominal organs.

MYOCARDITIS.

Myocarditis or inflammation of the muscular fibres of the heart may be acute or chronic.

Anatomical and Pathological Characteristics.—The usual seat of myocarditis is the wall of the left ventricle. Very acute inflammation is marked by infiltration and swelling of the muscular fibres together with their sheaths, and tends to their rapid disorganization and the formation of small abscesses circumscribed by connective-tissue proliferation. Exceptionally the process ends in diffuse purulent infiltration.

Abscesses weaken the wall of the heart, give rise to dilatation, rupture, or aneurism of the organ, and may themselves discharge into the pericardial sac, producing pyo-pericardium, or into the ventricle, causing pyæmia.

Chronic myocarditis is essentially interstitial, and eventuates in cirrhosis, making the organ larger and heavier than normal, varying in color from gray or pink to a bluish hue. The muscle becomes tough and inelastic and either increased in thickness or attenuated. The process is gradual, and may begin in the parts adjacent to the endocardium or the pericardium or may primarily involve the intermuscular septa.

New cells, tending to organize, produce pressure—atrophy of the muscular fibres or fatty degeneration from disturbed nutrition. The growth of fibroid tissue may be so extensive as largely to replace muscular elements, or it may exist only as cicatrices, scattered at irregular intervals, commonly most marked at the apex (Hamilton, Text-Book of Pathology).

As a result, the affected wall is tough and leathery, either distinctly attenuated or much thickened and of a gray color. This fibroid tissue sometimes undergoes calcification. The entire wall of an auricle has been found in such a condition.

Etiology.—Acute myocarditis is usually of septic origin, either occurring as a part of pyæmia or developed in the course of typhoid or other infectious fevers. The chronic form usually accompanies rheumatic endocarditis and pericarditis, but may occur alone. Huber, however, holds that it rather follows arterio-sclerosis of the coronary artery. Syphilis may also produce it (Hamilton, *op. cit.*).

Symptomatology.—*Acute myocarditis* is a rare affection, and of its symptoms and signs we know little, apart from its association with endocarditis or pericarditis. If, during the progress of either of these diseases, the heart's action becomes intermittent or irregular, and there is a tendency to syncope, it is probable that the muscular tissue of the organ has become involved.

The symptoms and signs frequently observed are: extreme pallor of the countenance, with coldness of the surface and a tendency to syn-

cope; also pain and oppression at the præcordia, with dyspnœa amounting to orthopnœa, and sighing respiration. The action of the heart is feeble, fluttering, and irregular. The area of cardiac dulness remains normal unless dilatation or pericardial effusion exists. Both sounds of the heart are sharp and valvular, the first very closely resembling the second. They may sometimes be represented by the *ta*, *ta* characteristic of the fœtal heart. With these symptoms and signs, the patient may complain of severe pain in the head and limbs, and there may be delirium or hemiplegia. All or only a part of these may be present or absent.

The symptoms of *chronic myocarditis* or *fibroid disease of the heart* most frequently noticed are cardiac pain, œdema, and dyspnœa, but all of these may be absent.

The *signs* are: a weak, irregular, and rapid pulse and feeble apex-beat, with coincident enlargement of the cardiac area of dulness. Reduplication of the first sound is also sometimes present.

Diagnosis.—If an acute affection of the heart is attended with pallor and coldness of the surface, syncope, pain in the cardiac region, and a feeble, fluttering, and irregular pulsation, we may fairly suspect acute inflammation of its muscular walls.

Neither the symptoms nor the signs nor these combined are sufficient to distinguish accurately *fibroid disease of the heart* from dilatation or fatty degeneration. In both, marked feebleness of the heart is present; in fatty degeneration, the heart is not so commonly enlarged as in the diffuse fibroid disease; the former is usually associated with anæmia, the latter with general sclerosis, chronic nephritis, or syphilis.

According to Riegel, the pathognomonic sign of chronic myocarditis is irregularity of action of the heart, a total loss of rhythm appearing early in the disease and remaining irrespective of the influence of digitalis and other agents in restoring the functional activity of the organ and dispelling dropsy and other symptoms of deficient heart power (*Zeitschrift für klinische Medicin*, 1889). Irregularity, though a feature of many other cardiac conditions, is in them always a late symptom, due to secondary weakness, and it disappears when heart tonics have been effective.

Prognosis.—Theoretically, the prognosis in myocarditis is always grave, especially in the acute form. Practically, a satisfactory prognosis is rarely possible, because an accurate diagnosis can seldom be made. When occurring with endocarditis and pericarditis, it adds to the danger of death from heart failure, cardiac aneurism or rupture, or from pulmonary congestion and œdema, or embolism and pyæmia. The chronic form may terminate in general dropsy or in death from cerebral anæmia.

Treatment.—The treatment for myocarditis is that for its associated and frequently causative diseases.

Patients suffering from endocarditis, pericarditis or any obscure heart trouble, from typhoid fever or other debilitating diseases, in whom myo-

carditis may be even remotely suspected require: perfect rest in the recumbent position; avoidance of all mental or bodily strain; nutritious and easily assimilated diet; the maintenance of elimination from skin, bowels, and kidneys and moderate stimulation of the failing heart with alcoholics, strychnine, digitalis, the ammonium compounds, or nitrites.

CHAPTER XIV.

CARDIAC AND ARTERIAL DISEASES.—*Continued.*

SIMPLE CARDIAC HYPERTROPHY.

Synonyms.—Enlargement of the heart; hypersarcosis cordis.

Simple cardiac hypertrophy consists of hypertrophy of the muscular walls of one or more of the cardiac cavities without enlargement of the cavity itself.

Anatomical and Pathological Characteristics.—Simple cardiac hypertrophy unattended by dilatation is comparatively rare and is seldom general. It may be localized in any part of the cardiac muscle, but it affects the wall of the ventricle more frequently than that of the auricle, being oftenest confined to the left side. The interventricular septum is not usually much implicated. In well-marked cases the organ is always large and heavy, and changed in shape according to the seat of hypertrophy. The wall is not uncommonly doubled in thickness. It is redder and more rigid than normal, the enclosed cavities remaining patulous after death. The affected wall of the left ventricle will be extra-friable; that of the right, tough and leathery (Loomis' Practical Medicine). The hypertrophy results from increase in the muscular structure of the heart, whether in number or size of the individual fibres or in both. The increase does not involve the connective tissue to any extent in simple hypertrophy, but may extend to the columnæ carneæ, especially of the left ventricle.

Etiology.—Simple cardiac hypertrophy may arise from functional over-action of the heart, due to prolonged or severe muscular efforts, to nervous or mental causes, or to the effects of alcohol, tea, and coffee. It may result from slight obstruction at the valvular orifices or to embarrassment of the heart's action from displacement or pericardial adhesions. It may be produced by obstructed circulation outside the heart, as from constriction of great vessels or pressure upon them; from degenerative changes of the arterial system, such as endarteritis obliterans, atheroma, and loss of elasticity; or it may be caused by the obstruction resulting from contraction of the arterioles associated with Bright's disease, alcoholism, and syphilis. It may be due to local or to visceral disease, as emphysema, cirrhosis of the lung, or pleural effusions which interfere with the pulmonary circuit. Physiological cardiac hypertrophy occurs in pregnancy.

Symptomatology.—The symptoms are not marked, though there is

a tendency to cerebral hyperæmia, and palpitation on exertion or excitement; a dry cough may be present at times, from slight pulmonary congestion.

The *signs* in this affection vary with the extent of the hypertrophy, and with the portion of the organ involved. The essential signs are: increased area of dulness and increased force of impulse while the heart's action remains regular.

Inspection in children frequently reveals a prominence of the præcordial region when the hypertrophy is general, but in adults this cannot be detected. The action of the heart is regular and forcible. If the left ventricle alone be hypertrophied, the apex beat will be farther than usual to the left, and the visible area of the impulse increased, often extending over the whole præcordia. If the right ventricle is affected, there will be strong epigastric pulsation, and the apex beat, if perceptible, will be below and to the right of the usual position.

Palpation confirms the signs as to the position and force of the apex beat.

On percussion, the areas of superficial and deep-seated cardiac dulness are found to be increased. The latter in simple hypertrophy of the left ventricle seldom extends more than an inch to the left of the normal position. A larger area is almost always associated with more or less dilatation. In hypertrophy of the right ventricle, the dulness extends considerably to the right of the sternum.

In hypertrophy of the ventricles, auscultation finds the first sound of the heart greatly increased in intensity, and the elements of muscular contraction and impulsion are especially marked. The second sound is also increased in intensity and more widely diffused than normal. The action of the heart remains regular as long as hypertrophy compensates for the obstruction.

The respiratory murmur is diminished or absent over a portion of the præcordial region corresponding to the displacement of the lung.

Diagnosis.—Simple cardiac hypertrophy may be confused with several affections, which will be considered to better advantage under diagnosis of hypertrophy and dilatation of the heart, from which it is distinguished by the larger size of the heart and greater irregularity of action, with more of a heaving impulse in the latter. Again, in hypertrophy and dilatation of the heart, valvular murmurs are more commonly present than in simple hypertrophy; otherwise the symptoms and signs of the two affections are substantially alike.

Prognosis.—Simple cardiac hypertrophy as a compensatory process is usually favorable, providing the causative factors be not such as to produce eventual cardiac or vascular degeneration by their persistence or progressiveness. Cases dependent simply upon mental or muscular excitement are not serious under a properly regulated mode of life. When there is a marked tendency to cerebral congestion, especially in

alcoholic subjects or those in whom arterial degeneration has taken place, this affection is liable to eventuate in cerebral apoplexy.

TREATMENT.—Usually, hypertrophy of the heart should be favored rather than retarded; but in some instances, symptoms of cerebral congestion appear such as pain, fulness of the head and vertigo, which require prompt attention. Bleeding will temporarily relieve these, but it is not to be recommended. Tincture of aconite root in doses of two or three drops every two hours until relief is obtained is the most efficient remedy in such instances. Veratrum viride may be used for the same purpose.

It must not be forgotten that similar symptoms are caused by passive congestion depending upon cardiac failure, and that in such cases the aconite would be harmful. These latter cases I have found most quickly relieved by nux vomica. The causes of the hypertrophy should be sought and removed as far as possible.

HYPERTROPHY AND DILATATION OF THE HEART.

Hypertrophy and dilatation of the heart, also called eccentric cardiac hypertrophy, affecting the muscular walls and dilating the cavities, is caused by yielding of the walls to excessive pressure, which may result from the same causes which induced the hypertrophy, or from regurgitation of blood through incompetent valves.

SYMPTOMATOLOGY.—Dyspnœa on exertion, œdema especially of the ankles, and occasional vertigo, and palpitation of the heart are common symptoms. In this affection, the action of the heart remains regular if the hypertrophy is sufficient to compensate for the dilatation; but it becomes irregular if the dilatation predominates.

The essential *signs* are: increased area of visible impulse, with displacement of the apex beat downward and to the left, and a peculiar heaving impulse with increased area of dulness. Endocardial murmurs are nearly always present.

Inspection and palpation show that the area over which the cardiac impulse may be seen and felt is greatly increased, sometimes extending over the entire left side. The impulse often has a peculiar heaving or lifting character, sufficient in some instances to shake the bed on which the patient is lying. The apex beat may sometimes be two or three inches to the left of the left nipple, and as low as the eighth rib.

Upon percussion, the area of dulness is increased to the left and downward, in proportion to the enlargement of the organ; if the right ventricle is affected, it is also increased to the right.

In auscultation, both sounds of the heart are prolonged, and may often be heard over the entire chest. If valvular murmurs are present, they will be loudest in the normal areas, described in a previous chapter (Fig. 32), but they may also be heard in some instances over the whole thorax.

DIAGNOSIS.—Eccentric cardiac hypertrophy may be mistaken for retraction or consolidation of the lung, cardiac dilatation, pericardial effusion, cardiac displacement, thoracic aneurism, or for simple cardiac hypertrophy.

Retraction of the lung due to pleuritic adhesions or pulmonary cirrhosis, by exposing a larger surface of the heart, may increase the area of superficial cardiac dulness and thus simulate hypertrophy; but the history of former trouble, pulmonary symptoms and signs of more or less prominence, and the normal condition of the pulse, heart sounds, and force of the apex beat distinguish it from cardiac hypertrophy. The distinctive features between eccentric cardiac hypertrophy and *consolidation of the lung* are as follows:

HYPERTROPHY AND DILATATION OF THE HEART.	CONSOLIDATION OF THE LUNG.
Symptoms.	
Cough not prominent.	Cough prominent.
Inspection.	
Impulse at apex forcible, action tumultuous.	Force of apex beat normal.
Palpation.	
Pulse full and strong.	Pulse normal or weak and rapid.
Percussion.	
Outline of dulness quadrilateral and confined to præcordia.	Outline irregular and extending beyond the limits of the heart.
Auscultation.	
Heart sounds intensified.	Heart sounds normal. Bronchial breathing, bronchophony, and râles.

Eccentric cardiac hypertrophy differs from dilatation of the heart as below:

HYPERTROPHY AND DILATATION OF THE HEART.	DILATATION OF THE HEART.
Symptoms.	
Symptoms of cerebral hyperæmia.	Progressive general weakness, and œdema of feet.
Inspection.	
Face flushed; carotids prominent; apex beat heaving and forcible, and distinct over large area.	Face pale or livid, veins turgid, perhaps pulsating jugulars; apex beat feeble, not always visible, though it may be seen over an area larger than usual, but less than that of hypertrophy and dilatation.
Palpation.	
Apex beat forcible; pulse full and strong.	Apex beat diffused, weak; pulse weak and irregular.
Auscultation.	
Sounds intensified; first sound prolonged.	Sounds feeble, and first sound short.

Eccentric cardiac hypertrophy and pericardial effusion and hydropericardium have the following distinctions:

HYPERTROPHY AND DILATATION OF THE HEART.	PERICARDIAL EFFUSION.
Symptoms.	
Slowly developed and not prominent.	Symptoms acute in pericarditis.
Palpation.	
Apex beat strong, displaced to the left, and depressed.	Apex beat weak, carried slightly to left and apparently raised.
Percussion.	
Outline of dulness quadrilateral, and not extending to left of apex beat.	Outline triangular, and extending to left of apex beat.
Auscultation.	
Heart sounds distinct.	Sounds feeble.
No friction sounds.	Friction sounds have been present in pericarditis, and may be still, at base of heart.

Eccentric cardiac nypertrophy and cardiac displacement differ thus:

HYPERTROPHY AND DILATATION OF THE HEART.	CARDIAC DISPLACEMENT.
Symptoms.	
Cerebral hyperæmia.	None characteristic.
Palpation.	
Heaving apex beat over great area.	Apex beat of normal force; area not necessarily enlarged.
Percussion.	
Area of dulness increased.	Area of dulness not necessarily increased.
Auscultation.	
Sounds intensified.	Sounds normal.

Eccentric cardiac hypertrophy differs from thoracic aneurism as shown below:

HYPERTROPHY AND DILATATION OF THE HEART.	THORACIC ANEURISM.
Symptoms.	
No aphonia, dysphagia, or boring pain.	Boring pain, dysphagia, aphonia, etc., due to pressure.
Palpation.	
Impulse heaving and below fourth rib.	Impulse dilating and above fourth rib. Aneurismal thrill.

Hypertrophy and dilatation of the heart.	Thoracic aneurism.
Percussion.	
Dulness increased to the left and downward.	Dulness increased upward.
Auscultation.	
Heart sounds intensified.	Bruit; heart sounds normal.

Prognosis.—The prognosis depends largely upon the removability of the cause, or, if this is permanent, upon its progressive or non-progressive character. Existing hypertrophy, though sufficient to meet the ordinary demands of the case for years, may be rendered inefficient by undue muscular strain, exhausting diseases, great and continued emotional disturbances, or in some cases by pregnancy; the latter condition, however, is not contra-indicated in moderate cases.

When great force must be habitually exerted by the ventricle to overcome increased resistance due to obstruction or regurgitation, the evil effects are apt to be manifested in chronic congestion of the lungs, in degeneration of the arteries generally, or in rupture of cerebral vessels which may already be the seat of atheroma.

Treatment.—The treatment of this condition is essentially that of chronic endocarditis with valvular disease of the heart, with which it is nearly always associated.

As long as hypertrophy is perfectly compensatory, no treatment is demanded except in case of excessive cerebral congestion, with danger of apoplexy, when cardiac sedatives are indicated. Otherwise the hygienic and medicinal treatment suggested for disease of the heart should be carried out.

DILATATION OF THE HEART.

Synonyms.—Passive aneurism of the heart; cardiectasis; cardiac dilatation.

Anatomical and Pathological Characteristics.—Dilatation of the heart refers to an abnormal increase in the cavities of the heart, irrespective of the condition of its walls, which may be relatively normal or attenuated. The auricles are most frequently affected, and the right ventricle oftener than the left. The shape of a dilated heart depends upon the amount of dilatation, and upon the cavity or cavities involved. The shape may be irregular from bulging of a single auricle or ventricle; or more uniformly enlarged, from stretching of all the cavities. The walls, if not normal, may be atrophic or slightly hypertrophic and may be the seat of various degenerations or infiltrations according to the cause of the affection.

Etiology.—Dilatation of the heart is dependent upon a disparity between the power of the cardiac muscle and the intra-cardiac pressure.

Old age, tending to retrogressive change; sex and occupation, as influencing exposure; and heredity, are remote factors in its production. The predisposing causes include all the conditions which weaken the walls of the heart. Prominent among these is atony of its muscular fibres resulting from anæmia, chlorosis, exhausting febrile and infectious diseases; derangements of innervation incident to sexual, alcoholic, and other excesses; or certain nervous disorders, as Graves' disease. The muscular walls may be weakened by degeneration. This may result from obstruction of the coronary artery by embolism, arterio-sclerosis or contraction of old pericardial adhesions; or it may be secondary to rheumatic, gouty, or syphilitic pericarditis, endocarditis, or myocarditis; or atrophy may occur, due to old age or to pressure from amyloid or fatty infiltration, new growths, or chronic pericardial effusion. The exciting cause of dilatation is increase of intracardiac pressure. This may occur from valvular disease, and from the pressure of tumors upon the aorta, pulmonary artery, or other great vessels; from general increase of arterial tension associated with Bright's disease; from obstruction of smaller vessels or stasis incident to prolonged muscular efforts, or to fibroid phthisis or other diseases of the lungs; or from local vascular degeneration due to alcoholism, syphilis, and gout, notably to endarteritis obliterans.

Symptomatology.—The most frequent symptoms are: rapid and feeble or irregular, intermittent pulse; cardiac palpitation and sensations of oppression and uneasiness; sighing respiration, dyspnœa, and syncope; dropsy, turgescence of the veins, and congestion of the various organs, causing œdema of the lungs, jaundice, or albuminuria.

The most important *signs* are: feeble and irregular action of the heart; an enlarged area of dulness, oval in form, and not extending far to the left of the apex beat; and feebleness of the heart sounds.

On inspection, the impulse of the heart's apex may not be visible. If seen at all, it is likely to extend over a wider area than in health, and the point of maximum intensity is not easily determined. It is occasionally of an undulatory character.

Permanent dilatation and varicosity of the jugular veins is a sign of a dilated right auricle.

By palpation, the apex beat is found below the normal position and to the left of it, and the heart's action is irregular in rhythm. The impulse is feeble, which enables us readily to distinguish this affection from hypertrophy, or hypertrophy with dilatation. A purring tremor may frequently be obtained, especially when there is mitral regurgitation.

Percussion shows the area of cardiac dulness increased to the right when the right cavities are involved, and to the left when the left cavities are dilated; dulness due to dilated auricles may extend upward, even to the first interspace.

This area maintains an oval outline, which enables us to distinguish

the disease from pericarditis, in which the signs, upon inspection and palpation, are nearly identical.

By auscultation both sounds of the heart are found short, abrupt, and feeble, and frequently of equal length. The second sound may be inaudible at the apex, and the first may be reduplicated.

If valvular murmurs have been present, these become less intense, and sometimes of a swirling character. The respiratory sounds over the upper portion of the left lung are often feeble.

DIAGNOSIS.—There is usually little difficulty in distinguishing dilatation of the heart from all other affections, excepting pericarditis. The distinctive features between these two are as follows:

DILATATION OF THE HEART.	PERICARDITIS.
History.	
Chronic.	Acute.
Palpation.	
Impulse feeble and irregular, felt *below* and to the left of its normal position, and not materially affected by leaning the patient's body forward.	Impulse feeble and irregular, felt *above* its normal position, and increased in force when the patient leans forward.
Percussion.	
Oval outline of dulness, which does not extend far to the left of the apex.	Triangular outline of dulness, which extends considerably to the left of the apex beat.
Auscultation.	
Heart sounds feeble, short, and valvular, and not altered by position.	Heart sounds feeble, and not so markedly valvular, but intensified by leaning the body forward.

Asystolism is a term which has been applied to a condition in which the ventricle cannot completely empty itself. It is nearly always associated with dilatation of the right ventricle.

In this condition, the impulse of the heart becomes very feeble, and shortly before death the valvular sounds or murmurs which may have been present become almost inaudible, or they may be supplanted by a continuous humming sound. Tricuspid regurgitation, with pulsation in the jugular veins, is likely to be developed during the course of this affection.

PROGNOSIS.—The prognosis is unfavorable according as dilatation relatively exceeds compensatory hypertrophy, the gravity depending upon degenerations of the muscular wall, and upon the degree of obstruction to circulation. When compensation is good and no complications exist, the patient may live for years; but associated valvular lesions, pulmonary affections, Bright's disease, general anæmia, hereditary predisposition

to heart disease, and weakness from any cause render the prognosis unfavorable.

Dyspnœa and irregular and intermittent pulse, tendency to dropsy, and syncope are grave signs, indicating that death may occur suddenly at any time, though the patient may linger for several months.

TREATMENT.—The treatment of dilatation of the heart and of asystolism should be the same as that recommended for chronic endocarditis with valvular disease of the heart.

Though the dilated cavities cannot be reduced to normal, compensatory hypertrophy of the walls may be induced and should be encouraged by avoiding all unnecessary exertion; by improving general nutrition with an abundance of easily digested food, tonics, and regulation of excretion; and by careful stimulation of the heart by digitalis, strophanthus, sparteine, convallaria, or caffeine, and in suitable cases by moderate exercise.

ATROPHY OF THE HEART.

Synonym.—Phthisis of the heart.

Atrophy of the heart is an extremely rare affection. It consists of simple attenuation of the walls of the heart, the cavities usually remaining of normal size, but in some cases both the thickness of the walls and the size of the cavities are diminished.

The affection is sometimes congenital. It may be caused by old age, chronic wasting disease, or by constriction of the coronary arteries.

DIAGNOSIS.—A diagnosis can rarely, if ever, be made during life; but in the congenital variety we may possibly detect decreased area of cardiac dulness independent of pulmonary emphysema.

FATTY HEART.

There are two recognized varieties of fatty heart: one, in which there is a deposit of fatty tissue upon the surface of the heart or between its muscular fibres (infiltration), and the other, in which the muscular fibres themselves undergo fatty degeneration.

ETIOLOGY.—The first variety of fatty heart is attributed, by Kennedy, to a fatty diathesis, and is associated with obesity; the second variety results from atheromatous degeneration of the aorta, old age, alcoholism, gout, or some prolonged wasting disease.

SYMPTOMATOLOGY.—The symptoms of fatty disease of the heart are practically the same in both varieties, and they are of the greatest importance from a diagnostic point of view. The most prominent of these are: melancholia or irritability of temper, partial loss of memory, or hesitating speech; palpitation of the heart, dyspnœa, and angina pectoris. Other symptoms which are frequently noticed are: pallor and a

sallow appearance of the surface, with congestion of the ears and lips; weight and pain in the head; a sense of pain in the epigastrium; double vision or loss of vision; and the arcus senilis. Pseudo-apoplexy, and Cheyne-Stokes respiration, when present, are symptoms of the greatest value.

Pseudo-apoplexy consists of attacks in which the individual suddenly loses consciousness and falls. It differs from true apoplexy in the rapidity of recovery. When these attacks first make their appearance, they seldom continue more than a minute or two, the patient coming out of them feeling perfectly well; but, as the disease progresses, they become more and more frequent, prolonged, and severe, and are attended with paralysis; even then the patient usually recovers completely in a few days at most.

The Cheyne-Stokes respiration, which appears late in the disease, consists in the occurrence of a series of inspirations increasing to a maximum, and then declining in force and length until a state of apparent apnœa is established. In this condition a patient may remain for such a length of time as to make his attendants believe him dead, when a low inspiration, followed by one more decided, marks the commencement of a new ascending and descending series of inspirations. Although this is an important symptom of fatty heart, it must not be forgotten that it occasionally occurs in dilatation and in valvular disease of the organ.

In fatty infiltration of the heart, obesity is a symptom of importance. In fatty degeneration, loss of weight, after a person has been fleshy, is a valuable symptom.

Among the *signs* of *fatty infiltration of the heart,* are: a pulse usually slow—forty or fifty per minute—full, and sometimes even bounding; increased area of cardiac dulness on very careful percussion.

In *fatty degeneration,* the pulse is weak and irregular and usually rapid. Auscultation over the apex will occasionally reveal slow pulsation; and even when the pulsation equals seventy per minute, it often conveys to the ear a sense of slowness.

The impulse of the apex is weak, and the intensity of the sounds feeble in either variety. If valvular disease coexists, a soft systolic *souffle* may sometimes be detected by careful auscultation over the aorta.

On inspection and palpation, the impulse is either indistinct or absent; the apex remains in its normal position. The pulse in fatty deposit is slow and full; in fatty degeneration it may be slow or rapid, but it usually appears to be rapid at the wrist, even though the heart is beating slowly.

By percussion, the heart is found of normal size in uncomplicated fatty degeneration, but slightly enlarged in fatty deposit.

In auscultation, the first sound is frequently absent, but if present it will be feeble, short, and valvular, having lost nearly all of its muscular element. The second sound is usually short, clacking, and distant.

A soft, blowing murmur may frequently be heard over the aorta with the first sound, especially if the patient is in the recumbent position.

Exceptional.—Sometimes the heart sounds in this disease are like those of the fœtus *in utero*. Sometimes they are metallic or ringing, and it is said that the second sound is sometimes prolonged and intensified.

Stokes considered the occurrence of pseudo-apoplexy with a soft *souffle* in the aortic area, with the first sound of the heart, and a slow pulse positive evidence of fatty degeneration of the heart; but these signs are seldom combined in the same individual.

A combination of several of the important symptoms and signs which have been enumerated is often present, and may justify a positive diagnosis.

DIAGNOSIS.—The physical signs are not always well marked, and a positive diagnosis is often impossible. Fatty heart is most likely to be mistaken for functional affections of the organ, from which it can only be distinguished by careful scrutiny of the symptoms and signs already enumerated, and the exclusion of hysterical affections and other functional causes. A distinction may sometimes be made by causing the patient to walk briskly, when if the trouble is functional the heart's action becomes more regular and stronger and the sounds more distinct, whereas if organic changes are present the pulsations become more irregular and feebler than before.

PROGNOSIS.—The prognosis is unfavorable in fatty degeneration. Fatty infiltration, when excessive, will produce degeneration of the muscular fibres from pressure; much can be done in mild cases by a proper system of diet and exercise. In either case, but especially in fatty degeneration, death by syncope is apt to occur suddenly and without warning, from excitement, overexertion or distention of the stomach or bowels by a too hearty meal or flatulence.

TREATMENT.—The general treatment consists of cardiac and general tonics and is the same as for valvular diseases. Patients should be cautioned to avoid doing anything which causes dyspnœa.

Arsenious acid is one of our best remedies in cardiac degeneration, as it not only increases the power of the heart, but also relieves the neuralgic pains, which are among the most distressing symptoms of this disease. When the affection consists of fatty deposit on the surface of the heart, or between its muscular fibres, much may be accomplished by regulating the diet. The patient should live principally on lean meat, avoiding as far as possible all fat-producing food, such as sugar, starch, and alcoholic stimulants. He should take as little fluid as possible, and should wear warm woollen clothing, even in summer, to favor free diaphoresis, and should take systematic gentle exercise. These measures will lessen obesity and strengthen the weak muscles.

ANEURISM OF THE HEART.

Aneurism of the heart is a rare affection, consisting of bulging of that portion of the cardiac walls which has been softened by inflammation. It usually occurs at the apex of the left ventricle, and occasionally involves the interventricular septum, bulging into the right cavity. Rarely, it includes nearly the entire ventricular wall, which in such cases is thin and dilated, and chiefly fibrous from loss of muscular fibre. Occasionally it is sacculated, sometimes reaching the size of a cocoanut, and connected with the ventricle by a narrow neck. The walls vary up to a quarter of an inch in thickness. The endothelium, though atrophied, usually remains intact. Commonly old stratified clots line its interior.

ETIOLOGY.—Cardiac aneurism may develop from any condition which weakens the wall of the heart, such as disease of the coronary arteries, fatty, fibroid, amyloid, or atrophic degeneration, or abscess whether or not the sequelæ of myocarditis, endocarditis, or pericarditis.

DIAGNOSIS AND PROGNOSIS.—A diagnosis can seldom be made before death, which usually occurs from rupture or heart failure due to weakening of the muscle or mechanical interference with its action.

TREATMENT.—The treatment must be entirely symptomatic; when there is much cardiac pain, rest, and potassium iodide in moderately large doses are most efficient. There are no symptoms or signs to distinguish cardiac aneurism from myocarditis.

RUPTURE OF THE HEART.

Rupture of the heart may follow myocarditis or fatty degeneration of the heart. In the latter case, it seldom occurs in persons less than sixty years of age.

SYMPTOMATOLOGY.—The symptoms are: sharp, sudden pain in the præcordial region, faintness, collapse, and speedy death; though some patients have lived forty-eight hours after the accident.

Death is usually so sudden that an examination cannot be made, but the signs must of necessity be those of distention of the pericardium by fluid, with extreme weakness of the heart. Treatment would be unavailing.

SYPHILITIC DISEASE OF THE HEART.

A few cases have been observed where heart disease seemed to have resulted from constitutional syphilis. Syphilitic affections of this organ consist of fibrinous exudations into the connective tissue, which may either soften and suppurate, forming ulcers or small abscesses, or be converted into masses of hardened fibroid tissue; and it is not improbable that, as suggested by Corvisart, vegetations on the valves may in some cases have a syphilitic origin. An accurate diagnosis is impossible. No treatment can be suggested where a diagnosis cannot be made.

TUMORS OF THE HEART.

The heart is very seldom the seat of neoplasms. Congenital angiomata may exist in its walls; sarcomata and carcinomata may penetrate it from adjacent organs. Hydatids are rarely found. Of these no *diagnosis* can be made. The *prognosis* is necessarily unfavorable in the case of progressive tumors. The *treatment* must be symptomatic.

MORBUS CÆRULEUS.

Synonyms.—Cyanosis, the blue disease.

Morbus Cæruleus is the result of congenital malformation of the heart. Cyanosis, usually marked in the cases, is ascribed to general venous congestion due to obstruction in the right heart, but it has also been supposed to result from admixture of venous with arterial blood. The morbid conditions, found post mortem, may be patency of the ductus arteriosus or foramen ovale, deficient interventricular septum or narrowing or complete closure of the pulmonic orifice. Two or more of these abnormities are not infrequently combined, the first mentioned being the defect most often present.

Symptomatology.—The unfortunate subjects are usually small and feeble young children. Cyanosis may be slight or it may amount to a deep purple or blue color. It occurs early, but may vary at different times. The superficial temperature is low, giving rise to chilliness. Cough, dyspnœa, and frequent attacks of palpitation are common, appearing after or increased by exertion or excitement.

As *signs*, inspection, in addition to the blueness of the surface, often reveals præcordial bulging and abnormal pulsation diffused to the epigastrium. By palpation, especially at the base of the heart, a thrill may be obtained. Percussion shows enlargement of the right heart; dulness, according to Gerhardt, may often be elicited along the left side of the sternum, as high as the second rib, owing to the enlargement of the conus arteriosus and distention of the pulmonary artery. Auscultation may discover a systolic murmur over the region of the pulmonary artery, and rarely a diastolic murmur. A systolic murmur during the first three years of life is said to be invariably of congenital origin.

Diagnosis.—In the London *Lancet*, May, 1879, Sansom formulates the following propositions relating to the diagnosis of congenital disease of the heart in children.

First, in cases of congenital cyanosis, in which no cardiac murmur is manifest, there is probably patency of the foramen ovale.

Second, in cases of cyanosis with murmur varying at intervals, and heard over the sternal ends of the third and fourth costal cartilages and intercostal spaces, there is probably patency of the foramen ovale.

Third, in cases of cyanosis with loud, unvarying systolic murmur, with maximum intensity internal to the position of the apex beat, but

heard also at the back between the scapulæ, there is probably imperfection of the ventricular septum.

Fourth, in cases of cyanosis and of marked anæmia, in children who manifest a pronounced superficial systolic murmur at the base of the heart, there is probably constriction of the pulmonary artery at its orifice. Such murmurs may be associated with anæmic murmurs which are heard above the clavicles.

Fifth, in cases of congenital affection of the heart in which there is evidence of considerable dilatation of the left chambers, it is probable that endocarditis affecting the valves has constituted a complication.

PROGNOSIS.—Most subjects of congenital malformation of the heart live but a few hours or days after birth, and very rarely reach advanced age. The prognosis is best in cases of congenital stenosis of the pulmonary artery.

TREATMENT.—No specific treatment can be recommended, but the same general rules should be observed as in cases of valvular disease of the heart.

NEUROTIC OR FUNCTIONAL DISEASE OF THE HEART.

Functional disorders of the heart are characterized by peculiar sensations and by change in the frequency, force, or rhythm of the pulse and apex beat, and in the character of the heart sounds, several of these being commonly associated.

The affection ordinarily manifests itself by frequent paroxysmal attacks of palpitation and irregularity of the heart's action. It is aptly stated by Balfour, that if a patient come complaining of disease of the heart who has not obtained the opinion of a physician, we may, in the majority of cases, assure him that it is only a functional affection, and that no organic disease exists; for the latter generally escapes notice until detected by the physician.

ETIOLOGY.—The variations from the normal conditions may be transient and paroxysmal, or more or less constant. They may arise from emotional causes, as, joy, fear, or shock, and from hysteria, or hypochondriasis. They are often associated with chorea, exophthalmic goitre, and other functional nervous derangements. They may result from over-exertion, from the exhausting influence of acute diseases, or from reflex irritation, especially of gastric, hepatic, or intestinal origin, or from excessive venery. They may be due to anæmia or to poisonous agencies acting through the circulation, whether referable to lithæmia, gout, rheumatism, lead poisoning, or inordinate use of alcohol, tobacco, tea, and coffee. Heredity and the nervous diathesis are also potent factors in their causation.

SYMPTOMATOLOGY.—Cardialgia and palpitation or a subjective sensation of the cardiac impulse, are the most constant symptoms of func-

tional disease, and usually give rise to much anxiety. Abnormally rapid pulse (tachycardia) or abnormally slow pulse (bradycardia), or irregularity, intermittency, weakness, or fulness of its beat, and morbid præcordial sounds and sensations frequently occur. Vertigo, tinnitus aurium, and photophobia are not uncommon, and marked pseudo-angina pectoris may occur.

Though the physical *signs* of the neurotic affection are in no way characteristic, physical diagnosis is of importance in excluding organic disease.

By inspection and palpation we find the apex in its normal position, but usually the impulse is comparatively feeble, though the stroke may seem sharp and quick. The action of the heart is usually irregular.

Percussion shows the heart to be of normal size.

In auscultation, both sounds of the heart are abrupt, and may be intensified. Occasionally the first sound has a metallic character. Frequently anæmic murmurs are found in the aortic area, and also in a space which has been improperly termed the pulmonary area, viz., a limited area, an inch or an inch and a half to the left of the sternum, in the second intercostal space. The murmur in the latter position is apparently due to slight mitral regurgitation dependent upon a weakened condition of the left ventricle which allows dilatation to such an extent that the mitral valves are unable completely to close the auriculo-ventricular orifice. In such cases the dilatation disappears, and the murmur ceases as the muscles regain their tonicity.

Diagnosis.—It is of great importance to make an accurate differential diagnosis between functional and organic heart disease. The chief points of distinction have been already noted in the differential diagnosis of chronic endocarditis.

The symptoms of functional disease of the heart may be associated with the signs of organic lesions merely as a coincidence. In such instances an exact diagnosis would be extremely difficult. It could only be made by repeated careful examinations and by the evidence afforded by treatment, under which many of the functional symptoms may disappear.

Prognosis.—Functional disorders of the heart usually continue for months or even years unless the cause can be ascertained and removed by proper treatment, but they are seldom if ever dangerous to life, if true angina pectoris be excepted.

Treatment.—The first thing in these cases is to impress upon the patient the fact that his heart symptoms are not due to organic disease, and that he is likely to recover entirely. This must be done *after* a careful and painstaking examination. Since neurotic affections of the heart are usually due to anæmia, hysteria, uterine irritation, sexual abuses, or the excessive use of alcoholic stimulants, or of tobacco, or of tea and coffee, we should ascertain which of these operates in the case before us, and advise accordingly.

General tonics are usually indicated. In a few cases digitalis will be found serviceable in controlling the action of the heart, but sparteine sulphate gr. ¼ to i., tinct. of strophanthus ♏v. to x., tinct. of convallaria ♏x. to xv., or fl. ext. of cactus grandiflora ♏i. to iv., three times a day are, as a rule, more efficient. In many cases strychnine and in others bromides are specially beneficial, and occasionally nitroglycerin, amyl nitrite, aconite, or veratrum viride may be beneficially employed in small doses.

TACHYCARDIA.

Tachycardia is a term which may be broadly applied to an abnormal rapidity of the heart, occurring either as a paroxysmal or as a more permanent affection, whether or not accompanied by weakness, irregularity, or intermittency of the pulse. The pulsations may run from one hundred and twenty to even three hundred per minute. If the action is rapid and the impulse forcible, it is commonly termed palpitation.

Tachycardia may be a symptom of organic or of functional disease; it also occurs as an idiopathic affection and is occasionally hereditary.

In some instances of paroxysmal tachycardia as described by L. Bouveret (*International Medical Annual,* II, p. 252) in a report of eleven collected cases, the heart, normal in the intervals, is seized with paroxysms of rapidity, which, if the attack be of short duration, may reach two and even three hundred beats a minute. If these attacks are prolonged for several days, symptoms of cerebral hyperæmia with embarrassment of the pulmonary and systemic circulation commonly appear. In such cases, change to the normal action may occur suddenly, and may be followed by decided prostration. Four out of the eleven cases died of asystole or syncope. Instances of hereditary tachycardia have been known in which the heart beat with infantile rapidity through life seemingly without detriment to the individual.

The so-called *irritable heart* of soldiers so well described by Da Costa (Medical Diagnosis, page 405) is characterized by habitual rapidity complicated by paroxysms of palpitation and præcordial pain brought on by exercise, with frequent attacks of headache, dizziness, and cutaneous hyperæsthesia.

With the paroxysmal form of tachycardia in addition to the palpable and visible rapidity of the cardiac impulse, physical exploration may elicit signs of pulmonary congestion. In irritable heart, Da Costa says the action is rapid, often irregular and rather abrupt and jerky, the first sound short and sharp like the second, but sometimes very faint.

Prognosis.—In severe paroxysmal cases, the prognosis is uncertain, varying with the persistence, frequency and severity of the attacks.

Treatment is that suited to functional disease.

BRADYCARDIA.

Bradycardia or abnormal slowness of the pulse though often seen in slight degree, is much rarer as a well-marked characteristic than rapid pulse. The frequency may fall as low as seventeen beats per minute (Balfour, *Edinburgh Medical Journal*, 1890). In one variety both heart and pulse beat alike, in another the pulsations of the heart while normal in frequency at the apex are so weak that all are not felt at the wrist. Prentiss' classification of the causes of slow pulse is as follows: disease or injury of the nerve centre causing paralysis of the sympathetic nerve or irritation of the pneumogastric nerve; disease or injury to the trunk of the vagus, increasing its irritability; disease or injury paralyzing the sympathetic; disease of the cardiac ganglia; disease of the heart muscles; action of poisons upon the nerve centre or endings (*International Medical Annual*, 1891). I have seen a few cases that seemed the direct result of prolonged severe pain. When well marked, it is usually an unfavorable sign, owing to the tendency to pseudo-epileptic and pseudo-apoplectic attacks. Death may occur during these seizures or from asthenia. It may be a symptom of fatty heart.

TREATMENT must aim at general nervous and cardiac stimulation.

ANGINA PECTORIS.

Angina pectoris is a term applied to attacks of severe paroxysmal cardiac pain, associated with a sense of impending death and minor phenomena commonly symptomatic of serious organic lesions. A distinction is to be drawn clinically and etiologically between true angina pectoris of organic origin and pseudo-angina or hysterical angina dependent upon diathetic or toxic influences. True angina most frequently attacks men of advanced years, but the false variety is commonly found in comparatively young neurotic women.

ETIOLOGY.—True angina pectoris seems in most cases to depend upon arterio-sclerosis and other diseases of the coronary arteries tending to their contraction or obliteration, and consequent deficient nutrition of the heart. According to Liégeois (*Bulletin médicale des Vosges*, 1888) three-fourths of all cases may be assigned to sclerosis or atheroma of the coronary arteries or aorta. Not infrequently the affection appears to depend upon cardiac dilatation, valvular disease, fatty and other degenerative changes, aneurism, or pericarditis, any of which may disturb the circulation through the coronary arteries. Douglas Powell believes vasomotor disturbance an essential factor in the majority of cases of angina pectoris (*British Medical Journal*, 1891). Sometimes no cause for the disease can be discovered. Among possible causes may be mentioned organic affections, such as cancer involving the pneumo-gastric or cardiac and

thoracic plexus of the sympathetic (*Lyon Médicale*, 1888), chronic neuritis and pigmentary and granular degeneration of nerve cells (*La Semaine Médicale*, March, 1890). The immediate cause of the paroxysm may be embolism of the coronary artery, but it is usually some mental or physical exertion, sexual derangement, error of diet, or excess, influencing the vasomotor mechanism. Occasionally the gouty and rheumatic diatheses, by vitiating the blood supply, are undoubted etiological factors both in producing the primary disease and in favoring the paroxysm. Pseudo-angina may be due to reflex causes or to direct central irritation. The former are commonly of gastric or hepatic origin, such as indigestion, gastric catarrh, flatulence, or the presence of gall stones; the latter include cerebral and spinal neurasthenia and locomotor ataxia.

SYMPTOMATOLOGY.—The most characteristic symptoms of true angina pectoris are agonizing sternal or præcordial pain probably caused in most cases by over-distention of the heart, with a peculiar fear of impending death. This pain usually radiates to the left shoulder and down the arm, often stopping at the elbow, but frequently extending to the ring and little finger. It is often severe up the side of the neck and behind the ear. It sometimes extends to the right side and may occasionally be felt in the thighs. The pain has been variously likened to a stab, a thrust with a red-hot iron, a sensation of suffocation, or grip of an icy hand. Pallor and fear are depicted on the countenance, and respiration is frequently interrupted as though the sufferer had forgotten to breathe. The pulse is usually, though not always, feeble and irregular or intermittent. The duration of acute attacks is usually from half an hour to two or even three hours, and they not infrequently terminate in syncope or death. If the patient survives the first attack others are liable to occur at irregular intervals, at first far apart, but ere long nearer together until one finally proves fatal. Attacks of pseudo-angina are generally of longer duration but of less severity.

No characteristic *signs* accompany either variety of the affection, but valvular disease, fatty degeneration, or dilatation of the heart is commonly present in true angina.

DIAGNOSIS.—Angina pectoris proper may be confused with the hysterical form, or, if mild, may possibly be mistaken for intercostal neuralgia, acute pleurisy, or myalgia. It may be distinguished from pseudo-angina pectoris by the following points:

TRUE ANGINA PECTORIS.	HYSTERICAL OR PSEUDO-ANGINA PECTORIS.
History.	
Usually in men over forty; cardiac lesions, especially arterio-sclerosis of the coronary arteries and fatty degeneration. Attacks caused by exertion any time of day.	Oftenest in women; any age; neuralgic diathesis, but no cardiac lesions. Attacks spontaneous; usually at night.

True angina pectoris.	Hysterical or pseudo-angina pectoris.
Symptoms.	
Pain very severe and of short duration.	Pain less severe and of longer duration.
Comparative silence and immobility; often speedily fatal. Not relieved by anti-neuralgic remedies.	Comparative agitation and activity; seldom if ever fatal. Relieved by anti-neuralgic medication.
Signs.	
Murmurs and enlargement frequent.	No organic disease.

It may be differentiated from *intercostal neuralgia* by the history and presence of the characteristic painful points in the latter disease. In *myalgia*, the character and seat of the pain, the tenderness of the muscles, and other symptoms are sufficiently diagnostic. The pain of *acute pleurisy* is attended by cough, pyrexia, and distinct physical signs not present in angina.

Prognosis.—The first attack of angina pectoris is often fatal within two or three hours, and sometimes a sudden sharp pain is the only warning of instant death. More frequently the patient survives the first paroxysm, but after a few months dies in the second or third attack. Sometimes patients live for many years subject to occasional attacks which gradually become more and more frequent until finally resulting in death. A considerable number, however, recover under appropriate treatment or at least live many years with but few and light attacks of the cardiac pain. In pseudo-angina, the prognosis is favorable providing its cause can be removed.

Treatment.—For the paroxysms, alcoholic stimulants, opiates, or inhalations of amyl nitrate ♏v. to vi., or of chloroform are most efficient. Chloroform, though apparently a dangerous remedy, has proved harmless, prompt, and efficient when administered as recommended by G. W. Balfour, of Edinburgh (Clinical Lectures on Diseases of the Heart, 1876). Half a drachm is poured upon a sponge at the bottom of a wide-mouthed bottle, from which the patient may breathe *ad libitum* until relieved. The patient drops the bottle as soon as he becomes partially unconscious, and it rolls away. Nitroglycerin has been recommended for the cure of angina pectoris, and from the published reports it appears that numerous cases have been benefited by it. I have found it of much value in stimulating the heart and relieving the painful paroxysm, but I have not witnessed curative results. It is administered either in pill, tablet triturate, or solution. The dose administered to relieve the paroxysm is ordinarily gr. $\frac{1}{100}$, which may be repeated once in twenty minutes until three or four doses have been taken or relief is obtained, unless its physiological effects are too strongly developed.

When the susceptibility of the patient to the remedy has been ascertained, doses two or three times larger may sometimes be given. To prevent recurrence of the attack, it may be given three times daily, at first in doses of gr. $\frac{1}{100}$, but these may be increased to five, ten, or even fifteen times as much, providing that it does not cause severe headache, giddiness, or overpowering somnolence. During the intervals between the attacks of angina, the same hygienic rules should be observed as in valvular disease. Arsenious acid should be given in moderate doses, with or without iron, strychnine, and digitalis, accerding to special indications.

Huchard claims that large doses of potassium iodide (grs. xl. to l. daily) continued several years with intervals of eight or ten days each month during which it is suspended, will cure angina pectoris and arteriosclerosis of the heart (*Gazette des Hôpitaux*, 1890). The remedy is certainly very efficient in relieving the pains of aneurism and sometimes in relieving cardiac pain. In pseudo-angina, the cause must be ascertained and removed if possible. Remedies usually should be directed to the relief of rheumatism, anæmia, or debility, or, most important, to the correction of indigestion.

CHAPTER XV.

CARDIAC AND ARTERIAL DISEASES.—*Continued*

AORTITIS.

THE symptoms ascribed to acute exudative inflammation of the aorta have been described by Frank, Bizot, and others; but as stated by R. Douglass Powell, the disease as a primary affection is of very doubtful, if not impossible, occurrence. We need not attempt to describe any of the signs or symptoms it might possibly occasion.

ATHEROMA OF THE AORTA.

Synonyms.—Aortic endarteritis; atheromatous degeneration of the aorta.

Atheroma of the aorta may be defined as a degeneration of the coats of the aorta, consisting of an irregular thickening and softening of its walls, especially of its inner coat.

It seldom occurs before the forty-fifth year of age.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—The disease consists of thickening and fatty degeneration, usually followed by calcareous infiltration and occasionally by ulceration. It is primarily confined to the intima, but not infrequently involves the muscular coat. It begins with inflammation, occurring in scattered patches, which have the milky opacity characteristic of the first stage of acute endocarditis; later these become yellow from fatty change. These areas may coalesce to some extent, and deposits of lime salts commonly take place, giving the surface a scaly or nodular appearance and chalky hardness. Ulceration occasionally results from rapid central softening of the patch and discharge of the débris. Microscopically, the thickened intima early shows round and spindle cell infiltration and more or less increase of fibrous elements, but no blood-vessels. Later the spots of softening are found to contain oil globules, crystals of cholesterin, and a granular débris. These processes result at first in thickening of the arterial wall, finally weakening, loss of elasticity, dilatation, and in some cases aneurism.

The affection is usually limited to the initial portion of the blood-vessel; indeed clinical evidence of its existence beyond the transverse portion of the arch is very rare.

ETIOLOGY.—The chief causes are: gout, rheumatism, syphilis, chronic

nephritis, high living with insufficient exercise, and the excessive use of alcoholics. It sometimes results from undue strain of the artery, as in excessive muscular efforts.

SYMPTOMATOLOGY.—The symptoms of atheroma of the aorta are always obscure, and its physical signs, in many cases, are far from positive. Among the most prominent symptoms and *signs*, we observe attacks of palpitation or anginal pain and dyspnœa, which are usually brought on by exercise, but may occur independent of exertion. During these attacks the pulse is commonly very weak. Signs of general atheroma may often be detected in the abnormal rigidity of the temporal, radial, and brachial arteries.

By inspection and palpation, when dilatation has taken place, feeble pulsation may be seen or felt in the second intercostal space close to the sternum, on the right side.

Upon percussion, there is found a somewhat increased area of dulness over the ascending or transverse portion of the aorta.

On auscultation early in the disease, there may be some evidence of hypertrophy of the left ventricle, as indicated by an increased impulse and muffling of the first sound of the heart. These signs, however, are not characteristic, as they might arise from emphysema or other cause of obstructed circulation.

With the advent of dilatation, the first sound of the heart becomes more indistinct, while there is accentuation of the second sound over the aortic valves, thought by some to be diagnostic of dilatation of the aorta. A short murmur is usually heard over the aorta, immediately after the systole of the ventricles, especially when the action of the heart is rapid. As dilatation progresses, the bruit becomes more distinct. It is sometimes rough in character, and may be associated with a purring tremor.

The second sound may be partially supplanted by a faint diastolic murmur, due to dilatation at the origin of the artery, which renders the semilunar valves incompetent to close the orifice, and allows regurgitation into the ventricles.

When the heart is beating slowly and regularly, both the first and second sounds may be accentuated over the upper part of the sternum, and the systole of the heart may be attended by a slight impulse in the aortic area; but this latter sign, to be of value, must be obtained when the patient is perfectly quiet.

Later in the disease, dyspnœa becomes marked, the attacks of angina are more frequent and persistent, and the symptoms of embolism, such as hemiplegia, rigors, hæmaturia, superficial hemorrhages, or gangrene, may make their appearance; or the formation of a sacculated aneurism from the affected portion of the artery may be indicated by the sudden occurrence of pain, dyspnœa, and faintness. Finally, sudden death may result from heart failure or from rupture of the aorta.

DIAGNOSIS.—The principal symptoms and signs of atheroma of the

aorta are: palpitation, pain, and dyspnœa, with rigidity of the superficial arteries, muffling of the first sound of the heart, and accentuation of the second, over the aortic valves. The first heart sound is usually followed by a more or less distinct systolic murmur. Sometimes there is a diastolic murmur in the region of the ascending or transverse portion of the arch of the aorta, with slight increase in the area of dulness during the later stages. The affection might be mistaken for simple disease of the aortic valves, or inorganic disease of the heart, with anæmic murmurs.

Though it may cause many of the symptoms and signs of atheroma, *disease of the aortic valves* is not attended by a rigid condition of the superficial arteries, or the peculiar neuralgic pains which usually attend atheroma, and it does not cause accentuation of the second sound at the aortic valves or an increased area of dulness at the base.

When *anæmic murmurs* are associated with functional disease of the heart, they are not attended by rigidity of the superficial arteries, by the peculiarly distinct accentuation of the second sound, by the systolic shock, by the diastolic bruit, or by increased area of dulness.

TREATMENT.—Morphine, nitroglycerin, or other anti-spasmodic remedies are indicated during the attacks of dyspnœa. Potassium iodide continued for months, with short intermissions, is sometimes useful. Excessive exertion must be avoided.

AORTIC OR THORACIC ANEURISM.

An aneurism is a sac the cavity of which communicates with the lumen of the artery.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—Aneurism may exist as a fusiform dilatation of the artery, but usually, when well marked, it is saccular, forming a pouch-like projection from the vessel. The wall of the aneurism may be composed of all the coats of the vessel, though commonly the muscular tunic is wanting. Rarely, the walls are formed by a condensation of the surrounding tissues into which the artery has ruptured, called *diffuse aneurism*. If the blood effects separation of the arterial coats, a *dissecting aneurism* is formed. The cavity is generally lined with concentrically stratified blood clots of varying age, thickness, and consistence, which are occasionally calcified. As the aneurism enlarges, pressure upon adjacent respiratory, circulatory, nervous, or bony structures produces characteristic symptoms and may eventually effect their destruction. The walls of the sac generally undergo atheromatous degeneration, and may rupture into the pleural cavity, lungs, bronchi, trachea, pericardium, œsophagus, or through the chest wall.

ETIOLOGY.—Aneurism occurs generally in adults, oftenest between the ages of forty and fifty. Occupations which subject the individual to exposure and severe bodily strain favor its development. Atheroma

of the walls of the artery is the chief predisposing cause, whether due to syphilis, chronic nephritis, gout, rheumatism, chronic alcoholism, lead or mercurial poisoning, or to several of these combined. The immediate cause may be sudden strain, a blow, fall or wound, or continued excesses.

ANEURISM OF THE SINUSES OF VALSALVA.

Aneurism of the sinuses of Valsalva is usually so small as to give rise to no peculiar symptoms or signs, but the indications of atheromatous degeneration, with a pulmonary systolic or diastolic murmur due to pressure of the aneurism on the origin of the pulmonary artery, might lead us to suspect the true nature of the lesion. The diagnosis can rarely, if ever, be made with certainty, as the tumor lies enveloped in the pericardium, so close to the heart that it is almost impossible to distinguish between the murmurs which it produces and those of valvular origin.

ANEURISM OF THE ARCH OF THE AORTA.

Aneurism of the arch of the aorta consists of preternatural dilatation of the artery, which may be general involving the whole circumference in a fusiform, cylindrical or globular swelling; or sacculated, forming a pouch-like projection from one side of the artery.

Sacculated aneurisms are usually globular at first, but may subsequently acquire different forms, especially the conical.

Aneurisms may occur in the ascending, transverse, or descending portion of the arch of the aorta. About one-half have their origin in the ascending portion; a few involve both the ascending and the transverse, or simply the transverse portion of the arch. Nearly one-fourth arise from the descending arch, and about the same number from that portion of the aorta between the arch and the diaphragm.

ANEURISM OF THE DESCENDING AORTA.

Aneurism of the descending aorta ultimately causes a pulsating tumor behind, at the left of the spinal column, between the third dorsal vertebra and the point at which the aorta perforates the diaphragm. Erosion of the vertebræ, with consequent curvature of the spine, is usually produced by pressure. Subsequent compression of the spinal cord may cause paraplegia. The tumor, if large, usually displaces the heart forward and to the right. In exceptional instances, aneurisms of this portion of the aorta may be detected upon the right side of the spinal column. The bruit, in an aneurism of the descending aorta, may be distinguished from a mitral regurgitant murmur, frequently heard in a similar position, by the fact that the aneurismal murmur is heard not only between the fifth and the eighth dorsal vertebræ, but also above and below this position. The mitral regurgitant murmur is not heard

distinctly above the lower border of the fifth or below the upper border of the eighth vertebra.

Symptomatology.—Tumors of this character may sometimes be diagnosticated from the symptoms, when they cannot be located by the physical signs. The more prominent symptoms, though not individually characteristic, may be sufficient for the purpose of diagnosis when grouped together, and are of great value when taken in connection with the physical signs. Enumerated nearly in the order of their importance, they are: pain, dyspnœa, palpitation, dysphagia, headache, and disordered vision.

The *pain* in aortic aneurism is persistent, of a peculiar wearing, aching, or burning character, and is referred to the region of the tumor. Frequently there are neuralgic exacerbations, with pain radiating in the course of contiguous nerves.

Dyspnœa of varying degree is generally present, and is usually aggravated by much slighter causes than those which would occasion the same symptom in other varieties of intrathoracic tumors. It frequently occurs in severe paroxysms, which may be due to one or more causes. Ordinarily, such attacks are ascribed to spasm of the glottis, resulting from irritation of one or both of the recurrent laryngeal nerves. More probably they are due to paralysis of the abductor muscles of the glottis which are supplied by these nerves, with consequent falling together of the vocal cords, and obstruction of the glottis during inspiration.

The exacerbations of this symptom are due in some instances to a collection of mucus at the glottis; in others to the varying pressure of the aneurism upon the nerve which, at one time, completely suspends its function, at another interferes with it but slightly. The voice is also modified more or less by the same cause, and may be entirely lost.

Dyspnœa is sometimes dependent upon narrowing of the trachea or of the bronchi from pressure of the aneurism. In such instances, the paroxysms are probably due to a collection of mucus which the patient may be unable to expectorate at the point of stricture.

Palpitation of the heart is generally produced by slight exertion; it may be due to irritation of the sympathetic nerve or paralysis of the vagus from pressure.

Dysphagia, due to pressure upon the œsophagus, is often present, though it is a less frequent symptom with aneurismal than with other tumors.

Headache, due to interference with the return of blood to the heart, is not uncommon.

Disordered vision is due to pressure upon the sympathetic nerve, and consequent interference with the action of the iris. Ordinarily the pupil upon the affected side is strongly contracted, but in rare instances, from complete paralysis of its sympathetic nerve, it may be dilated.

Hæmoptysis, to a slight degree, is an occasional symptom due to congestion of the mucous membrane. Copious hæmoptysis frequently occurs at the close of the disease, when the aneurism ruptures into the air passages.

The essential *signs* are: a pulsating tumor in the region of the aorta, with systolic and diastolic shock and sometimes bruits.

Upon inspection, we often observe marked lividity of the face, neck, and upper extremities; with turgescence and a varicose condition of the veins, and perhaps œdema, due to obstruction in the return of blood to the heart from pressure of the aneurism upon one of the venæ innominatæ or the descending vena cava. Occasionally a thick fleshy collar is found about the base of the neck, due to capillary turgescence.

Œdema and turgescence are ordinarily limited to one side, and are caused by pressure on one of the venæ innominatæ. If the pressure is upon the descending vena cava, which is most likely to occur with an aneurism of the ascending arch, these signs will be found upon both sides.

The surface of the chest is seen to have a marbled appearance, caused by the prominence and blueness of the veins.

A tumor may usually be observed in the course of the aorta, the position of which will indicate the part of the blood-vessel affected.

When an aneurism originates in the sinuses of Valsalva it causes no external tumor. When in the ascending portion of the aorta, if bulging occurs, it will be seen in the second intercostal space at the right side of the sternum; but if large, it may extend far into the infraclavicular region, and even to the mammary.

Aneurism of the transverse portion of the arch causes a tumor at the upper part of the sternum.

When the descending arch is involved the tumor generally presents posteriorly at the left of the spinal column.

Exceptional.—In exceptional cases, an aneurism of the descending arch of the aorta may be seen in front, and in very rare instances it may be found at the right of the spinal column.

Aneurisms of the descending aorta present posteriorly below the fourth dorsal vertebra at the left of the spine. Very rarely they are seen at the right of the spinal column.

These tumors vary in size from a slight prominence to one as large as a child's head. The absence of a tumor does not necessarily prove that no aneurism exists; for, while the aneurism is small, it may not press upon the chest walls, and even when of considerable size the position may be such that no bulging is occasioned. The larger of these tumors are generally conical in form, and present very much the appearance of an immense boil, covered by thin glazed integument.

If pulsation of the tumor be observed, it will occur rhythmically with the apex beat of the heart. Pulsation, which cannot otherwise be seen, may sometimes be detected by bringing the eye to the level of the surface of the chest, as in standing behind the patient and looking down over his shoulders. No pulsation will be visible if the aneurismal sac is occupied by fibrin or coagulated blood.

If the tumor press on one of the main bronchi the respiratory movements on the corresponding side will be diminished or absent.

By palpation we may frequently detect a tumor, the impulse of which cannot be seen; we can ascertain the condition of the chest walls, whether there be perforation of the costal cartilages, sternum, or ribs, and may usually determine whether the contents of the tumor are fluid or solid. The character of the pulsation is expansile, that is, alike in every direction, and not simply lifting as is the case when a solid tumor rests upon an artery.

The most valuable sign obtained by this method is the detection of two pulsating points, as though there were two hearts, one beating in the normal position in the fifth interspace, and the other above the third rib.

If the aneurism is so small as to escape observation by ordinary palpation it may sometimes be detected by pressing firmly with one hand over the aorta in front, and with the other posteriorly.

The impulse obtained over an aneurism may be systolic, occurring with the contraction of the ventricles; or it may be both systolic and diastolic. The latter, produced by contraction of the artery, is usually slight, but occasionally quite forcible. When found, it is a valuable sign.

Frequently these tumors give rise to a peculiar thrill, similar to the purring tremor; sometimes very early in the course of an aneurism of the transverse arch, an impulse or a thrill may be felt by pressing the finger downward behind the suprasternal notch.

Valuable information may be obtained in some cases from the pulse, or from sphygmographic tracings (Fig. 42). If the aneurism press upon the arteria innominata, or upon either of the subclavian arteries, or if either of these vessels is obstructed by a coagulum, the radial pulse will be feebler upon the corresponding side. The carotids are sometimes similarly affected. If atheromatous degeneration of the arteries be general, the superficial arteries, especially the radial and temporal, will be found rigid and non-elastic.

Alterations in the movements of the chest walls and in the vocal fremitus are also to be sought by palpation. Pressure on the air passages will diminish the respiratory movements, and cause local or general diminution or absence of the vocal fremitus, according as a bronchus or the trachea is obstructed or the lung itself compressed.

Percussion must be performed gently, especially over large aneurisms, as a forcible stroke might possibly rupture the weakened blood-vessel.

Upon gentle percussion, the extent of dulness will not correspond to the size of the tumor, because of the overlapping borders of the lungs; but by a more forcible stroke, or by auscultatory percussion, we may determine the limits accurately.

The area of abnormal dulness is usually much smaller than in other tumors, causing symptoms of equal gravity.

The sense of resistance felt upon percussion is a valuable sign in distinguishing between aneurisms and other intrathoracic tumors. Over a tumor filled with fluid, the resistance is much less than over a solid growth or over an aneurism filled with fibrinous deposits.

If the aneurism present posteriorly, dulness will be obtained in the interscapular region. If it press upon a main bronchus, or upon one lung, causing collapse or congestion of this organ, dulness will be found over the corresponding side.

In auscultation, upon listening over an aneurism, we first notice an impulse or shock with each contraction of the heart. This is frequently followed immediately by a second or diastolic shock, due to contraction of the arteries. The impulse is usually attended by one or two sounds which consist mainly of the transmitted heart sounds, but are in part produced by dilatation and contraction of the artery.

These sounds may be associated with or supplanted by murmurs somewhat similar in character to endocardial murmurs. However, they are ordinarily less intense, though they may be even louder than the loudest heart murmurs. They are usually harsh in quality, and are not transmitted into the same regions as endocardial murmurs. Sometimes neither sounds nor murmurs can be detected over the aneurism.

If the tumor press upon a main bronchus, the respiratory murmur will be diminished or absent upon the corresponding side, while on the other it will be exaggerated. In these instances a forced inspiration will sometimes distend the lung, and bring out the respiratory murmur where it could not be heard during ordinary breathing. Vocal resonance will be diminished or absent over the obstructed lung, and absent over the aneurism. If the lung be condensed by pressure, broncho-vesicular respiration may be heard.

If the tumor press upon the recurrent laryngeal nerve, so as to cause paralysis or spasm of the vocal cords, there will be stridulous respiration, with dysphonia or aphonia, and inspection of the larynx will usually reveal the existence of paralysis of the cord on the corresponding side, with possible paresis of the other. Occasionally the pressure is upon both nerves, with consequent paralysis of both vocal cords.

Ferdinand Schnell (*Münchener medicinische Wochenschrift*, April, 1890) claims a new means for diagnosis of deep-seated thoracic aneurisms in the aneurismatoscope. This consists of a soft rubber tube closed at the lower end and filled with colored fluid, a piece of glass tubing being inserted into the upper end. When this apparatus is

partly inserted into the œsophagus, it is said that the pulsations of an aneurism of the descending arch are communicated to the tube and are indicated in the rise and fall of the fluid.

DIAGNOSIS.—Aneurism of the thoracic aorta may be confounded with solid tumors; with aortic pulsation, due to regurgitation through the semilunar valves; with pulsating empyema; with dilatation of the auricle; and with consolidation of the anterior border of the lung, with aneurism of the pulmonary artery, and with aneurism of the arteria innominata.

Venous turgescence, displacement of the heart, dulness on percussion, and modifications of the respiratory sounds, due to pressure, are signs common to these and to other varieties of intrathoracic tumors. Variation in the force and volume of the pulse on the two sides, expansile pulsation of the tumor, with a shock and bruit, are usually characteristic of aneurisms, but occasionally even these signs may be caused by solid growths. A diastolic bruit and shock over an intrathoracic tumor, accompanied by a clear second sound at the base of the heart, is diagnostic of aneurism, especially if following a distinct systolic bruit and shock. A murmur at the base of the heart, taking the place of the second sound, when associated with the signs of a tumor in the course of the aorta, is valuable evidence of probable atheromatous degeneration of the aorta, and the formation of an aneurism.

The differential features between aortic and pulmonary aneurisms and other diseases are pointed out below.

Aneurisms may be distinguished from other intrathoracic tumors by attention to the history and symptoms as well as to the physical signs.

The distinctive features between aneurism of the aorta and *solid tumors* are as follows:

ANEURISM OF THE AORTA.	SOLID TUMORS.
History.	
Seldom or never occurring before the twenty-fifth year of age, and usually not until after the forty-fifth year. Slight, if any, constitutional disturbance.	Usually malignant. They may occur in early life, and not infrequently before the twenty-fifth year. Grave constitutional disturbance.
Symptoms.	
Pain constant, and of a burning, wearing, or aching character and usually aggravated by exercise; frequently subject to neuralgic exacerbations. The symptoms and signs of pressure vary from time to time, owing to changes in the direction of the pressure.	Pain not so deep-seated as in aneurism; may be sharp and lancinating in character; not affected by exercise; not subject to neuralgic exacerbations. The symptoms and signs of pressure are constant, and steadily increase from day to day.

ANEURISM OF THE AORTA.	SOLID TUMORS.
Signs.	
Expansile pulsation. Often disparity between the radial pulses of the two sides. The area of dulness small in proportion to the size of the tumor and the length of its history. Sense of resistance slight.	No pulsation, or if any, simply a slight lifting impulse, caused by the tumor resting upon a large artery. Usually no disparity in the pulse of the two sides. Area of dulness large, and rapidly increases. Sense of resistance well marked.

Aortic aneurism is distinguished from *aortic pulsation* by the following symptoms and signs:

ANEURISM OF THE AORTA.	AORTIC PULSATION.
Symptoms.	
Symptoms of pressure upon the trachea, œsophagus, or recurrent laryngeal nerve.	No symptoms of pressure.
Signs.	
Pulsation in a limited space over the arch of the aorta.	Pulsation not only over the aorta, but in the carotids, subclavians, and brachials.
Radial pulse not exaggerated on either side by elevation of arm; usually feeble on one side.	Pulse sharp and apparently forcible; hammer pulse exaggerated by elevation of the arm, and alike on both sides.
Increased area of aortic dulness.	No increase in the area of dulness.
Arterial bruits, systolic or diastolic, generally distinct from endocardial murmurs.	Aortic regurgitant murmur, but no special bruit over the pulsating vessel.

Aneurism may be simulated by *pulsating empyema*, but ordinarily it can be easily distinguished by its position. If, however, perforation of the chest walls should take place in the course of the aorta, as in a case recorded by Flint, the diagnosis would be much more difficult.

ANEURISM OF THE AORTA.	PULSATING EMPYEMA.
Symptoms and Signs.	
Symptoms and signs of pressure upon adjacent organs.	Usually no symptoms of pressure upon the trachea, œsophagus, and other adjacent organs.
Dulness confined to the region of the aorta.	Dulness or flatness over the pulsating tumor, and also over the lower part of one side.
Arterial bruits. No pulmonary signs, unless there be pressure upon the trachea, bronchus, or lung itself. Expansile pulsation of the tumor.	No bruit. Signs due to compression of the lung by fluid in the pleural sac. Pulsation somewhat similar to that of aneurisms, but usually less expansile.

An aneurism of the aorta is distinguished from a *dilated auricle* as follows:

Aneurism of the aorta.	Dilated auricle.
Symptoms and Signs.	
Signs and symptoms due to pressure upon adjacent organs. Pulsation following the systole of the ventricles and the apex beat.	Few, if any, signs and symptoms of pressure. Pulsation preceding the apex beat.
Dulness in the region of the aorta. Arterial bruits common, but propagated mostly over the arteries.	Dulness extending far beyond the region of the aorta, and usually at a lower level; usually endocardial murmurs propagated in directions different from those of the aneurismal bruit.

Aneurism of the aorta is differentiated from *consolidation of the lung* by the position of the dulness and by the signs upon auscultation. If the consolidation is due to an aneurism, care must be taken not to overlook the signs of the latter.

Aneurism of the aorta.	Consolidation of the lung.
Signs.	
Dulness limited to the course of the aorta.	Dulness not limited to the aortic region, but extending externally, and usually involving the whole apex of the lung.
A normal respiratory murmur may often be heard over the greater portion of the aneurism. Arterial bruits.	Râles and other signs of consolidation. No bruits excepting possibly a systolic subclavian murmur.

Aneurism of the Pulmonary Artery.—Aneurism of the pulmonary artery is one of the rarest affections of the circulatory system. From the few cases which have been described, we are unable to obtain any characteristic symptoms or signs. The principal indications which have been noticed are: extreme cyanosis, with dropsy and great dyspnœa, associated with a strongly pulsating tumor, located in the second intercostal space of the left side, and limited to this region. This tumor is likely to yield a thrill upon palpation. Upon auscultation, systolic or diastolic murmurs, or both, may be detected, but they are not propagated above the clavicles. It is hardly possible to distinguish aneurism of the pulmonary artery from one of the aorta, which happens to present to the left of the sternum.

The position of a pulmonary aneurism is different from that of most aneurisms of the aorta. An aneurism of the ascending portion of the aorta might possibly present to the left of the sternum, though in this locality we are more likely to observe aneurism of the descending aorta. The distinctive features between aortic aneurisms and those of the pul-

monary artery may be stated, from the symptoms and signs which have been observed up to the present time, as follows:

ANEURISM OF THE AORTA.	ANEURISM OF THE PULMONARY ARTERY.
Aneurism of the ascending arch presents to the right of the sternum, and those of the descending arch usually present behind at the left of the third dorsal vertebra, and very rarely in front.	The tumor is confined to the second intercostal space of the left side.
Signs and symptoms due to pressure upon the trachea, bronchial tubes, œsophagus, blood-vessels, or recurrent laryngeal nerve, common.	The signs of pressure are comparatively slight, but usually there is congestion of the face, anasarca, and great dyspnœa.
Bruits, which may be propagated into the carotids and subclavians.	Bruits, not propagated above the clavicles.

Aneurism of the Arteria Innominata.—Aneurisms of the arteria innominata cause pulsating tumors similar to those of the aorta.

An aneurism of the arteria innominata may be distinguished from an aneurism of the arch of the aorta—first, by its position; second, by the comparative absence of signs due to pressure; and third, by the effect on the pulsation of compression of the subclavian and carotid arteries. Such an aneurism is located entirely upon the right side of the sternum, and causes a prominence in the region of the inner end of the clavicle. It is not likely to cause much pressure upon the recurrent laryngeal nerve with consequent obstruction of the larynx; or on the œsophagus, so as to interfere with deglutition; or upon the trachea so as to cause dyspnœa. Compression of the carotid or subclavian artery on the affected side greatly diminishes the pulsation in an aneurism of the innominate artery, but does not affect the pulsation of an aneurism involving the arch of the aorta alone.

PROGNOSIS.—The average duration of thoracic aneurism is two years and a half (Loomis, Practical Medicine). Recovery rarely occurs. In some cases the affection seems to remain stationary for many months. Death may occur suddenly at any time; the prognosis as to duration is therefore extremely uncertain. It depends somewhat upon the position of the aneurism, the structures pressed upon, and the occupation, temperament, habits and general health of the individual. Death usually occurs from rupture of the sac, but may be due to asphyxia, pneumonia, gangrene, or cerebral embolism.

TREATMENT.—A mixture composed of equal parts of tincture of belladonna and chloroform liniment has been recommended for relief of pain, but when this is acute opiates will generally be required for temporary relief. The persistent boring pain will usually be greatly or completely relieved after a day or two by potassium iodide given in doses of gr. x. to xx., three or four times a day. These methods of

treatment have been successfully employed in a few cases for the relief or the cure of aneurisms.

Tufnell's method, which in several cases has succeeded in at least greatly relieving the patient, is a modification of Valsalva's starvation plan. It consists of perfect rest in the recumbent position with moderate diet.

Ciniselli's method of galvano puncture first proposed in 1846 has been successfully employed in a few cases and may be tried if the foregoing methods fail. It is especially applicable in sacculated aneurisms near to the surface. Before making the puncture the patient may be given a full dose of morphine, or a small amount of cocaine may be injected at the points when the needles are to be inserted. From fifteen to thirty small cells should be used, and insulated needles connected with both poles should be thrust vertically into the aneurism an inch or two apart. Electrolysis should be continued fifteen or twenty minutes and may be repeated after a week if necessary. Great care should be used in withdrawing the needles to avoid loosening the clot.

During and after the operation, the patient should be kept quiet in the recumbent position.

Another method consists of the use of large doses of potassium iodide. This treatment usually soon relieves the severe neuralgic pains, and possesses the advantage of allowing the patient to move about, though it is more effective if the patient can be kept continuously in a recumbent position. The remedy should be given in doses of ten to thirty grains three times a day. The larger dose is much the best. Coryza may be relieved by moderate doses of nux vomica. If the stomach becomes irritable, the medicine should be suspended for a few days. Sometimes patients will bear large doses who cannot tolerate small ones.

When an aneurism causes dyspnœa through spasm or paralysis of the vocal cords, tracheotomy may be necessary; but this operation can do no good when the difficulty of breathing results from pressure on the trachea.

COARCTATION OF THE AORTA.

Synonym.—Stenosis of the aorta.

Coarctation of the aorta is one of the very rare affections of the circulatory system. The constriction may be ring-like, as though a cord had been tied about the artery; it may consist of a cicatricial band, partially obstructing the calibre of the blood-vessel; or it may be due to irregular contraction of the artery, the result of inflammation. The narrowing of the vessel may be slight, or the aorta may have dwindled to an impervious cord. In a few instances the constriction has been found to be general, involving both the arch and the descending aorta. In such cases usually no symptoms have been observed until about the age

of puberty, when deficient development of the lower extremities, and especially of the sexual organs, has been the first indication of the condition.

Inspection reveals *signs* of hypertrophy and more or less dilatation of the heart; usually, dilatation of the arch of the aorta, of the subclavian arteries, and of the carotids; a dilated and tortuous condition of the superficial arteries, which in the normal state are not visible. This condition of the superficial arteries is attended by marked pulsation, and sometimes by small aneurismal enlargements of the intercostal arteries which may be sufficient to cause erosion of the ribs.

A thrill can generally be detected by palpation over the large arteries. The obstruction of the vessel renders the pulsation feeble in the branches of the abdominal aorta, and causes feebleness or absence of the pulse in the tibial and popliteal arteries. Percussion gives no signs. On auscultation, a harsh, high-pitched, and usually intense systolic or postsystolic murmur will be heard over the aorta and larger blood-vessels. This is usually most intense close to the edge of the sternum in the second intercostal space upon the right side. This murmur is propagated through the carotids and subclavians toward the shoulder, and may also be heard posteriorly over the course of the aorta.

The occurrence of such a murmur will lead us to suspect the existence of an aneurism; but the latter may be excluded by absence of the symptoms and signs due to pressure, and by the want of an increased area of dulness on percussion.

DIAGNOSIS.—The diagnosis of coarctation of the aorta rests mainly upon the enlarged and tortuous condition of the superficial arteries in the upper portion of the body, and the feeble pulsation in the lower extremities, associated with an aortic systolic murmur.

TREATMENT.—No treatment can be recommended.

SOLID MEDIASTINAL TUMORS.

Excluding aneurisms, tumors within the chest are nearly always malignant in character, and are therefore attended with grave constitutional symptoms; some are of syphilitic and others of tubercular origin.

SYMPTOMATOLOGY.—A growth usually causes pain of a persistent character, sometimes lancinating, but not subject to the neuralgic paroxysms which attend an aneurism.

The principal *signs* are: turgescence of the veins, œdema, dyspnœa, dysphagia, and other evidences of pressure on surrounding organs, with dulness and loss of respiratory murmurs over the growth.

By inspection we commonly find persistent turgescence of the veins, and œdema of the neck and upper extremities in a more marked degree than from an aneurism. A tumor is nearly always accompanied by enlargement of the lymphatic glands in the neck and axillary re-

gions. The condition of these glands is an important point in the differential diagnosis; for, if it is due to malignant disease, they will be adherent to the surrounding tissues, but, if the conditions are not of malignant origin, they may usually be moved freely beneath the integument. The symptoms and signs caused by pressure on the surrounding organs are persistent, and they gradually increase in severity. A malignant tumor is not usually confined to the course of the aorta, but is apt to extend a considerable distance beyond the borders of the sternum. A solid tumor does not ordinarily pulsate, and, when it does, the pulsation is not expansile, but is simply lifting. This impulse is caused by the pulsation of a large artery upon which the tumor rests.

On percussion, the sense of resistance is marked, and the area of dulness is usually much larger than over an aneurism, because the malignant disease gradually involves the adjacent lungs, instead of crowding them before it.

By auscultation, no bruit can be heard over a tumor, unless it presses upon an artery, and then the murmur is distant and comparatively feeble.

Exceptional.—In those unique cases where a tumor coexists with a quiescent aneurism, some peculiar phenomena have been observed. The sense of resistance to the percussion stroke over an aneurism may be great; whereas over a solid tumor there may be only slight resistance, and in the same position we may detect an expansile pulsation, which should naturally be found over an aneurism.

Diagnosis.—The essential features which enable us to distinguish between a solid tumor within the chest and an aneurism were referred to in the consideration of aneurisms.

Prognosis.—Sarcomata and carcinomata of the mediastinum are commonly fatal within a twelvemonth. Syphilistic growths will often subside under proper remedies. Enlargement of the bronchial glands is not infrequently followed by suppuration, and often eventually terminates fatally.

Treatment.—No special treatment can be recommended excepting that indicated by the constitutional dyscrasia.

Diseases of the Throat.

CHAPTER XVI.

THE THROAT.

EXAMINATION OF THE FAUCES.

A CONSIDERATION of the diseases of the chest is very properly associated with a study of the upper air passages, since diseases of the nose, fauces, pharynx, or larynx often cause symptoms which simulate those of pulmonary affections. In some instances so slight a difficulty as elongation of the uvula will cause the symptoms of laryngitis, or even the persistent cough, emaciation, and other symptoms of the later stages of phthisis.

For the examination of the fauces it is generally necessary to depress the tongue. For this purpose a great variety of tongue depressors have

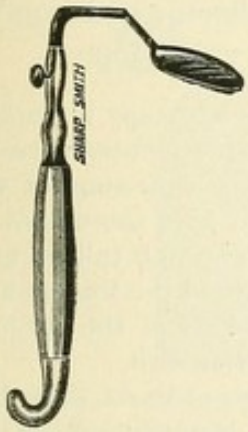

FIG. 50.—TÜRCK'S TONGUE DEPRESSOR (¼ size).

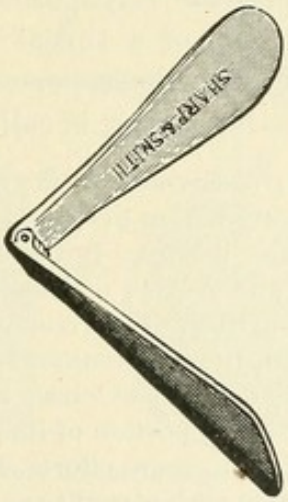

FIG. 51.—POCKET TONGUE DEPRESSOR (2-5 size).

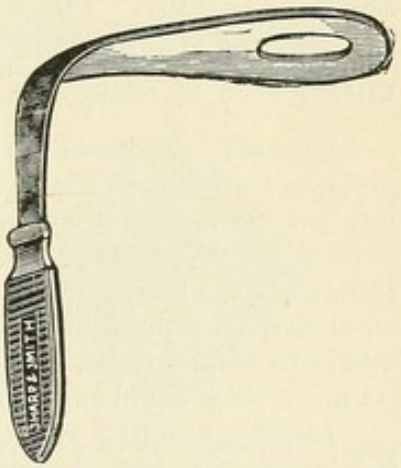

FIG. 52.—BOSWORTH'S TONGUE DEPRESSOR (2-5 size).

been devised which will be found useful, but, if not at hand, a spoon-handle, lead-pencil or the forefinger will answer the purpose.

For ordinary use, a spoon-handle is perhaps the best, as many patients object to an instrument which is used promiscuously. Of the different varieties of tongue depressors, for carrying in the pocket those which are jointed are most convenient (Fig. 51). In office practice, some of the larger, stronger varieties are preferable (Figs. 50 and 52). Some patients can so control the base of the tongue as to allow a view of the throat without the aid of a depressor, but this is not the rule. A fair view may often be obtained in children while they are crying or coughing. If the child resists, a spoon-handle or other depressor may be

passed well back upon the base of the tongue, so as to induce retching, which will afford a good view of the pharynx.

We should embrace every opportunity for inspecting the healthy throat, in order to become familiar with its normal conditions, otherwise we are unable to recognize quickly the signs of disease. Upon inspection of the healthy fauces, we first notice the soft palate with the pendent uvula, which forms the back part of the roof of the mouth. Running downward from either side of the soft palate will be seen two folds of mucous membrane, known as the anterior and posterior pillars of the fauces, between which may be seen a glandular mass, termed the tonsil. Posteriorly we observe the posterior pharyngeal wall, which closely covers the bodies of the cervical vertebræ. Superiorly, our field of vision is obstructed by the palate; inferiorly, by the base of the tongue.

LARYNGOSCOPY.

In order to look beyond the lines of direct vision, we must use mirrors. Inspection of the larynx with these is called laryngoscopy, and the same method applied to the nasal passages and nasopharynx is called rhinoscopy. The essentials for laryngoscopy are, a throat mirror and a good light. The combination of a throat mirror and a reflector for directing the light is called a laryngoscope. A reflector and smaller mirror used in examining the nasopharynx is called a rhinoscope.

HISTORY.—The credit of having discovered the art of laryngoscopy is usually given to Czermak, of Pesth, but many before his time had experimented more or less successfully in illuminating the larynx. Bozzini in the beginning of the present century, Bennatti in 1832, and Avery, of London, in 1844 attempted to illuminate the larynx by means of artificial light conducted through tubes; but, as shown by Trousseau and Bellocq, these instruments crowded the tongue and epiglottis before them, so as nearly or quite to close the orifice of the larynx. At most, they could expose only a small portion of its posterior wall.

About a hundred years previous to these efforts, Levret, of Paris, probably the first experimenter in this direction, attempted to see the larynx by means of a small throat mirror, similar to that now in use. Senn, of Geneva, in 1827; Babbington, of London, in 1829; Baumes, of Lyons, in 1838; and Liston, of London, in 1840, employed similar instruments with equally unsatisfactory results. Warden, in 1844, made experiments with a couple of prisms. All of these investigators failed more or less completely, for the reason that they could not secure suitable illumination.

The first to demonstrate the larynx in the living subject was Signor Manuel Garcia, a teacher of vocal music in London. He became quite expert in autolaryngoscopy, and also succeeded in demonstrating the larynx in others.

Garcia's observations were communicated to the Royal Society of London in 1855. They attracted little attention at first, for the art was thought to be of no practical value in the diagnosis of disease, because a thorough inspection was supposed to depend upon a peculiar education of the muscles which would enable the patient to control the position and movements of his throat. However, Garcia's writings induced Türck, of Vienna, to experiment with similar mirrors

in the hospital during the summer of 1857. Although Türck was fairly successful in these experiments, he finally threw aside his mirrors as the autumn came on, because of the difficulty in obtaining sunlight. His experiments were not lost, for Czermak, of Pesth, who had been visiting in Vienna during the summer, borrowed the mirrors and continued the investigations. He overcame the difficulties which had previously prevented a clear view of the larynx, by employing the reflector and causing the patient to protrude the tongue, instead of depressing it, and by substituting artificial light for the direct rays of the sun. Soon a rivalry sprang up between Czermak and Türck as to the priority of their claims. Their letters, which were published in the various medical journals, spread a knowledge of the new art throughout the medical world.

THROAT MIRRORS have been made in various forms. Some are round, others oval or lozenge-shaped, and still others quadrilateral. For gen-

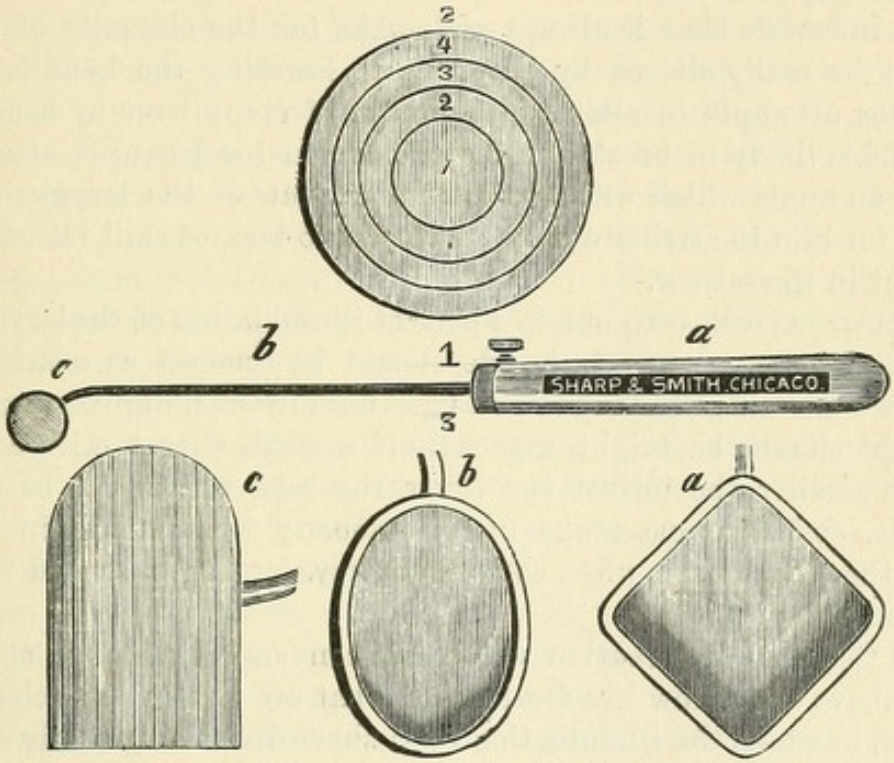

FIG. 53.—THROAT MIRRORS FOR LARYNGOSCOPY. 1. *a*, Handle; *b*, stem; *c*, mirror. 2. Different sizes of round mirrors. 3. *a*, *b*, *c*, Different forms of throat mirrors.

eral use the round mirrors, varying in diameter from three-eighths of an inch to an inch and a quarter are preferable. Mirrors should be made of clear and perfectly white glass. The quality of the glass may be tested by placing a white card before the mirror. If the glass is perfectly white, the reflection will also be white; if the glass is tinged with color, it will give a corresponding shade to the reflected image of the card, and would necessarily similarly affect the laryngeal image.

The glass and its setting should be thin, in order to economize space in the throat.

The glass should be set firmly in a metallic frame, which must encroach as little as possible upon the anterior surface of the glass, so that the largest possible reflecting surface may be secured. Some of these mirrors are backed with amalgam, and others with silver-leaf. Silver-

leaf renders a mirror more durable, as it is less affected by heat and moisture. I have used mirrors backed with amalgam many times daily for several months without injuring them, though one may be ruined in a week if heated too much or left in the water. The mirror should be firmly attached to a wire stem about four inches in length, at an angle of not less than one hundred and twenty degrees. This stem may be fixed in a small handle about three inches long, or the handle may be removable, the stem when inserted being held by a set-screw. Some laryngologists recommend a flexible stem, so that the angle of the mirror can be easily altered; but it is likely to become bent by contraction of the palatine muscles, when the mirror is in position, in such a manner that the larynx cannot be seen.

An inflexible stem is always preferable, for the obliquity of the mirror can be easily altered by elevating or lowering the handle. If the beginner attempts to alter the obliquity of the mirror by bending the stem, he is likely to break the instrument in his frequent attempts to secure an angle which will give a different view of the larynx; and it is better for him to attribute want of success to lack of skill rather than to a defect in the mirror.

Illumination.—To obtain a perfect illumination of the larynx, three things are necessary: first, the eye should be brought as nearly as possible into the centre of the beam of light used in the illumination; second, the light should be bright, especially if a small throat mirror is used, for the smaller the mirror the fewer the rays which can be reflected from it, and we must make up in intensity what is lost in volume; third, the focal point, when convergent rays are used, should fall upon the part to be inspected.

All forms of illumination which cast convergent rays into the larynx cause above and below the focal point what are known as circles of dispersion, in which the illumination for a short distance is nearly as bright as at the focal point. In examining the larynx, an effort should be made to concentrate the rays of light on the vocal cords; the circles of dispersion will then give a good illumination for half an inch above or below the plane of the glottis. In men, the glottis is about three inches below the mirror when it is held in the posterior part of the mouth, and in this position the mirror is about three inches from the lips; therefore in men the glottis is about six inches within the lips, but in women about five inches. As the eye cannot be brought nearer to the mouth than five inches, without interfering with the manipulation of the instrument, the radiant or focal point must fall eleven inches from the reflector, which is worn on the forehead.

Being myself hypermetropic, I find it most convenient to have the eye at least eight inches from the patient's mouth; and therefore must use a reflector which will concentrate the rays of light at a point fourteen inches from itself.

Persons with presbyopic eyes may obtain a good view in the same manner; deficient accommodation in the eye may be corrected by glasses.

Myopic eyes of less than one-tenth will necessitate the use of concave glasses; but for eyes, myopic from one-tenth to one-seventeenth, glasses will not be needed, excepting to view the bifurcation of the trachea.

To examine the bifurcation of the trachea, which is five or six inches below the plane of the vocal cords, we must remember that the focal point should be at least sixteen or seventeen inches distant from the reflector.

The larynx may be illuminated by a simple flame, or a plane or concave reflector with or without condensing lenses may be employed to reflect the rays of light into the throat. In illuminating the larynx by the direct rays of the sun, lenses are not used, and reflectors are not absolutely necessary. When diffused daylight is employed, reflectors are required to concentrate the rays. Though direct sunlight, or sometimes diffused daylight, gives a beautiful illumination, artificial light will be found indispensable for general use. Natural light cannot usually be secured in the proper position at the time we wish to use it.

Illumination with Direct Artificial Light.—When using a simple flame without a reflector, the lamp must be placed directly in front of the patient's mouth, and shaded toward the eye of the observer. This will give a good illumination if the light is very bright, but with the ordinary lamp or gas-jet it is not satisfactory. This method may be improved by using a condensing lens with a focal distance of six or seven inches. The lens should be held between the light and the patient's mouth, and about five inches from the latter. The flame should be placed at a point which will cause its rays to be brought to a focus eleven inches beyond the lens at the plane of the glottis. The observer's eye must then be brought near the edge of the lens.

Illumination with Reflected Artificial Light.—The above-named apparatus may be supplemented by a plane perforated reflector, which, placed in front of the observer's eye, reflects into the mouth the rays from the condensing lens; or this reflector may be used with the simple flame without a condenser.

In order to fulfil the three essential conditions—that is, to have the eye in the centre of the cone of light, to obtain a bright illumination, and to have the focal point fall upon the part to be examined—laryngologists generally resort to a *perforated concave reflector*. Such a mirror, by collecting many rays otherwise lost, and concentrating them on the point to be examined, intensifies the illumination, and the perforation in its centre brings the observer's eye into line with the centre of the cone of light. Many laryngologists prefer to place the reflector above the eye, but unless a very bright light is employed this position will not give a good illumination of the larynx, and if a brilliant light is used it is very trying to the eyes.

The reflectors vary in size, in focal distance, and in the material of which they are constructed. Those used in laryngoscopy are usually from three to four inches in diameter, with a focal distance ranging from five or six to fourteen or sixteen inches. They are made of either glass or metal; the former are best, as they do not become dim by tarnishing. For ordinary use, a reflector with a focal distance of seven or eight inches will give better satisfaction than one with a longer focus, except when parallel rays of light, as those of the sun or of diffused daylight are to be reflected. The rays coming from any artificial light are necessarily divergent, and consequently cannot be brought to a focus in the larynx by a reflector with a focal distance of eleven inches, which would concentrate only parallel rays at the proper point.

With the ordinary position of the flame, and of the observer's eye, a reflector of seven inches focal distance will throw the radiant point upon the glottis. The radiant point may readily be moved toward and from the eye by increasing or lessening the distance of the flame from the reflector, so that reflectors of varying focal distances may be employed, providing the light is sufficiently intense.

On account of its simplicity, the formula $\frac{1}{F} = \frac{1}{A} + \frac{1}{A'}$ has been generally adopted in determining the focal distance of the reflector, or the proper position of a flame, which, with a reflector of known focal distance, will cause the image of the flame to fall upon the glottis. The image of the flame and the radiant point are in this connection used as synonymous terms. The focal point is the same as the radiant point when parallel rays of light are employed.

In this formula, F represents the focal distance of the reflector; A, the distance of the reflector from the flame; A′ the distance of the reflected image of the flame (focal or radiant point) from the reflector. Knowing the focal distance of the reflector, seven inches, and the proper distance of the image of the flame, which, as already explained, should fall upon the glottis, and will therefore be eleven inches from the reflector—five inches from the observer's eye to the patient's mouth, and six inches from the patient's lips to his vocal cords—we can readily ascertain the proper position of the flame by substituting the known quantities in the formula thus: $\frac{1}{7} = \frac{1}{A} + \frac{1}{11}$. This, reduced, will give a fraction over nineteen inches as the value of A, which will represent the proper distance of the flame from the reflector.

To find the focal distance of the reflector by artificial light, we proceed in a similar manner with the same formula. Placing the light at a fixed point and the reflector in front of it, we find the distances from the flame to the reflector, and from the reflector to the image of the flame, by direct measurement with an ordinary tape. These two known quantities being then inserted in the formula in the place of A and A′, the value of F can readily be obtained. The focal distance of a reflector

may be easily ascertained with solar light by placing it in the sunlight, throwing the radiant point on some object, and measuring its distance from the centre of the reflector. The focal distance may be measured with diffused light by reflecting the image of some distant object, as a window, on some plane surface, and measuring the distance from this image to the reflector.

In using reflectors, it is essential that the light be so managed that the radiant point will fall on the part to be illuminated.

Students of laryngoscopy usually have great difficulty in obtaining a uniform illumination. Sometimes the parts will be brilliantly illuminated; at other times with the same light and the same laryngoscope the larynx is only seen in a deep shadow. This is generally due to the improper position of the light. We must not forget that the larynx is necessarily from eleven to fourteen inches from the eye, and that, with a reflector of seven or eight inches focal distance, if the flame be placed too near the eye, the radiant point will fall a considerable distance beyond the glottis; or if too far from the eye, the radiant point will not reach the glottis. We should always know the focal distance of our reflector, and ascertain by the formula just explained the proper distance at which to place the flame, remembering that the distance of the radiant point from the reflector will vary inversely as the latter is carried toward or from the flame.

Practically, if we have a proper reflector of seven to eight inches focal distance, it will not be necessary to measure accurately the distance of the flame. Placing the light beside the patient, we may sit in front with the reflector, ten or eleven inches from the patient's mouth; carry the light forward or backward until its perfect inverted image falls on the patient's lips, this will be the proper position for the light. By bringing the reflector about four inches nearer the mouth, the radiant point falls upon the glottis.

Various contrivances are employed for holding the reflector. Czermak at first had it fastened to a mouthpiece of orris root, which he held between his teeth. Semeleder and others are in favor of a spectacle frame, to which the reflector is so fastened that it may rotate in any direction. If the physician happen to be myopic or hypermetropic, lenses may be fitted in this frame to correct the error in accommodation. Jointed arms for holding the reflector accompany many forms of illuminating apparatus. These are inconvenient for, if the patient moves after the arm has been adjusted, each movement may require a change in the position of the reflector. Kramer's head band, or some modification of it, is the most common, and, I think, the best device for holding the reflector. It consists of a head band with a metallic or vulcanite plate in front to which the reflector is attached by a ball-and-socket joint, which enables one to fix it in any position. Most of the head bands are open to two objections; first, they cannot be made tight enough to hold the reflector

firmly without causing headache; and second, the ball-and-socket joint is so constructed that, after it becomes a little worn, it is impossible to fix the reflector firmly. Schrötter's head band made of firm non-elastic webbing, with nasal rest, obviates these difficulties.

FIG. 54.—SCHROTTER'S HEAD BAND WITH NASAL REST.

Whatever the means employed for holding the reflector, it must be borne in mind that the flame must have a certain definite relation to the reflector, depending on the focal distance of the latter and its distance from the glottis, so that the image of the flame will fall upon the vocal cords.

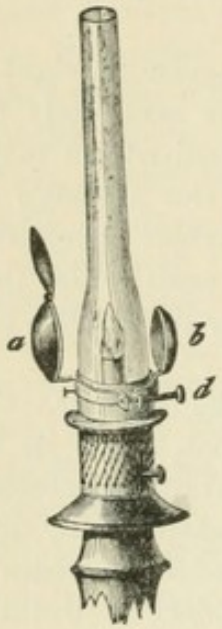

FIG. 55.—KRISHABER'S ILLUMINATOR. *a*, Lens; *b*, reflector.

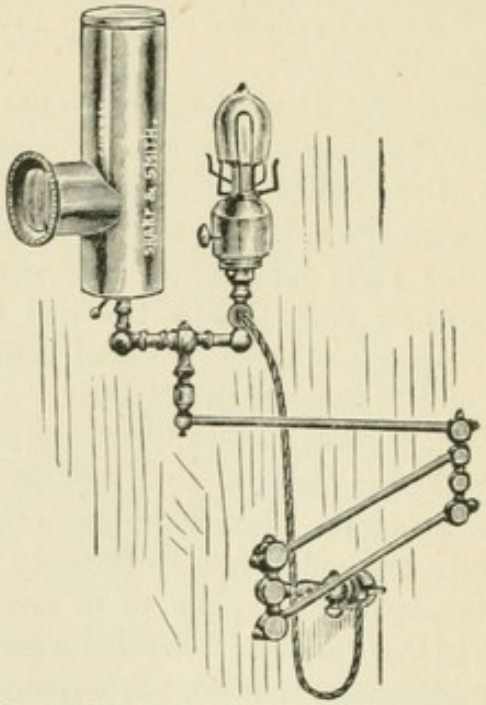

FIG. 56.—MODIFIED MACKENZIE'S RACK-MOVEMENT BULL'S-EYE CONDENSER. For gas or incandescent electric light.

In place of throwing the radiant point on the glottis, some physicians prefer to illuminate the parts to be examined with the bright disc of light which may be obtained in the circle of dispersion above or below the radiant point.

Several instruments have been devised for the purpose of rendering the light in this disc more intense.

One of the simplest of these is Krishaber's illuminator (Fig. 55). It consists of a reflector and a convex lens, which may be fastened by the clamp to an ordinary lamp.

This apparatus will often give very satisfactory results.

Mackenzie's bull's-eye condenser is used for the same purpose. It consists of a rack-movement gas fixture with a metallic chimney, which can be adjusted to the ordinary gas-burner (Fig. 56). The chimney has an orifice on one side for the condensing lens, and the latter is placed at a fixed point in front of the flame, so that the rays of light on leaving

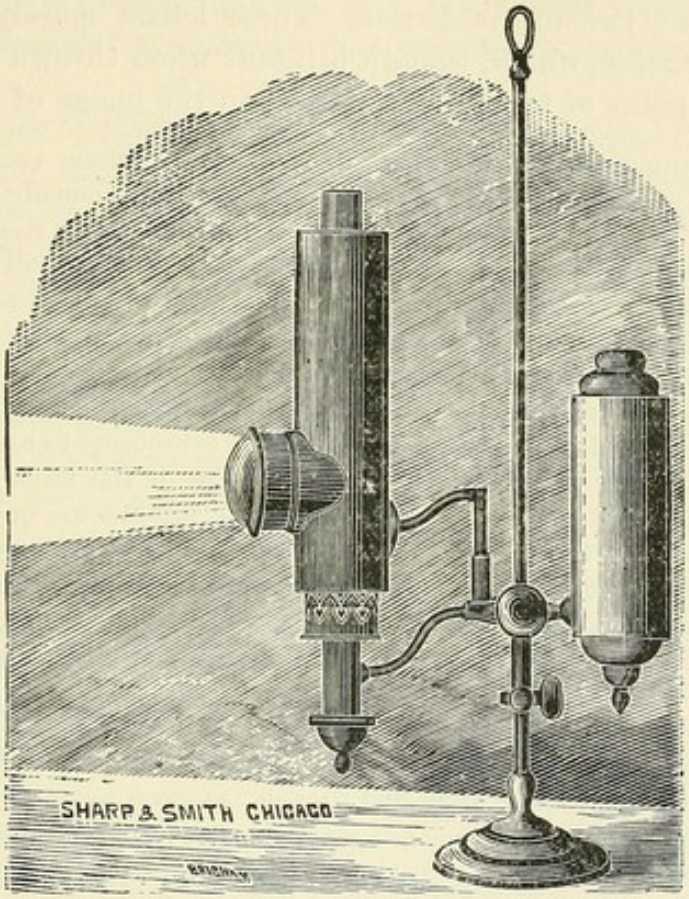

FIG. 57.—MODIFICATION OF MACKENZIE'S ILLUMINATOR, WHICH MAY BE USED EITHER WITH A STUDENT'S LAMP OR AN ARGAND GAS-BURNER.

it will be nearly parallel. This illuminator may be brought directly in front of the patient's mouth for direct illumination, but it is generally used with a reflector of from eleven to fourteen inches focal distance.

Fraenkel's illuminator is somewhat similar in construction as regards the condensing lens, but is so arranged that the rays of light on leaving the lens may be made either divergent, parallel, or convergent, according to the size and focal distance of the reflector which is employed.

In accordance with my suggestions a similar condenser has been constructed, which may be used with the ordinary Argand gas-burner or German student's lamp (Fig. 57). In this condenser the lens, which has a focal distance of three and one-half inches, is set about two inches from the flame, so that the rays of light are divergent on leaving it, and are thus adapted

for a reflector with a focal distance of seven or eight inches. If it is desired to obtain a bright circle of dispersion for illumination, or to use a reflector with a longer focal distance, the cap in which the lens is set can be drawn out so that the rays will be less divergent.

This condenser is comparatively inexpensive, and possesses all the advantages of the last two described, as well as those of Tobold's illuminator, without the imperfections of the latter. With this condenser and Fraenkel's, either the radiant point or the circle of dispersion may be used for illuminating the glottis.

Tobold's illuminator, a combination of lenses devised by Tobold, is in common use. Weil has shown that the apparatus is improved by removing one or two of its lenses. These lenses merely cause a large circle of dispersion, which, though brilliant when thrown on an external object, is, in point of fact, less intense than the image of the flame.

Tobold's apparatus has a combination of three lenses, two of which, each having a focal distance of about three inches, are placed closely together, and so near the flame that they collect divergent rays as they leave the lamp, and concentrate them to a focus about six inches in front of the second lens. The third lens, farthest from the flame, has a focal distance of about five inches. It is placed four inches in front of the second lens, about two inches within the point at which the rays of light are concentrated by the latter, so that the rays of light falling on it are convergent. The convergent rays, by passing through the third lens, are rendered still more convergent, and are brought to a focus about three inches in front of the apparatus, where the image of the flame is perfect. The reflector is fixed about four inches in front of the apparatus, or one inch beyond the radiant point of the last lens. Here the rays, having crossed, are so widely divergent, that a reflector of one and a half inches focal distance would be required to concentrate them upon the glottis. The reflector used has a focal distance varying, in different instruments examined, from five to nine inches. Therefore the rays must also leave the reflector widely divergent, so that most of them will be lost. Hence, we see that the large bundle of rays collected by the first lens, which might then have been entirely utilized, is first subjected to the loss incident to refraction, and then largely thrown away. We must admit that a sufficient number of rays are still retained to give a good illumination, though less intense than when only one lens is employed.

No advantage can be derived from such a combination, except where cheap lenses of a moderate convexity are placed together to secure a short focal distance. A single lens of sufficiently high power to accomplish the same result would be comparatively expensive. Tobold has also devised a smaller instrument known as the pocket illuminator, the construction of which is similar to that of the one just described.

The image of the flame may be so magnified by a single lens, as found in the condensers already mentioned, that it is as large as can possibly be reflected from any throat mirror.

In using condensing lenses, any one of three methods may be adopted: the flame may be placed at the focal point of the lens; it may be placed beyond the focal point; it may be placed nearer to the lens than its focal point.

With the flame at the focal point, the rays which always leave the

light in a divergent direction are refracted, so as to leave the lens in a parallel direction, and they must then be managed in the same manner as the parallel rays of sunlight or diffused daylight. In this instance, a reflector of a diameter the same as that of the lens should be employed, having a focal distance of from eleven to fourteen inches. This will bring the image of the flame upon the glottis, providing the eye is from five to eight inches from the mouth.

When the flame is placed beyond the focal distance of the lens, its divergent rays, after passing through the lens, become convergent. Here the reflector may be smaller than the lens, but it must have a focal distance of more than eleven inches; otherwise the rays will be brought to a focus too soon.

When the flame is placed nearer the lens than its focal distance, the rays, after passing through, are still divergent, and, in order that none be lost, they must be received on a reflector larger than the lens, which must have a focal distance of not more than eight inches, the same focal distance as that required when a flame is used without a condensing lens. This is by far the best method for practical purposes, as it gives an illumination equally as good as the other methods, and does not necessitate the possession of a number of reflectors.

Some form of condenser is desirable for office use, but I have always found a simple concave reflector of large size and short focal distance sufficient for purposes of diagnosis, and ordinarily for operations within the larynx. Such a reflector may be used with an ordinary gas-jet or with any lamp, and may be sufficient, even if one is obliged to rely on candles. For general use it will certainly be found more satisfactory than a cumbersome illuminating apparatus.

When performing operations in the larynx, it is desirable to have as large a field illuminated as possible. This may be attained by means of the bull's-eye condenser with the ordinary flame, or with a brighter light and a reflector with a long focal distance, so that the circle of dispersion can be utilized in place of the radiant point.

Several laryngoscopes, illuminated by electric light, have been invented, but they are not usually so satisfactory as the simple reflector and Argand burner or German student's lamp.

A bright electric light, if properly arranged, would perhaps be the best for laryngoscopy, and, next to it, the oxyhydrogen light. The former, however, cannot always be obtained, and the latter, besides being difficult to manage, requires a great deal of apparatus, and is consequently expensive. A good Argand gas-burner or a German student's lamp with a bull's-eye condenser is all that is necessary for illumination, even during operations. I have sometimes obtained brilliant illumination even with a common kerosene lamp, having a circular wick like that shown in Fig. 55. For purposes of diagnosis, any ordinary lamp, freshly trimmed, and with a clean chimney, will generally be

sufficient. As suggested by J. Solis Cohen, two or three candles tied together, and placed in front of the bowl of a spoon used as a reflector, may be made to answer the purpose if a lamp cannot be obtained.

Diffused daylight, when properly managed, gives a beautiful illumination of the larynx. Artificial light more or less discolors the image, causing the normal larynx to appear yellowish or red, whereas diffused daylight shows the parts in their natural colors. Unfortunately the latter is seldom sufficiently bright. On a bright day, if light can be admitted through a small opening into a darkened room, so as to fall upon the reflector, it will give a good illumination. If it is impossible to admit the light through a small aperture, a good view may sometimes be obtained by placing the patient at the farther side of the room, opposite a single window left uncovered, with his back to the light. This position will give a much better view than when the patient is placed near the window.

Direct sunlight may be employed, with the patient facing the window, in such a position that the rays fall upon the throat mirror held in the pharynx. A serious hindrance to this method is that the light cannot often be obtained in a suitable position. Reflected sunlight may more frequently be employed with the aid of a plane reflector, or of one with a long focal distance, but it is only in comparatively rare instances that we have a proper exposure and find the sun at the desired altitude.

Heliostats have been constructed for reflecting the sunlight in a given direction. They may be arranged by a system of clockwork to maintain the beam of light at a given point throughout the day. This apparatus is very expensive, and not to be recommended.

An ordinary toilet mirror may be so placed as to receive a beam of sunlight, and direct it horizontally in any desired direction; but this is not often satisfactory for consecutive work. For the reasons named, we are usually compelled to use artificial light.

Laryngoscopy should be practised with both natural and artificial light, to give familiarity with the appearance of the parts under both forms of illumination. The same larynx will have different shades when viewed by different lights; what appears congested when viewed by artificial light, may seem of normal color by daylight.

For the purpose of magnifying the image of the larynx, Wertheim recommended concave throat mirrors, and Türck suggested a small telescope, some improvements in which were made by Voltolini; but these have all been found practically useless.

The laryngoscope which I prefer consists of a perforated reflector four inches in diameter (Fig. 58), with a focal distance of eight inches, attached to Schrötter's head band, with nasal rest, by means of a ball-and-socket joint; with three round throat mirrors, three-eighths, seven-eighths, and nine-eighths of an inch in diameter respectively, the small-

est for children, and one oval mirror three-fourths of an inch in diameter, for use in cases of enlarged tonsils. As before stated, these throat mirrors should be backed with silver-leaf and firmly fastened to an inflexible stem, which may be permanently fastened to the handle or not, as is most convenient. The reflector need not be more than three and one-half inches in diameter, but the larger instrument will reflect a greater number of rays, and thus give a somewhat brighter illumination. The four-inch reflector possesses the additional advantage, when worn before one eye, of shading the other from the light. The only objection I have found to it is that the attachment for the ball-and-socket joint is in some instruments placed too far from the perforation, causing diffi-

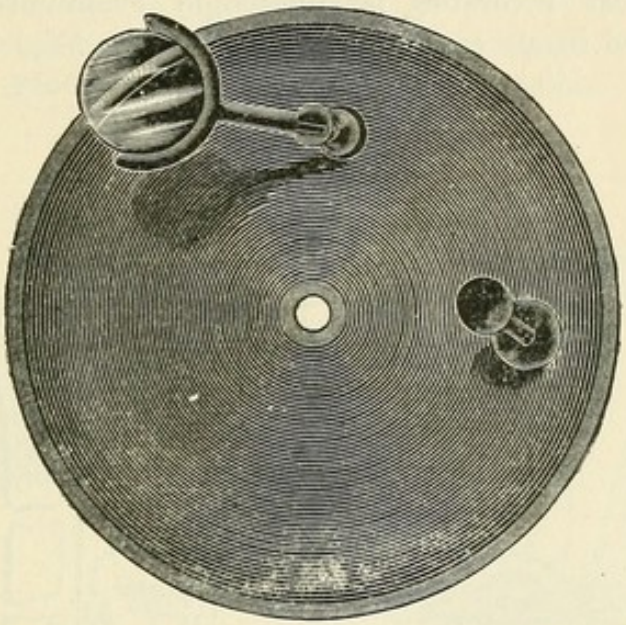

FIG. 58.—LARYNGOSCOPIC REFLECTOR, with attachment for holding lens to correct defective accommodation. The ball for ball-and-socket joint should be placed accurately 1⅝ inches from centre of reflector.

culty in bringing the perforation squarely before the eye. This objection should always be remedied by the manufacturer.

For an illuminating apparatus, we may use an Argand gas-burner attached to a rack-movement fixture, similar to the one shown (Fig. 56), or a German student's lamp, which may be supplemented by a condenser (Fig. 57).

Manipulation of the Laryngoscope.—After familiarizing ourselves with the laryngoscope and the rules for its use, before attempting laryngoscopy on a living subject, it is well to practise for some time on a dummy, or on a larynx which has been removed from the body and attached to a standard. If one of these cannot be obtained, we may easily make a model by boring a couple of holes in a block of wood—one about two inches in diameter to represent the mouth, and the other about an inch in diameter, intersecting the first at an angle of eighty degrees, to represent the larynx. By practising on it we may

familiarize ourselves with the management of the light, reflector, and throat mirror, and may educate our hands to steadiness.

Having learned to control the hands so that the mirror will not tremble, and to reflect the rays of light accurately to the objective point, we may begin to practise upon the living subject. A novice at first will find it of great advantage to practise upon a patient who has been trained and can undergo the manipulations of an unskilled hand without retching; subsequently he should practise upon healthy individuals for some time, in order to become so familiar with the normal appearance of the larynx that any deviations from it will be at once recognized.

For the most favorable laryngoscopic examination the patient should be seated in an erect position with the head thrown slightly

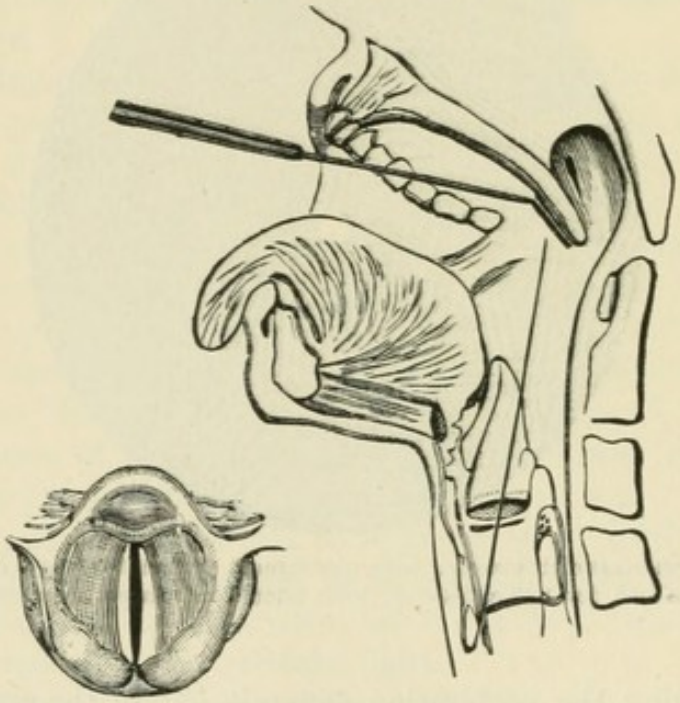

FIG. 59.—POSITION OF HEAD GIVING THE BEST VIEW OF LARYNX, AS SHOWN IN SMALL CUT AT THE LEFT (ALTERED FROM BROWNE).

back. The physician should be seated in front on the same or on a slightly higher level, and as close as possible, with one knee on either side of the patient's knees, which are brought together.

It is often necessary to make the examination with the patient slightly propped up in bed, and the physician sitting as best he may beside him; or with the patient standing, as when a library drop-light is used, which cannot be brought low enough to illuminate the throat when the patient is sitting.

The most suitable seat for the patient is a narrow chair, with a straight back, sufficiently high to support the head, and a seat not more than a foot in depth, which will compel the patient to sit erect. For the physician a small stool, which can be raised or lowered to any desired level, is most convenient.

The patient should be seated beside or just in front of the table which holds the instruments, with a cuspidor beside him, and a glass of water close at hand. If direct sunlight is employed, the patient should be placed near the window, facing the light, which, coming in over the physician's shoulders, falls directly upon the pharyngeal mirror. With reflected sunlight, the positions of patient and examiner as regards the window are reversed. When artificial light is employed, the examining-room should be shaded. The light should be placed on a level with the eyes of the patient, and slightly behind him, so that it will not shine on his face, and about six inches distant at one side, so that the rays may fall without obstruction on the reflector. If the flame is much above or below the level of the eyes of the patient, or far from his head, at one side, the angle at which the rays fall upon the reflector will be so great that a good illumination will be impossible. The patient's

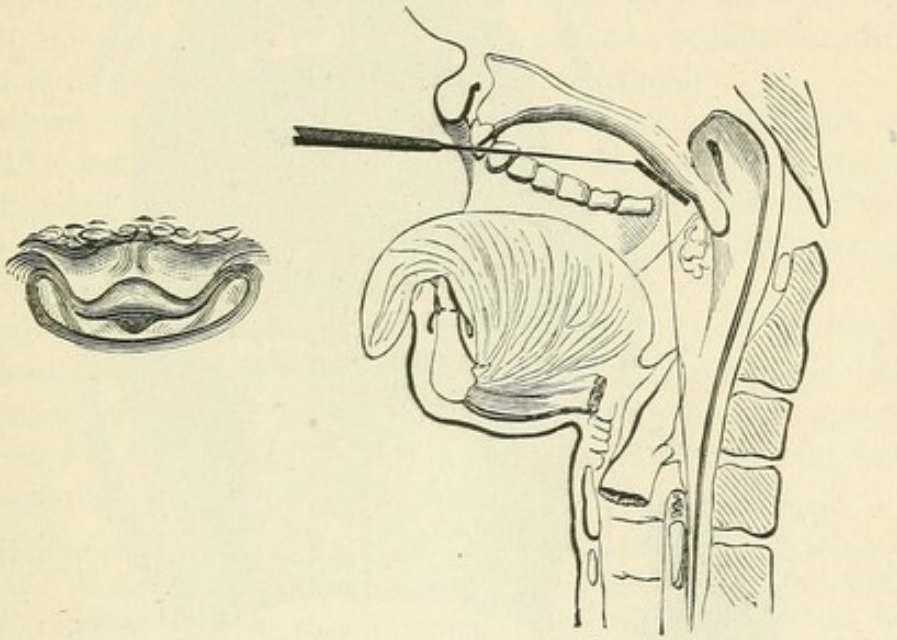

FIG. 60.—POSITION OF HEAD GIVING A POOR VIEW OF LARYNX, AS SHOWN IN THE SMALL CUT AT THE LEFT (BROWNE).

head should be inclined backward (Fig. 59), so that the edge of the upper incisor teeth will be nearly on a horizontal plane with the posterior margin of the soft palate.

The reflector may be worn on the forehead, or preferably before one eye. If the lamp is on the patient's right, the reflector should be placed in front of the examiner's left eye, or *vice versa*. The throat mirror may be held in either hand, the patient's tongue being held by the other or by the patient himself. Right-handed persons should educate the left hand to the task as soon as possible; for when other instruments are to be used, the right hand will be required for them. Even in diagnostic manipulations ambidexterity is very desirable, for by holding the mirror first with one hand and then with the other, any false impressions of asymmetry may be corrected.

In making a laryngoscopic examination, everything being in readiness, the physician takes his position in front of the patient, and fixes the reflector in its place; his eye is now brought within about ten inches of the patient's lips, upon which the light is directed. If the lamp has been placed at the proper distance, a perfect inverted image of the flame will be seen on the patient's lips; otherwise the light should be moved backward or forward until this result is obtained. The patient

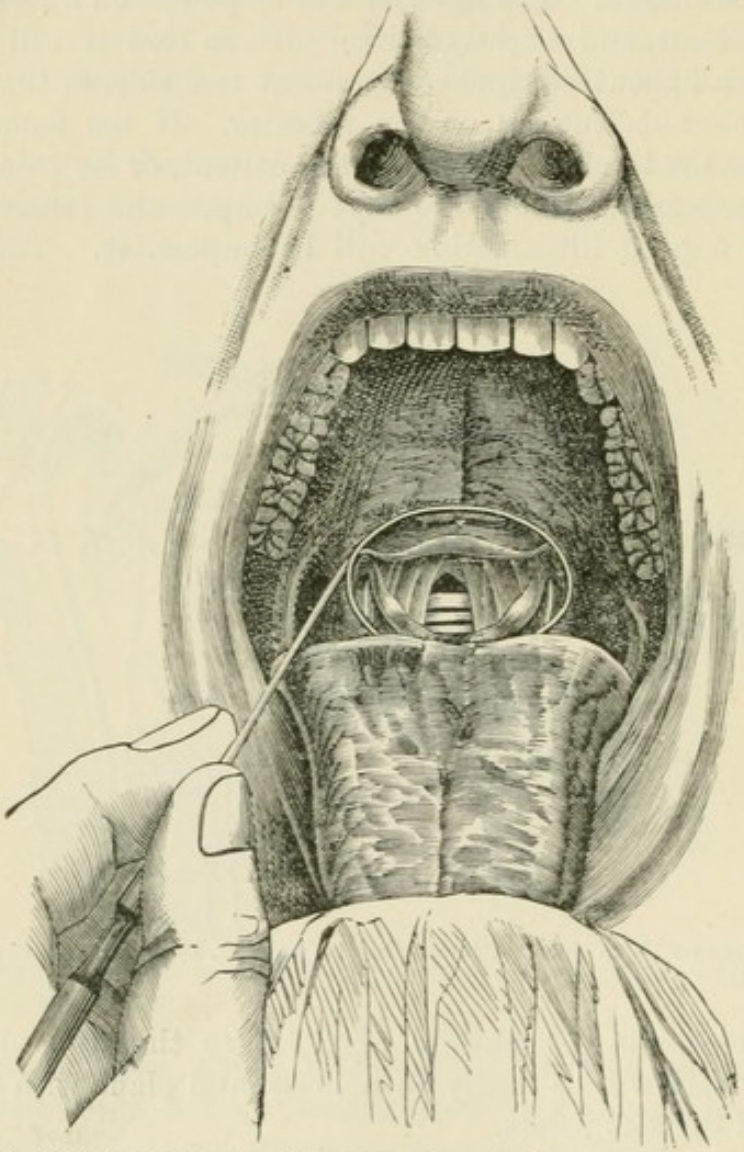

FIG. 61.—THE LARYNGOSCOPIC MIRROR IN POSITION. STEM TO ONE SIDE (COHEN).

is then directed to protrude his tongue, which the physician grasps and holds between his thumb and fore-finger, which have been previously enveloped in a soft napkin. The eye of the examiner is then brought about four inches nearer, and the light from the reflector is so directed that the brightest point falls on the base of the uvula, where it must be retained. The throat mirror, having been warmed for a moment over the lamp and its temperature tested on the cheek or back of the hand, is carried into position in the throat, and, by a slight, steady movement of the mirror, the image of the larynx is brought into view (Fig. 61).

The first difficulty which the beginner experiences is to direct the light into the mouth, and the second is to keep it there. These difficulties may be readily overcome by practice, and should always be mastered on a dummy or some other object before an attempt is made to examine a patient.

The patient should protrude the tongue as far as possible by the muscles of the tongue itself, and it must be held gently by the physician without an attempt to draw it farther out, for such an attempt would cause pain and contraction of its muscles.

A soft cloth is necessary in holding the tongue, not only for neatness, but because if it be grasped simply with the fingers it will slip away. In holding the tongue, the finger which is beneath it should be held slightly higher than the edge of the lower teeth, or the teeth may be covered by a napkin to avoid injury to the frænum.

Whenever both of the physician's hands are to be occupied with instruments, the tongue may be held by the patient; sometimes this is a useful aid in overcoming the individual's nervousness.

The throat mirror employed must correspond to the size of the fauces. The one most generally useful for adults is seven-eighths of an inch in diameter; but mirrors one and one-fourth inches in diameter, or even somewhat larger, may often be employed. The larger the mirror, the better the illumination.

The mirror should be warmed so that the moisture of the breath may not condense upon it. When first placed over the flame, a thin film will be seen to spread over its surface, which disappears as soon as the glass becomes warm. It is then of a proper temperature for use, but should always be tested on the cheek or back of the hand.

Instead of warming the mirror, its surface may be covered with a solution of glycerine and water to prevent condensation of moisture; this does not leave so good a reflecting surface, and, as a result, the image will be less distinct. Other devices have been suggested for preventing condensation of the breath on the mirror, but they are of no practical value.

The mirror is less irritating to the fauces when warm, and it will retain the heat as long as it ought to be kept in the throat. It should be held like a penholder between the thumb and fingers, with the hand bent slightly backward upon the wrist. It should be passed horizontally into the mouth, with the reflecting surface downward, and carried promptly midway between the tongue and the roof of the mouth, back to the uvula, which is caught upon it and carried upward and backward, until the rim of the mirror almost touches the posterior wall of the pharynx. If the uvula hangs too low to be easily caught on the back of the mirror, it may be elevated by causing the patient to take a deep inspiration or to phonate the syllable *ah* or *eh*. If the throat will tolerate it, the mirror may be rested against the posterior wall of the pharynx.

The stem of the instrument may be held either above or at one side, and its handle should be carried outward toward the angle of the mouth, so that the hand will not obstruct the light. The angle of the mirror should be about forty-five degrees to the plane of the horizon, though, in practice, it will be found that good views can be obtained from different points with the mirror in various positions, by altering the relative positions of the physician and patient, or by inclining the patient's head more or less.

If the light has been properly directed, it will now fall on the mirror, whence it will be more or less perfectly reflected into the larynx, an inverted image of which will be seen in the mirror (Fig. 61). If the view be not perfect, the mirror may be slightly rotated or its obliquity altered by moving the handle; but these movements must be few and precise, for if many or executed by an uncertain, tremulous hand, retching is apt to occur.

Beginners generally have considerable difficulty in this manipulation either by losing the light or by being unable to obtain a view of the larynx, on account of an improper position of the throat mirror. In either case, the mirror should be promptly withdrawn and reintroduced, for if held in position while the light is being rearranged, or if moved about in the throat to secure another view, it is likely to irritate the fauces.

With the throat mirror in position, one will obtain a more or less perfect view of the base of the tongue and of the larynx. If only the base of the tongue or the upper part of the epiglottis is brought into view, depressing the handle slightly will expose the parts below; if these are first brought into view, the superior structures may be exposed by elevating the handle. By rotating the mirror slowly, the lateral walls of the pharynx or larynx may be exposed.

To expose the anterior or larygneal surface of the arytenoids, the head should be thrown slightly backward during a deep inspiration, and the light should be directed more posteriorly than in illuminating the cords, by holding the throat mirror more nearly horizontal. To expose their posterior or pharyngeal surface, the head should be nearly erect, and the mirror should be held as just directed while the voice is sounded.

To examine either side, the mirror should be placed partly upon the opposite side of the fauces, with its obliquity changed so as to illuminate the parts to be inspected.

In order to obtain a good view of the laryngeal surface of the epiglottis, the patient should be directed to sound a high note quickly and with considerable force. This throws the cartilage upward with a sudden jerk. An inspiration accompanied with sound or an ironical laugh will answer the same purpose.

The hand which holds the mirror may be steadied by resting the ring and little fingers on the patient's cheek.

The mirror should not be kept in the throat more than twenty or thirty seconds, but the examination may be continued by reintroducing it several times.

Whenever the slightest indication of retching occurs, the mirror must be instantly withdrawn, but, after a few moments, another trial may be made, which the patient will usually tolerate as well as the first.

When inserting the mirror, its reflecting surface should not touch the tongue, nor its back rub against the palate. The former accident clouds the reflecting surface, and either is likely to cause retching or an attempt to swallow, which will prevent the examination.

OBSTACLES TO LARYNGOSCOPY.

The obstacles frequently encountered in laryngoscopy can usually be overcome by a little tact and patience, at least at a second sitting. We should not expect a thorough view of the larynx without introducing the mirror two or three times; though, if the patient's throat is not sensitive, by rotating the mirror slightly the entire larynx may sometimes be inspected with a single introduction of the mirror.

The principal obstacles to be overcome are: an elongated uvula, enlarged tonsils, irritable fauces, a short frænum, arching upward of the back of the tongue, and a pendent epiglottis. In two cases, one an actor, and the other an elocutionist, I have found difficulty in inspecting the larynx apparently on account of hypertrophy of the lingual muscles, which greatly restricted the space between the tongue and the posterior wall of the pharynx.

AN ELONGATED UVULA, hanging below the mirror, appears as though curled over the lower edge and resting upon the reflecting surface. This is very confusing and prevents a view of the parts below.

To obviate this difficulty in ordinary cases, it is only necessary to use a large mirror and to be careful in placing it against the uvula. Mirrors have been devised with a little pocket in the back for catching the uvula, but they are now rarely if ever used. If the uvula is so long that it cannot be managed with a large mirror, it may be contracted by astringents; if these are inadequate, it should be amputated and the examination made at a subsequent sitting.

On account of IRRITABLE FAUCES some patients cannot bear simple inspection of the mouth without gagging or retching; others are so affected when the tongue is protruded; still others as soon as the throat mirror touches the fauces.

To overcome these difficulties, the patient should be fully impressed with the necessity of the examination, and urged to restrain himself from retching; the mirror should then be introduced during a deep inspiration or as the patient says *eh* or *ah*, which elevates the uvula, and, by thus preventing the necessity for pressure against the palate, secures much greater tolerance of the instrument.

With nervous patients it is often best, for the sake of first gaining their confidence, to introduce the mirror once or twice so as just to touch the palate, and then withdraw it at once without attempting to see the larynx. Ice may be sucked for fifteen or twenty minutes, to produce some degree of temporary local anæsthesia. If these devices fail, the most feasible method for overcoming the disposition to retching is an application a few times of a small amount of a ten-per-cent solution of cocaine, by spray.

Many persons, in whom the pharynx is sensitive, will tolerate an examination at a second or third sitting, in whom hardly a glimpse could be obtained at the first. In such cases it is a good plan to have the patient educate the throat to bear instruments, by introducing a spoon-handle against the uvula before a mirror several times daily during the interim.

In cases of irritability of the fauces, some laryngologists recommend titillation of the palate with a probe or a penholder before attempting to introduce the mirror, in order that the parts may become accustomed to manipulation. Various other devices have been recommended for overcoming the sensitiveness such as painting the fauces with chloroform and morphine, inhalation of a few whiffs of chloroform, and the internal use of large doses of potassium bromide; but none of these measures are very satisfactory. Ordinarily we will succeed best simply by patience and care in introducing and holding the mirror, supplemented, when necessary, by the use of ice or cocaine. The fauces are more irritable when the stomach is disordered and during digestion than at other times; therefore it is best, whenever the throat is sensitive, to make the examination before eating or not until three or four hours afterward.

A SHORT FRÆNUM is one of the minor obstacles. If it proves very troublesome, it may be cut with a pair of blunt-pointed scissors.

ARCHING OF THE TONGUE occurs in some patients just as the mirror is being carried between the teeth, the posterior part of the tongue arching upward, so as to touch the soft palate, and thus preventing the passage of the mirror into the fauces; or remaining here to intercept the rays of light after the mirror is in position. This difficulty is best overcome by cautioning the patient not to strain and by care not to draw the tongue far out of the mouth or downward toward the chin.

Sometimes a good view of the larynx can be obtained in these instances by holding the throat mirror nearly horizontally against the palate, and reflecting the light upon it from below upward. In some cases, the patient, by watching the movements of his tongue in a hand mirror, may be able to keep its base depressed. Other patients will need to practise before a mirror at home for several days before control of the organ can be obtained. Tongue depressors seem indicated in these cases, but are of little value.

GREATLY ENLARGED TONSILS may prevent the introduction of any mirror into the throat; in such cases the only remedy is excision. When

they are only moderately enlarged, it will sometimes be impossible to introduce the ordinary mirror without touching them both, and perhaps causing retching; but in many cases, if the mirror is carried promptly between and behind the tonsils, the throat will remain quiet, even though both sides have been touched. In other cases it is best to use an oval mirror, which may be passed into the fauces without touching the tonsils.

A LARGE OR PENDENT EPIGLOTTIS is sometimes an insurmountable obstacle to laryngoscopy. When the glosso-epiglottidean ligaments are relaxed, or when the epiglottis is swollen, it falls downward, so that its

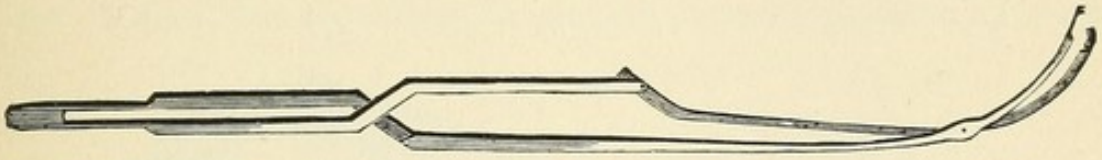

FIG. 62.—BRUNS' PINCETTE.

free edge may rest against the pharyngeal wall, leaving little if any space for the passage of light. In some of these cases we can obtain a view of the larynx by causing the patient to sound the letter *e* in a high key or to utter a high falsetto note. A vocal sound, as *ah* or *eh* made during inspiration, will have a similar effect. By a laugh or a cough the epiglottis may be thrown upward with a sudden jerk. In other instances it is only necessary for the patient to drawn a deep breath in order to raise the epiglottis sufficiently to give a view beneath it. Frequently by passing the mirror lower into the pharynx, and more perpendicularly than usual, the inferior surface of the epiglottis and other portions of the larynx may be seen.

Various instruments have been devised for lifting the epiglottis. The best of these is known as Voltolini's staff, a stout whalebone or metallic rod, bent nearly to a right angle about an inch from the end, with its terminal extremity turned slightly backward. It may be passed behind the lip of the epiglottis, so as to lift and draw it forward.

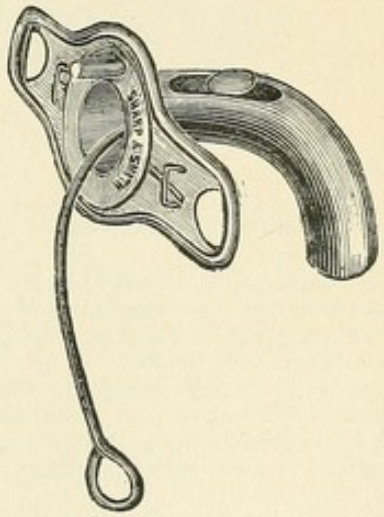

FIG. 63.—INFRA-GLOTTIC LARYNGOSCOPY. Small metallic mirror in position in the fenestra of the tracheal canula.

Occasionally when operations are to be performed, or for simple inspection, some special instrument may be necessary to hold the lip of the epiglottis forward. For this purpose Bruns' pincette has been recommended. Instruments of this kind, however, usually cause too much irritation to be tolerated, and a simple bent staff or strong probe will be found preferable.

It occasionally happens that only the posterior part of the larynx can be seen, and the vocal cords cannot be brought into view. In such in-

stances the movements of the arytenoid cartilages may be seen sufficiently to enable us to judge of the mobility of the cords; but the appearance of the tissue covering them is not an accurate indication of the condition of the mucous membrane in other portions of the larynx.

INFRA-GLOTTIC LARYNGOSCOPY.

It is sometimes desirable to inspect the larynx from below, which may be done, after tracheotomy, through a fenestra in the canula, by the aid of a small metallic mirror (Fig. 63).

CHAPTER XVII.

DISEASES OF THE THROAT.—*Continued.*

THE LARYNX AND RHINOSCOPY.

THE image of the larynx, as seen in the throat mirror, is reversed, so that the anterior portion, nearest the observer, appears in the glass above and farthest from its surface, the portion normally posterior appearing

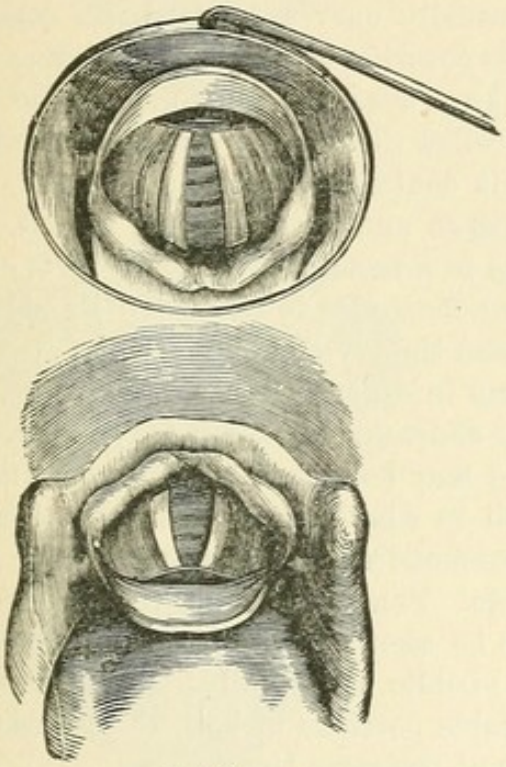

FIG. 64.

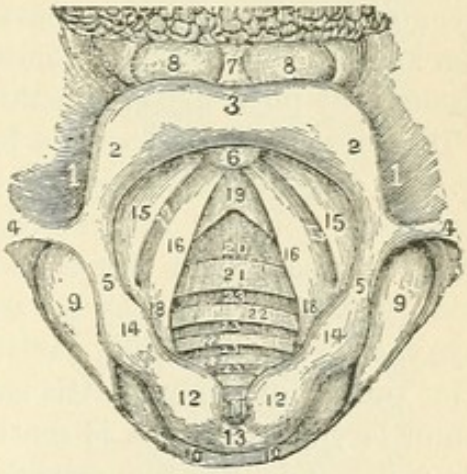

FIG. 65.

FIG. 64.—RELATIVE POSITIONS OF LARYNX AND ITS IMAGE IN THE LARYNGOSCOPIC MIRROR (COHEN).

FIG. 65.—NORMAL LARYNX IN RESPIRATION, ENLARGED. Parts exaggerated to render them more conspicuous. 1, 1, Lingual surface of epiglottis; 2, 2, laryngeal surface of epiglottis; 3, indented crest of epiglottis; 4, 4, pharyngo-epiglottic folds; 5, 5, ary-epiglottic folds; 6, cushion of epiglottis; 7, glosso-epiglottic ligament; 8, 8, valeculæ; 9, 9, pyriform sinuses; 10, 10, posterior pharyngeal wall and entrance into œsophagus; 11, inter-arytenoid incisure; 12, 12, cartilages of Santorini; 13, inter-arytenoid fold; 14, 14, cartilages of Wrisberg; 15, 15, ventricular bands; 16, 16, vocal cords; 17, 17, ventricles; 18, 18, posterior vocal processes; 19, thyroid cartilage; 20, crico-thyroid membrane; 21, cricoid cartilage; 22, 22, 22, rings of trachea; 23, 23, 23, 23, interspaces between rings of trachea (Cohen).

below close to the lower edge of the mirror. The sides of the larynx are not reversed in the image.

An image of the whole larynx can seldom be obtained at a single glance; but by slight rotation of the mirror, with elevation and depression of the handle, so as to alter the plane of the reflecting surface, the different parts may be brought into view. The vocal cords, because of

their white appearance and frequent respiratory movements, naturally attract the most attention, and when once seen can hardly be forgotten; but the epiglottis comes first into view.

THE NORMAL LARYNX is shown in a somewhat exaggerated form (Fig. 65) in order that the parts may be more clearly identified.

THE EPIGLOTTIS is a leaf-like valve, which covers the upper opening of the larynx and closes it during deglutition.

The base of the epiglottis—in reality the apex of the cartilage—is connected with the thyroid cartilage at its receding angle by a long narrow band, known as the thyro-epiglottic ligament; a small band, the hyo-epiglottic ligament, connects it with the posterior surface of the hyoid bone. The free extremity is broad and rounded. The lingual or upper surface of this cartilage usually curves forward, its concavity toward the base of the tongue. Its covering of mucous membrane forms a median and two lateral folds, known as the glosso-epiglottic folds. The central one of these is also called the frænum of the epiglottis, or the glosso-epiglottic ligament as it contains a ligamentous band. The lateral folds contain no fibrous tissue and are frequently absent. The laryngeal or inferior surface curves in a reverse direction. It is convex from above downward, and concave from side to side. To its sides are attached the pharyngo-epiglottic and the ary-epiglottic folds.

It varies greatly in size and form in different individuals (Figs. 66 to 71). It may be long and thin, or short and thick; it may be broad, or narrow and pointed; its free edge may be curved like a bow, it may be folded in upon itself like a scroll in what is known as the jews-harp form (Fig. 70), or it may be asymmetrical. It may cover the whole larynx, or it may be nearly invisible. Sometimes only the upper or anterior surface of the epiglottis can be seen, at other times its lower portion or laryngeal surface is most visible; again, only its tip is brought into view; and still again considerable portions of both the anterior and the posterior surfaces may be seen at the same time.

With respiration, the lip of the epiglottis rises and falls slightly. With phonation it is generally thrown upward, and in deglutition it is carried downward to the posterior border of the larynx.

The whole epiglottis is seldom visible even to a skilful laryngologist. Usually a portion of its upper surface is visible on each side. In the middle, its laryngeal surface is turned upward like a lip, and below this a small prominence may frequently be seen near the base of the epiglottis, known as its cushion, pad, or protuberance (Fig. 68).

The color of this organ varies in different parts. The upper surface is of a pinkish hue, and frequently blood-vessels may be seen crossing it. The lip looks like a yellow cartilage, as it really is, covered with mucous membrane. The cushion generally appears of a much brighter red color than other portions of the epiglottis. When the whole of the laryngeal surface can be seen, it often has a uniform bright-red color,

which might be easily mistaken for congestion. When only the edge of the epiglottis is visible, it appears like a pale whitish line just beneath the base of the tongue.

THE VALLECULÆ, upon either side of the frænum of the epiglottis, are two sinuses known also as the lingual sinuses, closely resembling depressions, such as might be made by pressing the tips of two fingers into

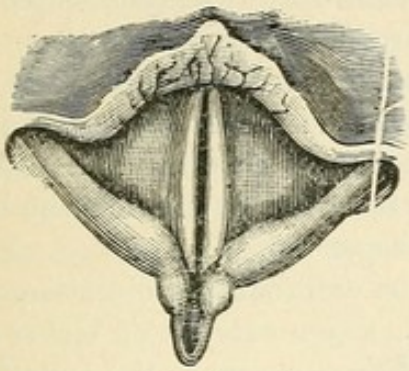

FIG. 66.

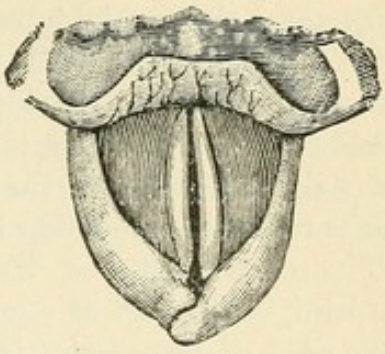

FIG. 67.

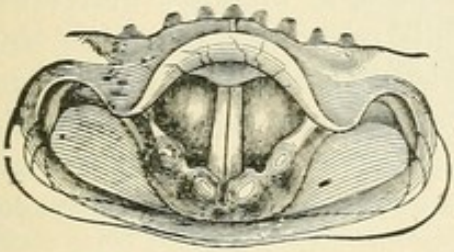

FIG. 68.

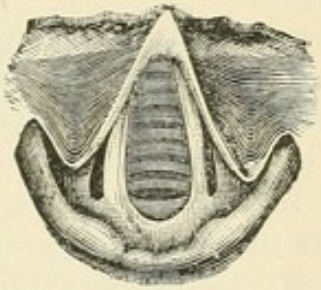

FIG. 69.

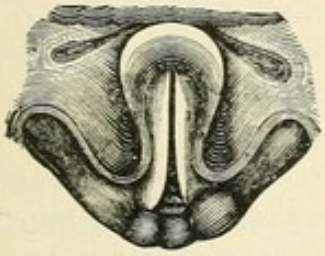

FIG. 70.

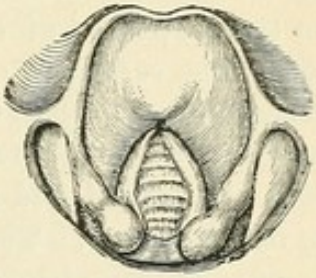

FIG. 71.

FIGS. 66 to 71.—NORMAL LARYNX, SHOWING VARIOUS FORMS OF EPIGLOTTIS AND SUPRA-ARYTENOID CARTILAGES.

FIG. 66.—PITCHER-SHAPED INTER-ARYTENOID FOLD, PHONATION.

FIG. 67.—LAPPING OF ARYTENOID CARTILAGES IN PHONATION, WITH GAPING OF VOCAL CORDS.

FIG. 68.—CUSHION OF EPIGLOTTIS VISIBLE; NO GAPING OF VOCAL CORDS IN PHONATION (ZIEMSSEN).

FIG. 69.—POINTED EPIGLOTTIS; VENTRICLES DISTINCT; INSPIRATION.

FIG. 70.—"JEWS-HARP" OR OMEGA-LIKE EPIGLOTTIS.

FIG. 71.—FEMALE LARYNX IN RESPIRATION (COHEN).

The female larynx may have the form shown in any of the preceding figures.

some plastic substance (Fig. 65). They vary greatly in depth and in width in different individuals, and in various positions of the epiglottis in the same individual. These sinuses should always be examined as they frequently give lodgement to portions of food which are a source of irritation, and they are sometimes the seat of ulcers.

THE ARYTENOID CARTILAGES—so named on account of their apparent resemblance during phonation to the nose of a pitcher—appear beneath the free edge of the epiglottis. They are two in number, one upon each side. They are located at the back of the larynx, resting upon the upper border of the cricoid cartilage. Each of these cartilages is somewhat pyramidal. The apex, which is slightly pointed and curved upward and inward, is surmounted by a small conical nodule, which has been named the corniculum laryngis or cartilage of Santorini.

THE CARTILAGES OF SANTORINI, which are usually about the size of a millet seed, are most prominent when the glottis is closed, as in phonation. The mucous membrane immediately covering their apices is of a lighter hue than that in other parts of the larynx, but the light color is usually surrounded by a zone of deeper red.

THE CARTILAGES OF WRISBERG are just external to the cartilages of Santorini, in the fold of mucous membrane which extends on either side to the edge of the epiglottis, prominences known also as the cuneiform cartilages.

These cartilages vary considerably in form in different individuals. They are usually round, but are occasionally triangular, the apices being directed downward. Sometimes they are hardly visible, but they are generally quite distinct and fully as large as the cartilages of Santorini. These, like the cornicula, are of a lighter color than the folds which contain them, but they are usually surrounded by a zone of mucous membrane redder than the general surface.

In a few instances a small nodule, due to a third cartilage, is seen between the cartilages of Wrisberg and the cartilages of Santorini on each side. The cartilages of Wrisberg and those of Santorini are sometimes termed the supra-arytenoid cartilages.

THE ARYTENO-EPIGLOTTIDEAN FOLDS or the ARY-EPIGLOTTIC FOLDS constitute the lateral and part of the posterior border of the superior opening of the larynx. They consist of folds of mucous membrane, one on each side, which extend like bows from the arytenoid cartilages upward and forward to the sides of the epiglottis. They are usually from one-twelfth to one-eighth of an inch in thickness, but are occasionally thin and sharp. In color they closely resemble the gums, and are somewhat lighter than the zones about the bases of the supra-arytenoid cartilages.

THE PYRAMIDAL, PYRIFORM, OR LARYNGO-PHARYNGEAL SINUSES are found external to the folds just named, and between them and the wings of the thyroid cartilage. The broad end of each sinus is directed forward, and its apex backward. It is bounded internally by the quadrangular membrane, the upper border of which is formed by the ary-epiglottic fold, anteriorly by the wing of the thyroid cartilage, and laterally by the wall of the pharynx. Like the valeculæ, these sinuses often give lodgement to foreign bodies, and are frequently the seat of ulcerations.

THE VENTRICULAR BANDS, known also as the superior or false vocal cords, the regulators of the glottis, or the superior ligaments of the larynx, are thick folds of mucous membrane which stretch across the larynx in an antero-posterior direction, about half an inch below its superior opening and a short distance above the true vocal cords. They are frequently very prominent, standing out in thick welts from the sides of the larynx. In other instances, they can hardly be distinguished from the surrounding tissues. They are of a deeper red color than the tissues above them, but their inferior or inner borders generally appear pale in the laryngoscopic image, on account of being illuminated more perfectly than the surrounding parts. Just beneath the anterior ends of the false vocal cords and above the true cords may frequently be seen a fossa, about the size of a pin's head which has been

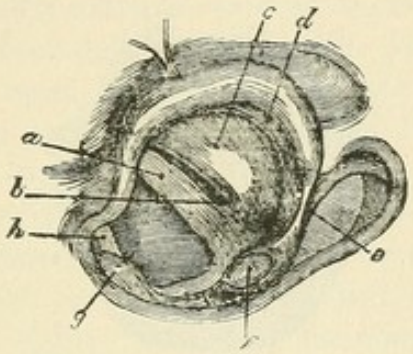

FIG. 72.—VIEW OF LEFT SIDE OF LARYNX (TÜRCK). *a*, Left vocal cord; *b*, posterior portion of ventricle; *c*, left ventricular band; *d*, posterior surface of epigloteis; *e*, border of ary-epiglottic fold; *f*, left cartilage of Wrisberg; *g*, right cartilage of Wrisberg; *h*, right vocal cord.

named by Mackenzie the fossa innominata. This communicates with the laryngeal sinuses upon either side.

THE VENTRICLES OF THE LARYNX are found immediately beneath the ventricular bands. These consist on either side of an oblong fossa, which is the opening to a *cul de sac* of mucous membrane, known as the sacculus laryngis. They are bounded above by the false vocal cords; below, by the true vocal cords; and externally, by the thyro-arytenoid muscles.

The ventricles are seldom seen, and, when visible, usually appear merely as dark lines; but occasionally they are patulous, with a width of nearly one-eighth of an inch.

THE SACCULUS LARYNGIS extends upward and outward in a conical form beneath the ventricular band. The mucous membrane lining it is studded with the openings of sixty or seventy follicular glands, the secretion from which is apparently intended for lubricating the vocal cords. This pouch is covered by a fibrous membrane, and this membrane by muscular tissue, which, according to Hilton, compresses the sacculus and discharges its secretion upon the vocal cords.

THE VOCAL CORDS, known also as the inferior or true vocal cords, are the most important objects to be seen on inspection of the larynx.

They appear as two pearly white bands stretched, one along each side of the larynx from its anterior to its posterior part.

In the adult they vary from five-eighths of an inch to one inch in length, and are usually about one-eighth of an inch in breadth; they are sometimes perfectly white in women, but in men they are usually of a yellowish white hue. They consist of fibrous bands covered by a thin layer of closely adherent mucous membrane, being attached anteriorly to a depression between the alæ of the thyroid cartilage, posteriorly to the anterior angles at the base of the arytenoid cartilages.

During respiration the cords alternately approach each other and recede, leaving between them a triangular opening for the passage of air. The cords and the space between them form what is known as the *glottis*. The free edges constitute the lips of the glottis, and the chink or

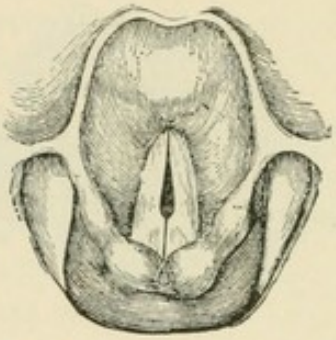

FIG. 73.—NORMAL LARYNX OF WOMAN IN FORMATION OF HEAD TONES (COHEN).

fissure between them is called the rima glottidis. The front of the rima is formed by the anterior commissure of the vocal cords, its sides by the cords themselves, and its base by the arytenoid cartilages and the inter-arytenoid fold. In the adult, this fissure varies in length from seven to ten lines in women, and from ten to thirteen in men. At its widest part it ordinarily measures from three to six lines, but on deep inspiration it may measure as much as eight or ten lines. In children, it is of course much smaller.

On inspiration, the cords separate widely at their posterior extremities; but their anterior extremities remain close together, thus forming a triangular opening. On expiration they approach more nearly together, and in phonation their two borders are more or less closely approximated, but there is usually a narrow fissure between them throughout their entire length. In women, and occasionally in men, during the production of head tones, the vocal processes are pressed firmly together, so that the fissure is left only between the anterior parts of the cords.

From a careful photographic study of the larynx during the production of the singing voice, Thomas R. French (Transactions of American Laryngological Association, 1888) concludes that the female voice has three and the male voice two registers; the transition from one to the next higher being usually marked by backward movement of the epi-

glottis, change in the shape of the glottis, shortening of the cords, and an apparent increase in their tension. Protrusion of the tongue does not materially affect the laryngoscopic appearance.

The cords are sometimes lengthened in men on changing to a higher register.

THE PROCESSUS VOCALES or vocal processes are sometimes seen as four yellowish spots, two anteriorly and two posteriorly, where the vocal cords are attached to the cartilages, but the anterior processes are not often visible. Usually, when we speak of the vocal processes, simply the anterior angles of the arytenoid cartilages are referred to. Carl Seiler has described narrow fusiform cartilages, found along the edge of the vocal cords in women. These are only rudimentary in men.

THE INTER-ARYTENOID FOLD or posterior commissure is a band of mucous membrane which extends between the arytenoid cartilages. The prominence of this fold depends upon the position of the cartilages. When the glottis is open, it may measure six or eight millimetres in length; but when the cords are approximated, it is folded upon itself so that it can hardly be seen.

THE CRICOID CARTILAGE may usually be seen a short distance below the vocal cords, separated from their anterior extremities by the lower portion of the thyroid cartilage and by the crico-thyroid membrane. This cartilage is of a lighter hue than the membranous tissue above or below it, and is similar in color to the rings of the trachea.

THE TRACHEAL CARTILAGES or rings of the trachea are usually visible, arching across this tube from side to side with their concavities directed inward and downward. The upper of these rings are very distinct and of a yellowish or a light pinkish hue. They are separated from each other by the intervening membranous tissue, which is of a darker color.

As we carry the inspection farther down the trachea, the cartilages appear narrower and narrower until their outlines are finally lost.

The mucous membrane lining the trachea is generally paler than that covering the surface of the larynx.

Considerable variety in the shape and movements of different parts of the larynx may occur within the limits of health. This is especially the case with the epiglottis; and variations in the appearance of the arytenoid cartilages and of the commissures, and slight alterations in other parts of the larynx may occasionally be found, as illustrated in Figs. 66 to 71. The epiglottis may possess any of the various forms already spoken of. The supra-arytenoid cartilages vary considerably in their size and form, as already mentioned. The position of the arytenoids varies constantly with respiration and phonation, and may be quite different in healthy individuals (Figs. 66 to 71).

In disease of the larynx, changes in its form and movements constitute the principal signs. There may be hypertrophy or swelling of its various parts, with more or less loss of movement, or ulceration may

have destroyed more or less of the tissues. Sometimes the epiglottis is so swollen and wrinkled as to be hardly recognizable; its free edge may be ulcerated, or the cartilage may be partly or entirely destroyed by the same process. Swelling of the inner extremity of the ary-epiglottic folds and of the tissues surrounding the arytenoid cartilages is frequently found upon one or both sides. Loss of movement occurs from cicatricial adhesions or paralysis. Morbid growths are of comparatively frequent occurrence.

EXAMINATION OF THE TRACHEA.

In order to obtain a good view of the trachea, it is usually necessary to hold the mirror more nearly horizontal than in the examination of the larynx, so as to reflect the light somewhat more posteriorly. The glottis must be widely opened, and the focal point of the light must fall

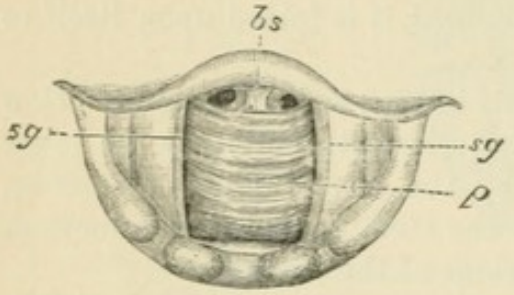

FIG. 74.—VIEW OF POSTERIOR WALL OF TRACHEA AND BRONCHI. *bs*, Bifurcation of trachea; *sg*, subglottic region; *p*, posterior wall of trachea (Mackenzie).

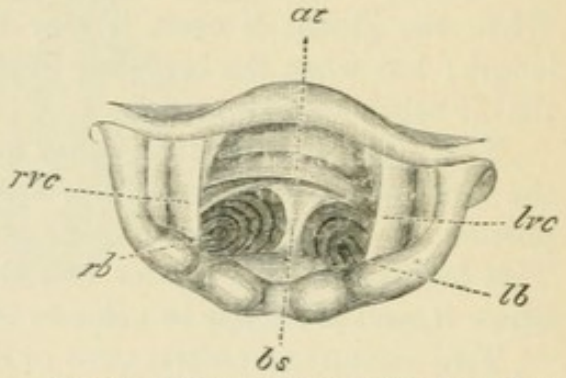

FIG. 75.—VIEW OF ANTERIOR WALL OF TRACHEA AND BRONCHI. *at*, Anterior wall of trachea; *rvc*, *lvc*, vocal cords; *rb*, right bronchus; *lb*, left bronchus; *bs*, bifurcation or bronchial spur (Mackenzie).

upon the parts to be examined; that is, at a distance of from seven to eleven inches within the lips, or from twelve to seventeen inches from the reflector, according to the portion of the tube to be examined. Sometimes we can obtain a good view by elevating the patient to a plane above that of the observer, and holding the throat mirror almost horizontal so that the light may be thrown upon it from below upward.

To expose the posterior wall of the larynx and the trachea, the patient's head should be kept erect, and the mirror held in a nearly horizontal position.

With a good light and a favorable condition of the larynx and trachea, the openings of the main bronchi can frequently be seen, and in some instances a few of their cartilaginous rings may be counted. To illuminate the bifurcation of the trachea, a good plan is first to obtain a view of the laryngeal surface of the epiglottis, and then, by gradually changing the obliquity of the mirror, direct the rays farther and farther downward along the anterior surface of the trachea until the deeper parts are brought into view.

RHINOSCOPY.

Rhinoscopy or examination of the nasal cavities is termed anterior or posterior according to the position of the parts inspected.

ANTERIOR RHINOSCOPY.

Anterior rhinoscopy or the inspection of the anterior nares is performed with the aid of the laryngoscopic reflector and a nasal speculum. Various instruments have been made for the purpose. A simple bivalve

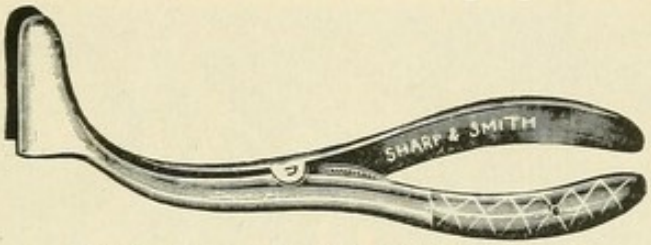

FIG. 76.—INGALS' NASAL SPECULUM (3-5 size).

speculum, such as shown in Fig. 76, is most satisfactory for purposes of diagnosis; but when operations are to be performed, instruments that will retain their position when placed in the nostrils are preferred by some laryngologists (Figs. 77 and 78). No special directions are needed for anterior rhinoscopy, excepting that, in order to view the back part of the nasal cavities from the front, a condenser, and a reflector as described with the laryngoscope, are very desirable, and it is absolutely

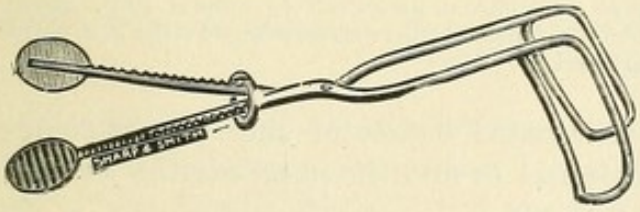

FIG. 77.—JARVIS' SMALL NASAL SPECULUM (½ size).

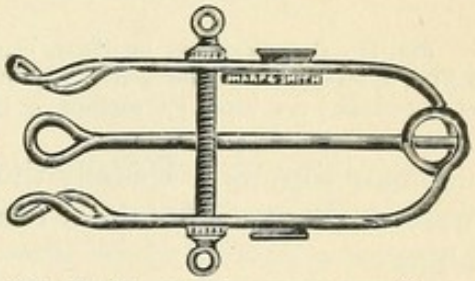

FIG. 78.—SAJOUS' SELF-RETAINING NASAL SPECULUM (3-5 size).

necessary that the light be properly focussed according to the principles laid down in speaking of condensing lenses. No obstacles will be found to the examination, excepting in unruly children, unless there be some deformity or swelling of the turbinated bodies. The latter is common, but may usually be quickly reduced by a small amount of a spray of cocaine. The nares are usually about one-eighth of an inch in width and from an inch to two inches in height. The inferior turbinated body is seen occupying about two-thirds of the outer wall; and the middle turbinated, much smaller, is seen at the upper part of the cavity occupying about one-quarter of the outer wall, and usually approaching to within from one-twelfth to one-sixteenth of an inch of the septum.

The superior turbinated body cannot be seen. The whole cavity is

covered with smooth mucous membrane, normally of about the same color as that covering the gums, but often, under less perfect illumination, appearing slightly congested. The normal relations of the parts, about an inch back of the nostrils, are shown in the accompanying cut from the photograph of a frozen section prepared for me by C. H. Stowell, of Washington, D. C. The soft tissues are somewhat shrunken, as always found in the cadaver.

In about two-thirds of all cases there is some disparity in size in the two cavities, due to deflection or to outgrowths from the bony or carti-

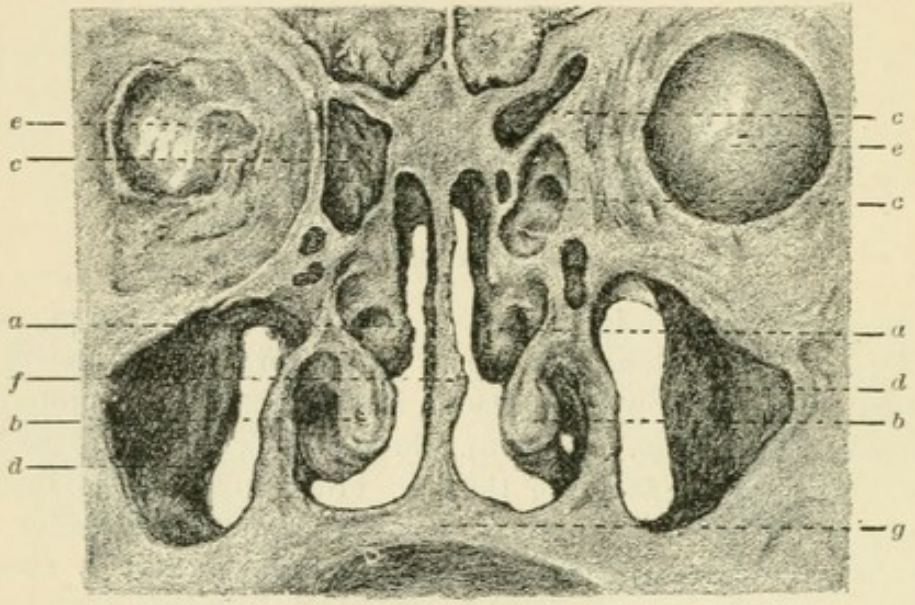

FIG. 79.—CROSS-SECTION OF HEAD, LOOKING FROM BEFORE BACKWARD (4-5 natural size). Showing: *a, a*, middle turbinated bodies; *b, b*, inferior turbinated bodies; *c, c, c*, ethmoid cells; *d, d*, antra of Highmore; *e, e*, orbits; *f*, septum; *g*, hard palate.

laginous septum. Usually the turbinated bodies of one side are somewhat swollen, so that it is exceptional to find the nasal cavities exactly alike.

POSTERIOR RHINOSCOPY.

Posterior rhinoscopy, or inspection of the vault of the pharynx and posterior nares, is practised with instruments similar to those used in the inspection of the larynx, and in much the same manner, excepting that a smaller mirror is necessary, and its reflecting surface is turned upward instead of downward.

A mirror from half to five-eighths of an inch in diameter is usually employed, and it is generally best to have a flexible stem, which may be readily bent to conform to the floor of the mouth (Fig. 81).

The mirror may be set at right angles to the stem, or at the same angle as the laryngeal mirrors, or at an angle between these two; but this is a matter of little importance, as the obliquity of the mirror may be easily changed by raising or lowering the handle. Special throat mirrors have

been constructed for rhinoscopy (Fig. 80), but they are not superior to those already described. A tongue depressor will commonly be needed in rhinoscopy, and various forms of blunt hooks and other instruments may be used for holding the uvula; these latter are rarely employed and are seldom if ever of use except during operations.

In rhinoscopy, the patient should sit erect, and the head must not be thrown backward, but may be slightly inclined forward. The physician should take a position as for laryngoscopy, or on a slightly higher level, and the light should be placed as for inspection of the larynx, except

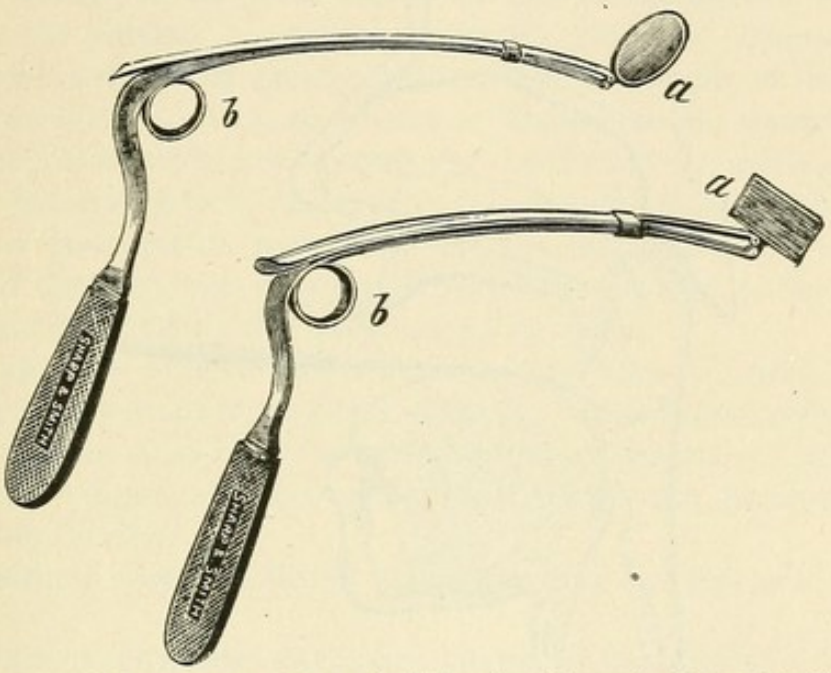

FIG. 80.—FRAENKEL'S RHINOSCOPE. The angle of the mirror (*a*) can be changed at will by moving the sliding rod at *b*.

that it should be on a level with the patient's mouth instead of his eyes. The patient's tongue should not be protruded, but must be left in the floor of the mouth, where it will generally need to be held by a tongue depressor, though some patients can control it better without an instrument.

The rhinoscope in general use is a number one or number two laryngeal mirror, the stem of which is bent to conform it to the floor of the mouth (Fig. 81). It is to be warmed and introduced with the same care as in laryngoscopy, with the reflecting surface upward. It should be carried back to the posterior pharyngeal wall, though it is better to avoid touching it. The surface of the mirror will then be at an angle of about thirty degrees to a horizontal plane. The stem may be rested on the dorsum of the tongue, but care must be taken not to touch the base of this organ. The handle should be depressed nearly to the lower incisor teeth. A common cause of failure in this examination is holding the mirror handle too high.

The mirror should be introduced first on one side of the uvula and

then on the other, to give a view of different parts. In some cases a larger mirror may be used if it is held completely below the uvula.

When the mirror is in position, if only the posterior wall of the pharynx is seen, in order to expose the posterior nares, the handle must be still farther depressed, or the mirror must be withdrawn and bent more nearly to a right angle with the stem. If at first only the uvula and posterior surface of the palate are exposed, the handle must be elevated to obtain a view of the posterior nares or vault of the pharynx.

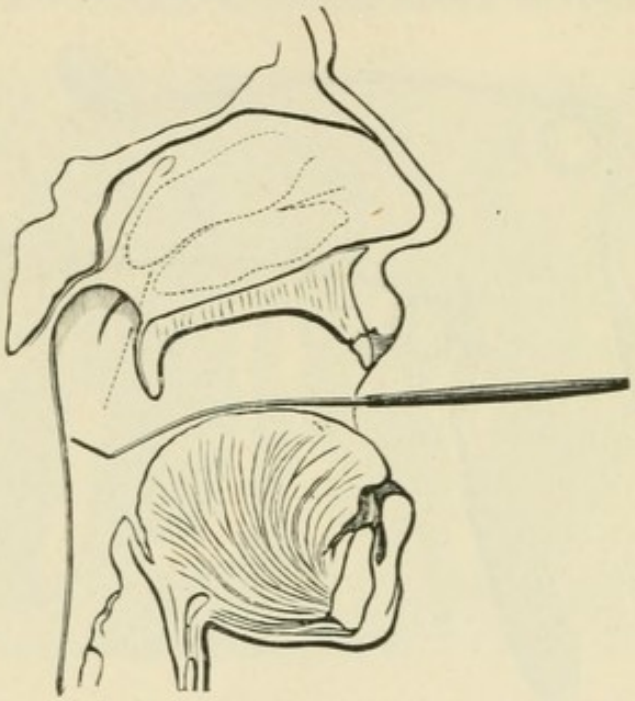

FIG. 81.—POSITION FOR RHINOSCOPY, SHOWING ALSO CURVE IN STEM OF MIRROR. (Slightly altered from Browne.)

The mirror may be rotated slightly to obtain an image of the lateral walls of the pharynx or of the orifices of the Eustachian tubes.

OBSTACLES TO POSTERIOR RHINOSCOPY.

Some of the obstacles to rhinoscopy are the same as those to laryngoscopy, and demand similar treatment. Thus, the uvula may be elongated and the fauces may be irritable.

The principal difficulties met in the examination of the posterior nares are: irritability of the tongue causing the patient to retch whenever an attempt is made to depress it with the spatula; an elongated or sensitive uvula; irritability of the fauces; too close approximation of the uvula and palate to the posterior pharyngeal wall.

IRRITABILITY OF THE TONGUE will sometimes prevent the use of a tongue depressor, but it may generally be employed if the physician is careful not to allow it to slip too far back on the base of the organ. In many cases it is not necessary to depress the tongue with any instrument, if patients are instructed to allow it to remain passive in the floor

of the mouth. A hand mirror, in which the patient can see his tongue, will sometimes aid him materially in controlling it. In other cases the tongue may be held as in laryngoscopy.

Some one of these methods will nearly always overcome this difficulty; but if they should all fail, the patient must practise at home before a mirror until a spatula can be tolerated, or until the tongue can be held without one.

Instruments have been constructed which combine a tongue depressor and the throat mirror; but they are not necessary, for, whenever the physician desires to use both hands, the care of the spatula may be intrusted to the patient. Instruments of this kind are objectionable, as the depressor necessarily greatly restricts the movements of the mirror.

An ELONGATED UVULA, so relaxed as to become an obstacle to the use of the rhinoscopic mirror, may be contracted by astringents. If the uvula is too long to be managed in this manner, it should be excised.

Various instruments have been devised for raising the uvula and drawing it forward, but they are of very little service, as they usually cause so much irritation that they cannot be borne.

IRRITABILITY OF THE FAUCES can be overcome in many instances by allowing the patient to suck bits of ice for ten or fifteen minutes. In other cases there must be prolonged practice by the patient at home in holding the tongue, and in touching the palate and pharyngeal wall with a spoon-handle.

In obstinate cases a solution of cocaine may be used as in laryngoscopy.

CLOSURE OF THE POST-PALATINE SPACE, by contraction of the palatine muscles, often occurs the moment a patient opens his mouth, and it sometimes continues in spite of our best directed efforts to overcome it. This is the most common difficulty with which we have to contend in illuminating the vault of the pharynx and the posterior nares.

Sometimes this difficulty may be overcome by cautioning the patient to allow the fauces to remain passive when the mouth is opened, or by directing him to simply open the mouth wide without attempting to show the throat. Then, by introducing the mirror carefully so as not to touch any part of the fauces, and removing and reintroducing it several times if necessary without attempting to obtain a view behind the palate, the patient's confidence may be secured and the examination completed.

If the patient can be taught to breathe quietly through the nose during the examination, the palate will hang loosely so as to cause no trouble.

Sometimes a view may be secured by directing the patient to sound *n* or *ng*. Frequently a glimpse may be had if the patient will attempt to expire through the nose.

Various palate or uvula hooks have been constructed for the purpose of overcoming the difficulty; but, as has been well stated, the time spent

in teaching the patient to tolerate them is usually more than is necessary to educate the throat to maintain a position which will require no instrument. Time, patience, and frequent practice by the patient at

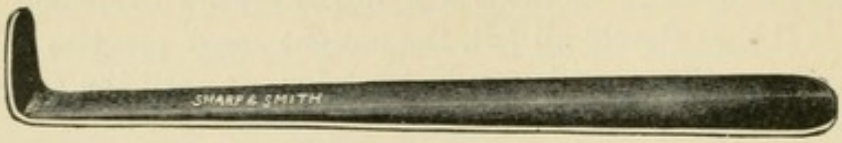

Fig. 82.—Rubber Palate Retractor (½ size).

home must be the main dependence for successful examination in these cases.

When operations are to be performed, the palate may be drawn for-

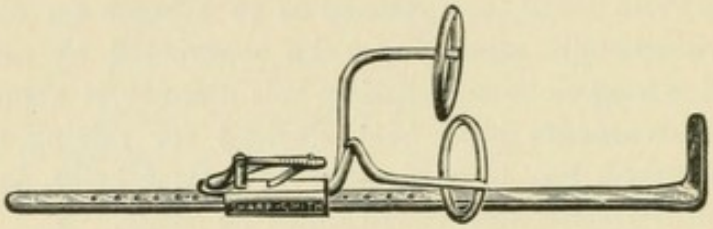

Fig. 83.—Porcher's Self-retaining Uvula and Palate Retractor (½ size.)

ward by the palate retractor (Fig. 82), or by tapes passed through the nares by means of a Bellocq's canula or a catheter, and brought out of the mouth and tied. Soft rubber catheters passed through the nares,

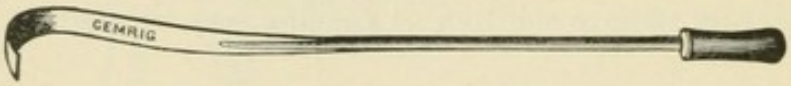

Fig. 84.—Palate Retractor (½ size).

brought out at the mouth, and tied over the lip are very convenient for this purpose; or the palate may be held by means of a broad, strong palate retractor. The palate retractor ordinarily sold (Fig. 84) is only

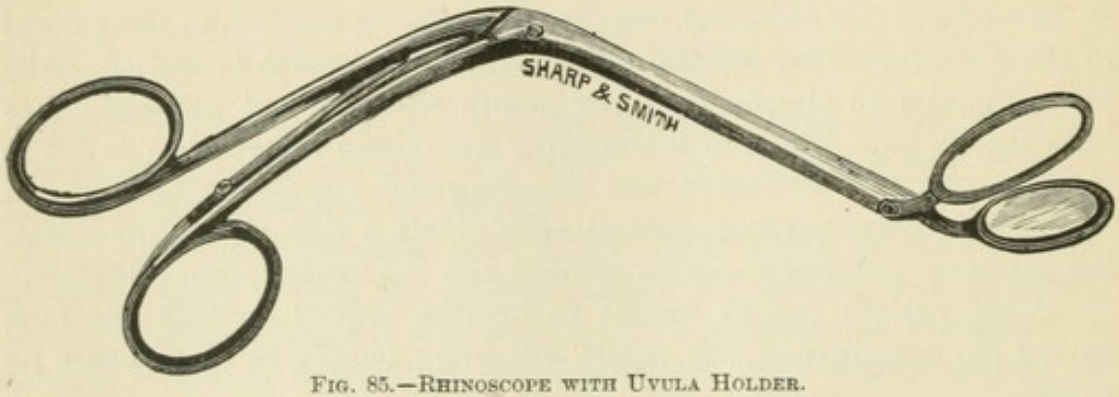

Fig. 85.—Rhinoscope with Uvula Holder.

two-eighths or three-eighths of an inch in width, and is therefore too small for this purpose. Combinations of mirrors and uvula holders have been constructed, but they do not give general satisfaction.

VAULT OF THE PHARYNX AND POSTERIOR NASAL CAVITIES.

On account of the small size of the mirror which we are generally obliged to use, and the limited space through which the rays of light can be reflected, it is impossible to obtain a complete image of the posterior region with the mirror in any single position, but by slowly turning it from side to side, elevating or depressing the handle, and introducing the mirror first on one side of the uvula and then the other, part after part can be brought into view.

The natural condition of these parts should be thoroughly studied from diagrams or models, before an attempt is made to inspect them in the living subject, and the student should make himself perfectly familiar with the description of different parts. When the mirror is first carried into the throat, we usually see in it the image of the upper sur-

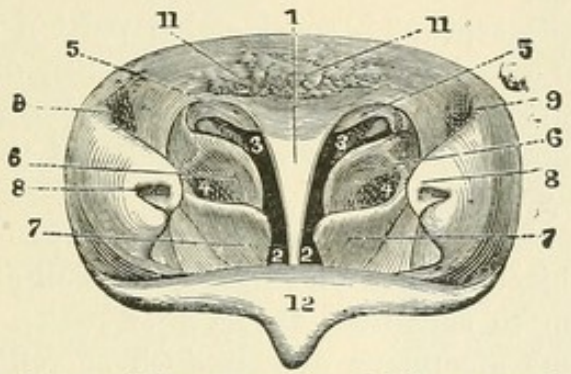

FIG. 86.—RHINOSCOPIC IMAGE. 1, Vomer or septum ; 2, 2, free space of nasal passages ; 3, 3, superior meatus ; 4, 4, middle meatus; 5, 5, superior turbinated body; 6, 6, middle turbinated body ; 7, 7, inferior turbinated body ; 8, 8, pharyngeal orifice of Eustachian tube ; 9, 9, upper portion of fossa of Rosenmueller ; 11, 11, glandular tissue at the anterior portion of the vault of the pharynx ; 12, posterior surface of velum palati (Cohen).

face of the palate, or of the posterior surface of the uvula, or of the posterior wall of the pharynx. If either of the first two is brought into view, we then elevate the handle of the mirror, or if the last is seen we depress it, and thus bring into the field of vision the parts just above the soft palate. We then search for the septum narium, which is to be taken as a starting point for further inspection.

Having found the septum, we trace it throughout its entire vertical length from the narrow lower extremity, where it joins the palate, to its upper broad base which arches outward on either side at the top of the posterior nares. On either side of the septum the irregular outer border of the posterior opening of the nasal cavity should be traced from above downward past the projecting turbinated bodies to the orifice of the Eustachian tube, and finally to the palate and lateral walls of the pharynx. The middle turbinated body is the most prominent object at the outer part of the nasal opening ; but it seems overlapped at its lower part by the inferior turbinated body.

External to the *middle* turbinated body, and just above that portion

of the *inferior* turbinated body which seems to overlap it, is a dark space known as the middle meatus; and slightly external to the latter is the orifice of the Eustachian tube.

Some physicians, instead of following this course in their inspection, prefer to start from the Eustachian tube, but this is merely a matter of habit.

THE SEPTUM NARIUM divides the rhinoscopic view into halves. It forms a narrow, shining column below, near the palate, which gradually increases in breadth toward its upper part. At the lower part it appears of a pinkish, yellowish, or whitish color, according to the brilliancy of the illumination; but toward the upper part or base the color deepens to a red like that of the surrounding mucous membrane.

The color of the parts, as here described, is that observed by means of artificial light. Natural light gives a paler hue.

The sides of the septum, a considerable portion of which may be seen, are usually of a drab or ashy-red color, slightly darker in hue than the posterior edge, probably on account of being less perfectly lighted. The septum seldom occupies exactly the centre of the posterior nares, but inclines slightly to one side.

THE MIDDLE TURBINATED BODIES are easily found, as they are the most prominent objects in view on the external wall of the nasal cavity, of which they seem to constitute the greater part. They are covered with a thin mucous membrane of a pinkish or yellowish white color. The middle turbinated body sometimes resembles a mucous polypus, for which it may be mistaken by the student.

THE INFERIOR TURBINATED BODIES lie just below the preceding. They are considerably smaller than the middle turbinated bodies, and do not approach so near the septum. They are of a darker color, probably from deficient illumination. Not infrequently they have the appearance of solid tumors.

THE EUSTACHIAN ORIFICE on either side is found a little external and posterior to the inferior turbinated body, usually on a level with the middle meatus, but sometimes slightly above or below it.

Thi opening has an irregularly triangular or crescentic shape. It usually measures about a quarter of an inch in its longest diameter, but it is sometimes large enough to admit the tip of the little finger. The opening looks downward, inward, and slightly forward; it is bounded by two more or less prominent projections called the anterior and posterior walls or lips of the orifice, which are covered with a light red or yellowish mucous membrane. The former consists mainly of the fibres of the levator palati muscle, and the latter of the cartilaginous extremity of the Eustachian tube. From the posterior or lower lip a prominent ridge, formed by the levator palati muscle, runs downward and inward to the soft palate. From the anterior or upper lip a dark groove runs upward

and outward toward the vault and the posterior walls of the pharynx. This groove is known as the FOSSA OF ROSENMUELLER or the RECESSUS PHARYNGEI.

THE CHOANÆ or posterior openings of the nares are seen in front of the retro-nasal space. They are of oval form and usually about one-half an inch wide by three-quarters of an inch in height. Harrison Allen (Transactions of the American Laryngological Association, 1888) has shown that they are not infrequently of unequal size, without deviation of the septum, the left being usually the smaller.

THE SUPERIOR TURBINATED BODIES are located at the upper part of the nasal fossæ and cannot be distinctly seen. They have the appear-

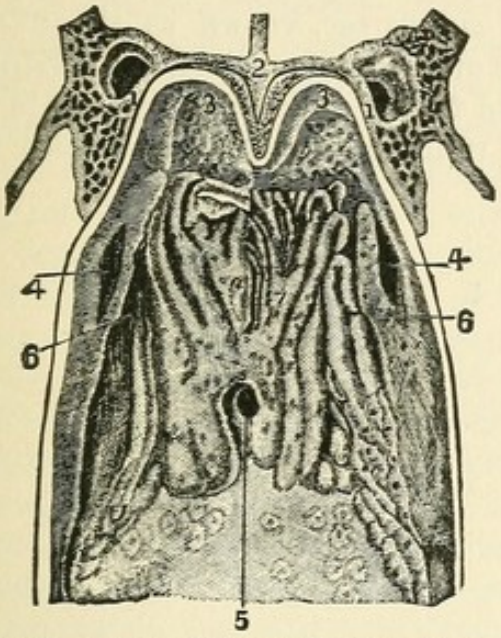

FIG. 87.—ADENOID TISSUE AT VAULT OF PHARYNX. Posterior wall of upper part of pharynx (Luschka). 1, 1, Pterygoid process; 2, section of vomer; 3, 3, posterior portion of the vault of the nasal fossæ; 4, 4, pharyngeal orifice of the Eustachian tube; 5, orifice of the bursa pharyngea; 6, 6, recessus pharyngeus (fossa of Rosenmueller); 7, median folds formed by the adenoid substance of the nasal portion of the pharynx.

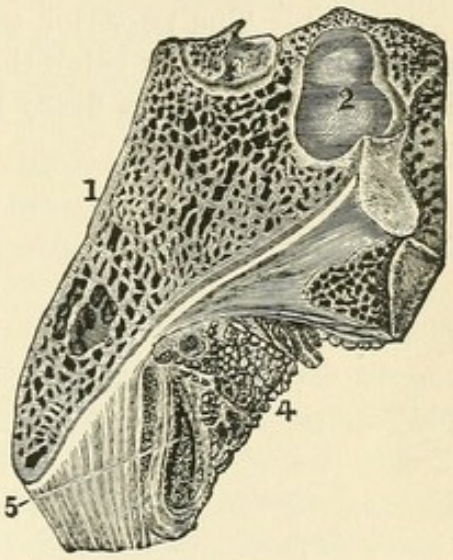

FIG. 88.—PHARYNGEAL BURSA. Antero-posterior section (Luschka). 1, Section of basilar process of the occipital bone; 2, body of sphenoid; 3, pituitary gland; 4, adenoid substance of the vault of the pharynx, behind which is seen 5, the pharyngeal bursa.

ance of narrow triangular projections, the apices of which point downward and inward. Their color is dark red, like that of the base of the septum.

THE SUPERIOR, MIDDLE, AND INFERIOR MEATUS are the spaces found between the turbinated bodies and the external wall of the nasal cavity. The superior meatus, which is the largest, appears as a large shadow at the upper part of the fossa, just below the superior turbinated body. The middle meatus is seen as a dark opening near the middle part of the fossa, external to the middle turbinated body. The inferior meatus, if seen at all, generally appears simply as a dark line.

THE VAULT OF THE PHARYNX is known also as the fornix pharyngis,

and is sometimes spoken of as the tonsilla pharyngea. It is that portion of the pharyngeal wall which begins at the posterior nasal orifices and extends backward along the basilar process of the occipital bone, and then downward to be lost in the posterior pharyngeal wall.

In the perspective view, which we obtain of this part by rhinoscopy, it appears shorter than natural. The mucous membrane is of a light red color, studded with minute whitish follicles, and broken on its surfaces into irregular, more or less longitudinal fissures and ridges, which give it much the appearance of the surface of the faucial tonsil. This appearance of the surface is caused by glandular tissue which has received the name of TONSILLA PHARYNGEA. Near the middle, at the lower part of this glandular tissue, is an opening about the size of a pin's head, which leads up into a small *cul de sac*, known as the BURSA PHARYNGEA. The posterior surface of the uvula, palate, and pillars of the fauces may be seen below the nasal fossae. The palate appears in the rhinoscopic image as a fleshy ledge running at right angles with the septum.

CHAPTER XVIII.

DISEASES OF THE FAUCES.

ACUTE SORE THROAT.

Synonyms.—Erythematous or catarrhal sore throat, cynanche pharyngea, and others.

An acute inflammation may affect the mucous membrane of the palate, pharynx, or tonsils, or all combined. Acute sore throat is found among people of all classes and occurs at all ages, but most frequently in young adults or children. It is said to be more common in those who have suffered from syphilis or who have been mercurialized, and among those who follow sedentary occupations. It is most often observed during the changeable weather of spring or autumn.

Anatomical and Pathological Characteristics.—There is at first simple active hyperæmia of the mucous membrane of the palate, pharynx, or tonsil, either circumscribed or diffused. Later, more or less swelling occurs, generally noticed at first in the uvula. In some cases the mucous membrane lies in thick folds, and occasionally the uvula and posterior pillars of the fauces are œdematous. The superficial blood-vessels are frequently distended, and soon the muscular and glandular tissues become involved, and the secretions, primarily arrested, are again established, but changed both in quantity and quality. In some cases the inflammation may terminate in suppuration.

Etiology.—Acute sore throat is commonly caused by exposure to colds or draughts, especially in subjects who are living under the depressing influence of poor food, bad air, or scanty clothing; it also arises from sitting in warm rooms with heavy wraps, or working in a superheated atmosphere, and then going out into the cold. Among the occasional causes are extension of inflammation from surrounding tissues, the inhalation of poisonous gases, the abuse of tobacco, the inhalation of steam, the taking into the mouth of irritant poisons or of hot fluids, the impaction in the fauces of foreign bodies, and possibly the excessive use of spices. Over-use of the voice in poorly ventilated rooms or in the open air, especially at night, may be an exciting cause. Among the predisposing factors are the syphilitic, rheumatic, and scrofulous diatheses.

Symptomatology.—In mild cases the patient at first suffers simply from malaise, but soon experiences more or less headache and pain in the neck, back, and limbs. In severe cases the pain and constitutional symptoms are marked. Early there is irritation or a sense of itching in the throat, with pricking pain. A few hours later pain becomes severe, especially as the patient attempts to swallow.

When the inflammation is in the upper part of the pharynx, the pain often radiates toward the ears, and there is more or less deafness, due to extension along the Eustachian tubes. If the inflammation is at the inferior portion of the pharynx, the patient suffers from movements of the larynx, which is also sensitive on pressure. In severe cases the skin is hot, the temperature ranging at about 103° F. Indeed, the constitutional symptoms are out of all proportion to the amount of inflammation in the throat. The pulse ranges from 90 to 120 or even 140, according to the extent of inflammation and the susceptibilities of the individual, all the symptoms being more marked in children than in adults. The voice often has a nasal twang, due to swelling of the palate and uvula and to pressure on the pharyngeal and palatine muscles by the inflammatory deposit. There is no hoarseness. Cough does not usually disturb the patient, unless the uvula becomes much elongated. There is, however, an annoying tendency to hawk and clear the throat of the secretions, throughout a considerable portion of the disease. At first there is but little expectoration; later the secretions are more abundant, thick and tenacious, and hard to expectorate; finally they become muco-purulent. The tongue is nearly always furred, the breath is feverish and offensive, the bowels are constipated, and the urine is high colored. Upon examination of the throat, the mucous membrane will be found of a bright red color, which may be limited to patches or diffused over the whole surface. The superficial blood-vessels are often, though not always enlarged; the uvula is usually congested and swollen, and occasionally the same condition extends to the posterior pillars of the fauces. The soft palate may also be considerably swollen, its edges having an œdematous appearance. Whenever œdema occurs, the mucous membrane is somewhat translucent and of a lighter red color. The inflammation may extend over the palate, tonsils, and pharyngeal wall, and sometimes the swelling of the mucous membranes causes large longitudinal welts back of the posterior pillars. Occasionally, in severe cases, the parts are almost livid. The cervical glands are very apt to be slightly enlarged.

Diagnosis.—Acute sore throat is to be distinguished from scarlatina, acute tonsillitis, and rheumatic sore throat. The constitutional symptoms in *scarlatina* are more marked than in acute sore throat, and usually after a few hours a characteristic rash appears upon the skin. There is at first congestion in *acute tonsillitis* and pain similar to that in acute sore throat, but shortly the glands swell sufficiently to distinguish it from the disease under consideration. Again in acute tonsillitis the inflammation is apt to be confined mostly to one side for the first two or three days. The pain is greater in *acute rheumatic sore throat* and the congestion usually, though not invariably, less than in simple acute sore throat, and there is nearly always a rheumatic diathesis or a history of previous attacks, which aid in establishing the diagnosis.

PROGNOSIS.—Acute sore throat runs its course in from seven to ten days, and is not dangerous to life; but often there remains a tendency to frequent recurrence of the attacks. In very rare cases it has proved fatal by extension to the larynx.

TREATMENT.—Patients subject to acute sore throat should be especially cautious about exposure; they should so clothe themselves as not to feel sudden changes of temperature; they should not sit in damp or overheated rooms, and, in a word, should avoid all the known causes of the affection. The cold sponge bath is of undoubted efficacy in preventing the taking of colds. I direct patients to sponge the trunk once a day with cold water as it comes from the hydrant, either morning or evening as best suits their convenience or inclination. For the rugged, the morning sponge bath is, as a rule, better, but for others I advise sponging at night in a warm room. The bath should be taken quickly, and the skin rubbed vigorously with a coarse towel to establish reaction. Full doses of quinine will sometimes abort an attack of acute sore throat. For this purpose, from six to ten grains should be given in a single dose, according to the peculiarities of the individual. Early in the attack, ice sucked continuously or applied about the neck in a rubber bag will frequently abort the inflammation. If the disease is not checked by these means, I advise small doses of opium, aconite, or belladonna, administered as follows: the tincture of opium, one minim every ten to thirty minutes at first, and less frequently as the patient experiences relief from the throat symptoms; or the tincture of aconite, one minim every fifteen to thirty minutes for three or four hours until perspiration is established, when the throat symptoms are generally relieved; subsequently once in one or two hours according to the fever; tincture of belladonna is given in similar doses with benefit in certain cases. I often rely upon potassium bromide alone, or with small doses of opium when the latter is well borne. The bromide is given in doses of ten or fifteen grains every three or four hours, according to the amount of pain. As the disease often occurs in persons of a rheumatic diathesis, and since it is sometimes impossible to determine whether or not the rheumatic diathesis exists, a good practice is to alternate potassium bromide with sodium salicylate in doses of seven and one-half grains or more every third hour. If the disease progresses, inhalations, from a steam atomizer, of solutions of the aqueous extract of opium, or of belladonna gr. i. to ij.; or carbolic acid gr. ij. in four drachms each of glycerin and water, will often be found very soothing. If there be constipation, it is desirable to give a saline cathartic. Some physicians favor a mercurial purge at first, especially in patients with engorgement of the portal system. It should be given in a single dose—for example, calomel gr. v., with sodium bicarbonate gr. v.—and followed after six or eight hours by a saline laxative. In nearly all affections of the throat, potassium chlorate is commonly administered; it is not certain that it has very much influence on these diseases; but

used as a hot gargle in connection with potassium nitrate, I find it often beneficial. I order one part of potassium chlorate to two parts of the nitrate in powder, and direct the patient to use of this a heaping teaspoonful dissolved in half a teacup of water, hot as can be borne, every half-hour or hour according to the severity of the symptoms. Sometimes the act of gargling is very painful. In such cases the patient should simply hold the solution in the throat as long as possible. In the latter part of the disease, astringent gargles of alum or tannic acid are usually recommended, but they are very unpleasant to most patients and do not appear to materially shorten the period of resolution. Astringent troches may be easily taken, and will be found beneficial at this time. For this purpose troches of krameria, each containing three grains of the extract, may be given every two or three hours. Guaiacum is recommended in cases of rheumatic origin, given during the first two or three days every two hours, in troches each containing two or three grains of the resin; or in the ammoniated tincture, best administered in doses of one drachm every three or four hours. I have seen a few cases benefited by it, but ordinarily it has been disappointing. Cocaine has been recommended for the pain, but the practice should be condemned, as an amount sufficient to give relief cannot be applied without producing marked constitutional effects, and the relief will not continue more than fifteen or twenty minutes; if at the end of this time the application is repeated, it is sure to do harm. Demulcents, as, for example, flaxseed tea, infusion of slippery elm bark, or rice water, are useful in allaying inflammation and furnishing some nutrition. The patient will be obliged for a few days to take light diet consisting of soups, broths, beef tea, and milk. If the uvula should become very œdematous, it should be scarified or punctured to allow the serum to escape, but it must not be cut off, lest, when the patient recovers, it be found much shorter than normal. If the patient suffers from heat and burning of the skin, sponging the surface with water, or alcohol and water, will be found very grateful.

ERYSIPELATOUS SORE THROAT.

Erysipelatous sore throat is a rare affection, which, when occurring, is usually associated with facial erysipelas. Cornil (*Archives Générales de Médecine*, 1862) makes three divisions of the disease: first, erysipelas in which there is diffused redness varying from scarlet to deep lividity of the mucous membrane, with more or less swelling and a shining appearance of the surface; second, erysipelas with phlyctænulæ or follicles ranging in size from a pin's head to a centimetre in diameter, similar to those sometimes found upon the skin in erysipelas, which contain serum at first, but soon rupture, the surface becoming coated with a thin, membranous formation; third, erysipelas which eventuates in gan-

grene, characterized by a dark pultaceous appearance of the mucous membrane and an odor peculiar to gangrenous tissue.

Etiology.—This variety of sore throat is produced by the same conditions that cause erysipelas of the face or of other portions of the skin, and is supposed to result from infection by a specific microörganism the streptococcus erysipelatosus. The affection is more frequent during epidemics of erysipelas.

Symptomatology.—In most cases the patient is attacked by facial erysipelas, which continues two or three days before the throat becomes involved. In rare instances, the inflammation starts in the fauces. Preceding its development, the patient usually suffers from malaise for three or four days. Constitutional symptoms are more marked in erysipelas of the throat than in simple facial erysipelas.

Fever ranging from 101° to 104° F. sometimes occurs before congestion is observed either of the throat or skin. Often there is nausea, and pain at the epigastrium. The patient complains of dryness or a stinging pain in the throat with stiffness of the jaws, so that there is difficulty in opening the mouth. Usually there is swelling of the submaxillary and cervical glands. Deglutition becomes exceedingly painful, and is sometimes difficult on account of paresis of the muscles. When the muscles of the palate alone are involved, food will be partially regurgitated through the nose.

Diagnosis.—Upon examination of the throat, in the erythematous variety, the mucous membrane covering the palate, tonsils, and pharynx has a shining surface and bright red color, or in severe cases displays a deep livid hue. In cases marked by phlyctænulæ or gangrene, the appearance of the eruption or the color and odor of the dead tissue would suggest the character of the affection; in those where the throat is attacked first, the speedy occurrence of an eruption upon the skin will clear up the diagnosis. Usually the skin is first attacked, so that when the throat symptoms appear, the nature of the disease is at once suspected.

Prognosis.—The affection may run its course to either recovery or death in two or three days, but in the majority of cases it lasts eight or ten days. One-half of the patients die, and in those who recover resolution is slow. In fatal cases, the disease may extend to the larynx, causing suffocation, or the patient may succumb to blood poisoning or exhaustion, with or without the formation of abscesses. In gangrenous cases, death is almost certain.

Treatment.—In a disease so often fatal, the treatment cannot be very satisfactory, but anything which offers hope should be tried. An application of a sixty grain solution of silver nitrate very early in the attack has seemed to cut it short in some cases. Constant sucking of ice has been found beneficial in moderating the severity of the inflammation, and is to be recommended, at least during the first few hours of the disease. As the patient suffers much from pain and restlessness,

opiates should be administered in sufficient quantity to give relief, unless there is an idiosyncrasy to the contrary. Because of the tendency of the disease to death by exhaustion, stimulants and tonics are indicated. Quinine should be given in doses of two or three grains, averaging about a grain for each hour of the day and night. The tincture of chloride of iron has seemed the best internal remedy for erysipelas of the skin, and is therefore recommended in erysipelatous inflammation of the throat. It should be given in doses of ten or fifteen minims about every two hours, diluted sufficiently to enable the patient to take it without pain; glycerin and syrup of ginger best cover its taste. In cases where applications of cold do not check the inflammation, Mackenzie recommends warm fomentations and inhalations of steam, or steam impregnated with soothing remedies, anodynes, or carbolic acid and glycerin. Hot applications should not be made, however, until we have become convinced that the inflammation cannot be aborted. Frequent gargling with a one per cent solution of carbolic acid is sometimes beneficial. If much œdema of the throat occurs, scarification should be practised to relieve the tension of the tissues; and if the disease extends to the larynx, as it frequently does, tracheotomy must be performed. Unfortunately, however, the operation is usually futile in this affection. In gangrenous cases, antiseptic washes of carbolic acid gr. vi. ad ℥ i., potassium permanganate gr. v. to x. ad ℥ i. or listerine ʒ ii. ad ℥ i. should be frequently used, and we should urge the patient to take freely of alcoholic stimulants and liquid food.

RHEUMATIC SORE THROAT.

ACUTE RHEUMATIC SORE THROAT.

Rheumatic sore throat may be considered as of two varieties, the acute and the chronic. The acute affection is often attended by marked constitutional symptoms and severe pain, and is especially frequent in patients of a rheumatic diathesis.

Anatomical and Pathological Characteristics.—The throat is more or less red and swollen, but usually much less so than in simple acute sore throat, and seldom sufficiently to account for the severe pain.

Etiology.—The disease is produced by the same causes which set up rheumatic inflammation in other parts.

Symptomatology.—There is almost always a rheumatic diathesis, the patient being subject to frequent attacks of muscular rheumatism, or having suffered at some time from the articular affection.

An attack comes on suddenly and is announced by severe pain in the throat, which is soon followed by constitutional symptoms. These usually continue for a couple of days, and then almost as suddenly disappear, the pain shifting from the throat to the muscles of the neck,

back, or extremities. Occasionally the disease passes off with acute articular rheumatism. The pain is so peculiar that patients who have once had the disease will usually recognize it immediately from the character of this symptom. It is very severe upon attempts at swallowing even saliva. Sudden shifting of the pain from the throat to the muscles of the neck or back, about the second day, is one of the notable features of the disease. The temperature is raised two or three degrees and the pulse is correspondingly quickened. Upon examining the fauces, we find more or less redness and swelling, which may be uniform, but often consists simply of red stripes running longitudinally behind the posterior pillars of the fauces upon each side, while other portions of the throat are but very slightly congested; yet the patient suffers intensely.

DIAGNOSIS.—The disease is not likely to be confounded with any other excepting simple *acute sore throat*. The distinguishing features are: the peculiar pain, the history of former attacks, the suddenness with which the attack comes on, and the shifting of the pain after thirty-six or forty hours to some other portion of the body. There is generally much less of redness and swelling than in simple sore throat.

PROGNOSIS.—The affection usually terminates in from two to four days. There is very little danger so far as life is concerned. I know of only one reported fatal case; in that, the disease extended to the larynx.

TREATMENT.—Prophylaxis is of first importance in this affection. Patients subject to it should wear either silk or woollen underclothing the year round, and should be careful to keep the feet dry and warm, and to avoid all undue exposure. Early, an effort should be made to abort the attack by means of salicylates, alkalies, or guaiacum. Sodium salicylate may be given in the manner recommended for acute sore throat, or salicylic acid in capsules or solution, in doses of five or ten grains every one or two hours. After a few doses, the patient usually breaks out in a profuse perspiration, and the pain subsides. When this occurs, the dose should be reduced one-half, and continued in that quantity for five or six doses, when it should be further decreased or substituted by the alkalies. When this remedy is administered in capsules, the patient should always take freely of water with each dose, to avoid irritation of the stomach. Potassium acetate in doses of twenty to thirty grains, or ammoniated tincture of guaiacum in doses of one drachm may be given every fourth hour, or troches of guaiacum may be taken every two hours. On account of the severe pain, anodynes may be required; of these, opiates are most efficient, but the peculiarities of many patients render this drug obnoxious, and therefore potassium bromide, phenacetine or antipyrine or similar substances are often preferable. Applications to the throat of warm fomentations or poultices often have a beneficial effect.

CHRONIC RHEUMATIC SORE THROAT.

Synonym.—Chronic rheumatic laryngitis.

Chronic rheumatic sore throat is a painful affection varying much in severity from time to time and attended by only slight physical changes in the parts involved. Though it usually affects the larynx, and therefore has been described as rheumatic laryngitis, yet in many cases it involves only the fauces, the hyoid bone, or possibly the trachea, without implicating the larynx; therefore the term chronic rheumatic sore throat is preferable. It is comparatively frequent, and has probably existed from time immemorial.

I have been unable to find any description of it prior to that which I gave at the Ninth International Medical Congress, held at Washington, D. C., in 1887.

The affection occurs mainly in the spring and fall, but may also be observed during the winter, and there are occasional cases in which it continues through the summer months. Though affecting all classes with the same impartiality as rheumatism of other parts, it is more frequent in men than in women, and all the cases I have seen have been in adults from twenty to sixty years of age.

Anatomical and Pathological Characteristics.—No very marked characteristics appear, although there is usually slight congestion, circumscribed in character, but changeable.

Etiology.—The disease is due to the same causes as muscular or articular rheumatism.

Symptomatology.—Chronic rheumatic sore throat comes on insidiously in many cases, in others suddenly. Commonly the patient will have been complaining for months when he applies to the laryngologist for relief. Most of the cases I have seen have previously consulted several physicians and have received almost as many different diagnoses, but all have feared either tuberculosis, syphilis, or cancer, most of them having a fixed dread of the latter affection. The general health is not impaired. The patient complains simply of a localized pain, commonly referred to the cornu of the hyoid bone; I have observed it most frequently on the right side. Next in frequency, pain is felt in the larynx, as a rule upon one side only. Occasionally, however, it is in the trachea or tonsils, and sometimes in the side of the base of the tongue. This pain is increased by pressure in nearly all cases, perhaps in all, and it may be increased by phonation or deglutition, but often it completely disappears while the patient is eating. In any case it is liable to shift its position from time to time, but it may persist for weeks in one place. Sometimes the person will complain of sensations of fulness or swelling or of dryness, itching, burning, or an indescribable sensation of discomfort instead of an actual pain. Usually the voice is not affected, yet it is common for these patients to complain of fatigue after speaking a short time. There is no fever, and no quickening of the

pulse except from alarm. Usually there is no cough, but in some cases, especially where the larynx is involved, an annoying, hacking cough is a prominent symptom. The digestive organs may act perfectly, but ordinarily the tongue is more or less covered with a whitish or yellowish white coating, and, although the appetite is usually good, the patient is often troubled with flatus and eructations of gas from the stomach. Upon laryngoscopic examination, we may find congestion, confined generally to a small spot in the region of the pain, and sometimes slight swelling. This condition, however, is liable to diminish, disappear, or change to other localities after a few days, and there is nothing characteristic in the appearance of the parts.

DIAGNOSIS.—The affection is apt to be mistaken for neuralgia, for enlarged glands or enlarged veins at the base of the tongue, for chronic follicular tonsillitis, glossitis, or pharyngitis, for gouty syphilitic or tubercular sore throat, for tobacco sore throat, or for cancer. The essential points in the diagnosis are the uncomfortable sensations of pain, which change usually with changes in the weather, the existence of the rheumatic diathesis, and the absence of any distinct physical signs.

Chronic rheumatic sore throat is to be diagnosticated from varicose veins, enlarged glands at the base of the tongue, and from chronic follicular tonsillitis, glossitis or pharyngitis, all of which sometimes present similar symptoms, by a careful inspection of the parts, by the course of the disease, and by the results of treatment. By inspection, we may at once ascertain whether the veins or glands at the base of the tongue are enlarged, but unfortunately we cannot tell whether enlargement of the glands or a varicose condition of the veins is the cause of the symptoms. Some persons have these conditions and yet suffer no inconvenience whatever, while in others serious discomfort arises. Therefore, if we find varicose veins or enlarged glands at the base of the tongue, with evidence of what seems rheumatic pain in this locality, these conditions must be remedied before we can be certain they are not the cause of the trouble.

If careful inquiry reveals evidence of a rheumatic diathesis, it favors the diagnosis of rheumatic sore throat. The signs upon inspection in *chronic follicular tonsillitis, glossitis,* and *pharyngitis* are characteristic, and when they are found we may usually take it for granted that the symptoms of which the patient complains are due to these diseases. We might possibly be mistaken in cases of this sort, but, if so, a failure to relieve the symptoms by curing these conditions would soon clear up the diagnosis. Sometimes the diagnosis is extremely difficult; but in the majority of cases, having inquired carefully into the history and excluded the affections here mentioned, we may come to an accurate conclusion. *Gouty affections* of the throat as shown by S. Solis Cohen (paper read at first Pan-American Congress) cause painful symptoms similar to the rheumatic affection. They may be distinguished from the

latter by the antecedent history and by the presence of gouty nodules and enlargement of the joints. The affection may be distinguished from *syphilis* by the history and by the physical signs. In the early period of syphilis, and in the secondary and tertiary stages, there are usually characteristic physical signs which are not found in chronic rheumatic sore throat. Cases of syphilitic sore throat occur, however, in which the signs are not characteristic, but in these I have never known the patient to complain of the persistent pain or discomfort which characterizes the rheumatic affection, and I have seen no reason for confounding the two diseases.

We may distinguish this sore throat from *tuberculosis* by the absence of constitutional symptoms in the rheumatic affection, and their great prominence in the tubercular disease; the relatively moderate pain or discomfort and the absence of ulceration in the former and in the latter the severe pain, with superficial ulceration, which may extend over a considerable part of the painful region, or occasionally deep ulceration.

Chronic rheumatic sore throat may be distinguished from *tobacco sore throat* by the history, and the absence of *plaques* which appear very much as if the surface had been brushed over with silver nitrate; these are common in tobacco sore throat, though in some cases we find no physical signs. With tobacco sore throat the patient commonly complains of a burning sensation in the part, usually relieved soon after the tobacco is discontinued. If we find the patient a habitual user of tobacco, if stopping its use relieves his discomfort, and if there are no symptoms of rheumatism in other parts of the body, there will be no difficulty in differentiating the disorders.

It is often difficult to distinguish rheumatic sore throat from *neuralgia*. The presence of slight congestion or swelling is of considerable value in the diagnosis, for in neuralgia there are no local signs. In most cases of rheumatic sore throat, pressure increases the pain, while in neuralgia it does not increase but may relieve it. In rheumatic sore throat, changes of the weather from fair or clear to cloudy and damp almost always aggravate the symptoms, while in neuralgia they have but little effect. In neuralgia the pain is commonly worse in the latter part of the day, when the patient is fatigued; in rheumatic sore throat it is apt to be worse in the morning, and is not particularly increased by fatigue.

The physical signs distinguish *cancer*. In most cases of cancer that I have seen, there have been in the early stage more or less induration, with gradually increasing, irregular swelling, and finally deep ulceration. These do not occur in rheumatic sore throat. In cancer, patients are not likely to suffer pain for any length of time before some of these physical changes occur; in the rheumatic trouble, pain is the essential symptom, and the physical changes are not marked.

PROGNOSIS.—We may expect the cases to continue for several months, or even for years. There is no danger so far as life is concerned.

TREATMENT.—In the treatment, our first attention should be directed to prophylaxis. With this in view, the patient must be well clothed and housed, and protected from undue exposure. Rheumatic patients should wear either woollen or silk next the body both night and day throughout the year—light in summer and heavy in winter. They should be careful that all the excretory organs perform their functions properly. They should eat sparingly of albuminous substances and live largely on vegetables and fruit; the vegetable acids are often advantageous, but, whatever is eaten, it is especially important that digestion be perfect, so that the formation of ptomaines shall be reduced to a minimum. For the local treatment, sedative or stimulant applications may be made, with almost equal chances of relief. Applications of the tincture of aconite to the painful spot four or five times a day, of morphine in solution or in powder will sometimes give considerable relief. I have frequently observed much benefit from the application of such stimulants as zinc sulphate or chloride and copper sulphate, in solution; but I have derived most benefit from a solution of morphine gr. iv., carbolic acid and tannic acid āā gr. xxx., in glycerin and water āā ʒ iv. It is applied by spray, and is frequently given to the patient in one-half this strength to be used at home. In some cases swabbing the surface with strong tincture of iodine or a sixty-grain solution of silver nitrate has proved beneficial. These latter applications apparently act much the same as blisters over rheumatic joints. The most important part of treatment is the internal medication. Here salol, sodium salicylate, potassium iodide, guaiacum, phytolacca, and the oil of gaultherium, one or all may be used at different times with benefit; sodium salicylate may be given in doses of seven to ten grains, the oil of gaultheria in doses of fifteen minims, the ammoniated tincture of guaiacum in doses of a teaspoonful administered in milk three or four times a day. The resin of guaiac in lozenges frequently repeated is of considerable value. I have observed most benefit from the extract of phytolacca and salol combined, āā gr. iij. to iv., with an occasional laxative; but sometimes they have been used conjointly with potassium iodide, or with potassium bromide for its sedative effects. I occasionally give the salol in doses of ten grains. Tinctures of bryonia and of cimicifuga are said to be valuable remedies in rheumatism. I have used them both, with apparently slight benefit in some instances, but the obstinate cases have done better under phytolacca and salol with occasional use of the other remedies already suggested

SORE THROAT OF SMALL-POX.

Sore throat of small-pox is characterized by an eruption similar to that which occurs upon the skin. In many cases it appears before the cutaneous eruption, in others not until the third or sixth day of the original

disease. The extent of the eruption will vary according to the severity of the variola.

Anatomical and Pathological Characteristics.—The mucous membrane is swollen, and the peculiar pustules are found, but without the contracted, depressed centre that is seen upon the skin, because the covering cannot become dry. The ulceration of these pustules frequently extends entirely through the mucous membrane to the muscular tissue, which is more or less involved in the inflammatory action. It is probably on this account that patients experience such severe pain in deglutition.

Diagnosis.—The diagnosis rests upon that of the constitutional disease.

Prognosis.—The throat affection *per se* is not dangerous; in serious cases of variola there are liable to be grave complications in the throat.

Treatment.—Locally, weak astringents and soothing gargles are recommended.

SORE THROAT OF MEASLES.

An eruption in the throat is present in nearly every case of measles as one of the first indications of the disease, but it generally disappears in a few days. It is usually a simple catarrhal inflammation of the mucous membrane, which may extend from the nostrils to the ultimate bronchial tubes. In comparatively rare cases there is a diphtheritic deposit.

Symptomatology.—On examination of the fauces, often one or two days before the disease becomes well marked, several small red points are noticed on the palate, pillars of the fauces, or the pharyngeal wall. At the time the eruption appears upon the skin, we nearly always find much congestion of the throat. In diphtheritic cases there is a fibrinous deposit upon the surface. In some instances the inflammation extends deeply into the tissues, and abscesses result. Many cases of measles are attended by hoarseness due to laryngitis, which sometimes becomes a serious complication, particularly where there is a fibrinous deposit. The inflammation and pain often extend to the ears.

Diagnosis.—The diagnosis will depend upon the cutaneous eruption and the other symptoms distinguishing measles from other diseases.

Prognosis.—So far as the throat is concerned, we expect the catarrhal inflammation to last seven or eight days in the majority of cases and to terminate in resolution. Where fibrinous deposit occurs, the prognosis is grave, especially if it extends to the larynx; of these cases eighty per cent die. In infancy there is peculiar danger from extension of the inflammation to the lungs.

Treatment.—The treatment for acute sore throat is appropriate, but often no treatment is necessary except that which may be indicated for the constitutional disease.

SORE THROAT OF SCARLET FEVER.

Sore throat of scarlet fever is characterized by congestion of the palate and fauces, which occurs early in the attack and is present in nearly every case, even in those where the cutaneous eruption is absent or slight.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—In some instances the congestion is slight, in others the parts are of a deep red or livid hue, and in anginose cases there is much swelling, and the palate, pharynx, and tonsils are all involved in the inflammation and the œdema. If the process is intense, the swelling may cause almost complete closure of the throat. The inflammation frequently extends to the submucous tissues, resulting in extensive suppuration, and not infrequently abscesses occur in other portions of the body. In a large number of cases the inflammation extends along the Eustachian tube to the middle ear, not infrequently resulting in permanent deafness. In some cases there is diphtheritic deposit, but it has not been determined whether this is a peculiar phase of the scarlatina or whether it is an association of the two diseases.

SYMPTOMATOLOGY.—The attack is usually ushered in by vomiting and fever, and the patient complains of more or less stiffness of the jaws and aching pain in the throat, which in scarlatina anginosa may be very severe. The tonsils and mucous membrane are swollen, and the glands at the angles of the jaws are often considerably enlarged. In many cases, in the beginning of the attack, the temperature rises to 105° F., and occasionally even to 106°. It usually continues high several days, and is not apt to disappear before the ninth or tenth day. In severe cases, with much swelling, respiration may be seriously obstructed. The tongue at first has a peculiar strawberry like appearance, due to prominence of the red papillæ, which are surrounded by a white coating, but later it is red and glazed. The breath is offensive, particularly in diphtheritic cases, and in scarlatina anginosa. Disturbance of the stomach, difficulty in deglutition, and loss of appetite are among the common symptoms. The degree of redness and swelling varies much. In simple cases there is a bright scarlet appearance of the throat, sometimes approaching a livid hue, and there may be very little swelling, but in the anginose variety the mucous membrane and tonsils are so much swollen as nearly to close the fauces. In many cases, during the first or second day a thin pseudo-membranous deposit occurs upon the inflamed tissues, and in some this becomes thicker and darker in color and finally acquires the appearance of the membrane in diphtheria. Occasionally in the beginning the symptoms and signs are those of tonsillitis only.

DIAGNOSIS.—The disease is to be distinguished from acute sore throat, from tonsillitis, and from diphtheria. The essential points in the diagnosis are the history and characteristic eruption of scarlet fever.

The appearances are much the same in *acute sore throat* as in scarlatina during the first two or three days, but the constitutional symptoms are usually lighter and the subsequent history different.

There is apt to be more swelling in *tonsillitis*, which is often confined to one side, and there is no cutaneous eruption excepting in rare instances.

A thick false membrane occurs early in *diphtheria*, while the temperature is comparatively low (101° to 102° F.), and other constitutional symptoms are not severe; in scarlatina there is high fever at first, with little, if any, fibrinous deposit; and thick pseudo-membrane, if developed at all, does not often occur until late in the disease.

PROGNOSIS.—In mild cases the throat symptoms usually disappear in from six to ten days, but in scarlatina anginosa or in malignant cases the throat may not be involved until the eighth or ninth day, but then extensive swelling takes place in the course of a few hours, and in a short time extensive pseudo-membranous deposits may occur. In simple cases there is no danger so far as the throat is concerned; twenty-five per cent of the anginose cases die, and of diphtheritic cases fifty per cent are fatal.

TREATMENT.—Emollient applications and antiseptic gargles or sprays are usually recommended. Solutions of carbolic acid gr. v. to viij. ad ℥ i. of glycerin and water, weak solutions of potassium permanganate gr. v. to x. ad ℥ i., or some of the other antiseptics may be employed for this purpose. As the patient progresses toward recovery, the ferruginous and bitter tonics will be found beneficial. If there is much depression, alcoholic stimulants are indicated, and should be given freely. Potassium chlorate has been recommended highly in the treatment of the throat affection of scarlatina, in quantities proportionate to the age of the patient: for an adult, gr. xl. to lx. daily in divided doses. It should be promptly discontinued if it causes irritation of the kidneys.

SIMPLE MEMBRANOUS SORE THROAT.

Synonyms.—Herpetic sore throat, aphthous sore throat.

This is a form of sore throat characterized by the occurrence of small blisters and herpetic patches in the fauces and on the pharynx, which, after a short time, rupture, and the surface becomes covered with an inflammatory deposit or false membrane similar to the membrane in diphtheria, though less dense and much more friable. The affection occurs most frequently in damp climates and in the colder months of the year, particularly when there are sudden changes, as in the spring or fall. It is more frequent in women and children than in men, and is observed oftenest among those who are naturally delicate. It occurs frequently during epidemics of diphtheria, and is occasionally associated with tuberculosis or syphilis.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—In the begin-

ning of the attack there are found several small distended follicles, about the size of a pin's head, with more or less reddening and tumefaction of the surrounding mucous membrane. These may occur singly or in patches, and may terminate in one of three ways: first, by resorption, in which case they may disappear in two or three days and the mucous membrane may be left in a healthy condition; second, they may burst and small deep ulcers may remain, which may either heal rapidly in twenty-four to forty-eight hours, or may become covered with membranous deposit; third, several of these ulcers may coalesce, forming a large patch which becomes covered over with false membrane. I have frequently seen, in the beginning of such an attack, patches five to ten millimetres in diameter, covered with this false membrane, which to all appearances, were not preceded by the small inflamed follicles.

ETIOLOGY.—The disease is attributed to exposure and to certain miasmatic influences not well understood. In occasional cases occurring at the menstrual period it is attributed to uterine disturbances. Certain epidemic influences appear to favor the disease, for it is more frequent when diphtheria is prevalent.

SYMPTOMATOLOGY.—The attack usually comes on with a slight chill, followed by fever and attended by severe pain in the throat. For the first day or two the patient complains only of the symptoms of simple acute sore throat. Usually there is first a sensation of dryness, and after a short time a severe burning or smarting pain, which, so far as we can judge from the patient's description, is more intense than that of any other acute affection of the throat. This pain sometimes radiates toward the ears, and is said to extend occasionally to the nasal cavities, and in rare instances to the larynx. Nearly always we find a herpetic eruption upon the lips at some time during the course of the disease. The fever is occasionally very high for a few days; in other instances there is but very little elevation of temperature. The pulse is accelerated; the tongue is usually flabby, indented at the edges by the teeth and covered with a thick, white fur; there is great difficulty in swallowing, because of the pain, which, however, varies with the location of the diseased follicles or patches. Upon inspecting the parts, we find small inflamed follicles or pustules, often not more than two or three in number, on the palate, fauces, or the side of the mouth; or in place of these small ulcers, or ulcers covered with false membrane; sometimes the pustules and ulcers are found together, because the inflamed follicles come out in successive groups for four or five days. Often early in the attack there is general redness of the parts with localized patches of deeper congestion, which may appear before the pustules are developed. In the majority of cases, the most pronounced physical sign will be the presence of one or more patches, round or oval in form, usually from five to ten millimetres in diameter but sometimes a little larger, and covered by a thin yellowish white false membrane which can be readily removed with a swab

of cotton. These are found on the side of the tongue, fauces, or inner surface of the cheeks, and sometimes even upon the lips. Under this membrane we may find an irritated and easily bleeding surface. In some instances, on removing it we find the mucous membrane beneath in a perfectly healthy condition. Occasionally early in the attack there is a thin membrane spread over the tonsils, with very little erosion. During the attack false membrane will sometimes form upon sores in other parts of the body. Usually the disease is more pronounced upon one side only, but it may spread over both sides and the pharynx, although it seldom or never extends forward upon the hard palate. The membrane is not apt to be continuous like that of diphtheria, but occurs in scattered patches.

Diagnosis.—The disease is liable to be mistaken for diphtheria only. Late in the attack it may sometimes be distinguished from diphtheria by the slight constitutional symptoms; though often there is high fever in the beginning of the attack. In simple membranous sore throat, herpes appears upon the lips during the first three or four days; not so in diphtheria. The membrane, in membranous sore throat, is superficial and thin, about one millimetre in thickness, and it may be easily detached, leaving beneath simply an excoriated, congested, or sometimes healthy surface. In diphtheria the membrane is three or four millimetres in thickness, is detached with difficulty if at all, seeming to extend into the original tissues and be a part of them, and leaves an irregular and deeply ulcerated surface. Membranous sore throat is occasionally followed by paralysis, leading one to question the accuracy of the diagnosis. In some cases the symptoms and signs are clearly those of membranous sore throat, but after a few days diphtheria becomes implanted upon it, giving all the characteristics of the latter disease. Some authors believe these affections identical, but the weight of authority is against this view.

Prognosis.—The disease may be expected to terminate in recovery in from eight to ten days; there is sometimes, however, a tendency to recurrence. We may assure the friends that there is no danger from the disease alone, but it is well to warn them of the possibility that diphtheria may become implanted upon it.

Treatment.—In the treatment of the disease a medium dose of magnesium sulphate or citrate is desirable early. This may be followed by quinine and anodynes to relieve pain. Arsenious acid in small doses has been highly recommended. I have given potassium bromide internally, for its anodyne effects, with benefit, and it is recommended in solution as an inhalation from a steam atomizer. The vapor of compound tincture of benzoin, ʒ i. ad O i. of hot water, is also recommended as an inhalation. Weak antiseptic gargles of potassium permanganate, carbolic acid, listerine, or Dobell's solution are useful to clear the throat of the mucus. Charles E. Sajous recommends that the false membrane be detached and the exposed surface touched every three hours with a

ten grain solution of potassium permanganate (Diseases of the Nose and Throat, 1885). I have derived most benefit from a solution of morphine, tannic acid, and carbolic acid (Form. 139). Applied to the ulcerated surface, this will often give relief for ten or twelve hours. Occasionally solutions of silver nitrate act well, but in some cases I have been unable to find anything that would give much relief. The free use of demulcents, such as rice water, an infusion of slippery elm bark, or flaxseed tea, is soothing to the parts. With these may be combined a little lemon juice if more agreeable to the patient. Potassium chlorate has been highly recommended for this, as it has for nearly every other disease of the throat; but in every instance in which I have given it trial, it has caused intolerable smarting. In cases subject to frequent recurrence of this disease, J. Solis Cohen especially recommends touching the spots with dilute nitric acid. Good diet is to be recommended, and the patient must avoid exposure.

CHAPTER XIX.

DISEASES OF THE FAUCES.—*Continued.*

DIPHTHERIA.

Synonyms.—Diphtheritis, angina diphtheritica, angina membranosa.

Diphtheria is a specific contagious disease, characterized by pronounced constitutional symptoms and inflammation of the mucous membrane of the fauces and upper air passages, with exudation of inflammatory lymph, which rapidly becomes formed into false membrane. It has long been recognized by the best authorities as one of the zymotic fevers. Many English authorities, with whom I am fully in accord, look upon this as a constitutional disease with local manifestations, but many continental authors and some American writers regard it as a primary local affection with secondary constitutional manifestations. The disease occurs sporadically, endemically or epidemically, and appears to have no geographical limitations, but is most frequent in temperate climates. It is most common in cold, damp weather and during the spring or fall months, but is often seen in winter, and not infrequently during warm weather. Lennox Browne states that those who have enlarged tonsils are especially receptive of the contagium (Diseases of the Throat, 2d ed.). The great majority of cases are observed in children under six years, but adults are not exempt. The disease is not often observed twice in the same individual.

Anatomical and Pathological Characteristics.—In the beginning of diphtheria there is congestion of the mucous membrane of the fauces, usually uniform, but occasionally in patches. This may gradually extend to the entire mucous membrane of the throat, and it is soon followed by the exudation of inflammatory lymph, which in most instances proceeds within a few hours to the formation of false membrane. The deposit originates generally in one place and gradually extends to the surrounding tissues, but it may commence in several spots at the same time. It is usually first found upon one or both tonsils, from which it gradually extends, according to the severity of the disease, to the palate, pharynx, naso-pharynx, and other portions of the air passage. Rarely, it is found lining the œsophagus and other parts of the alimentary canal. Wounds upon the skin are liable to become covered by the same process. Extension of the disease to the air passages gives rise to diphtheritic croup, or pulmonary collapse. Blood clots in the ventricles of

the heart or large arteries are not infrequently found in post-mortem examinations. Enlarged lymphatic glands are common, occasionally suppurating, and in the majority of cases the kidneys are congested or actually inflamed. Various bacteria have been found in the diphtheritic membrane, but most or all of these inhabit the mucous membrane of the mouth of healthy individuals.

ETIOLOGY.—The disease is generally conceded to be contagious, and may be communicated from man to the lower animals and *vice versa;* it is believed by most physicians to be due to a specific micro-organism. The researches of T. M. Prudden (*American Journal of Medical Sciences*, April and May, 1889) pointed to a streptococcus as the probable cause of diphtheria, but the results of his later investigations harmonize with those of most bacteriologists, who now attribute the disease to the Klebs-Löffler bacillus. This is a microscopic rod about the length of the tubercle bacillus, but twice its thickness. It is usually more or less bent, with rounded ends, one or both of which may be thickened, giving the club or dumb-bell appearance; it is immobile and contains no spores.

These bacilli do not readily absorb the common aniline stains, but are easily colored by a solution of Löffler's methylin-blue, the coloration often being most intense at the extremities. According to Armand Ruffer (*British Medical Journal*, July 26th, 1890), these bacilli are found most abundantly in the superficial portions of the false membrane, and nearly all experiments go to prove that they do not usually enter the lymphatics or blood vessels; therefore, of itself the bacillus is innocuous, but it produces a virulent ptomaine which is readily absorbed and which may cause the constitutional symptoms of the disease. Numerous clinical observations and experiments, however, have demonstrated with an equal degree of certainty that pseudo-membranous inflammation is often produced independent of the Klebs-Löffler bacillus, as, for example, that resulting from surgical operations in the throat; or from injury inflicted, boiling water, steam, cantharides, chlorine, and ammonia; or the exudative inflammations supposed to be of microbic origin, frequently observed in scarlet fever and measles. This latter variety of inflammation is termed by Smith and Warner (*Annual of the Universal Medical Sciences*, 1891) pseudo-diphtheria, and, as stated by them, can only be distinguished from true diphtheria due to the Klebs-Löffler bacillus by the fact that it is not followed by paralysis and is not attended by a peculiar form of albuminuria unassociated with dropsy or uræmic poisoning. The necessity for assuming that there are two varieties of diphtheria, one produced by the Klebs-Löffler bacillus, the other by other bacteria, seems to justify the statement, that the identity of the specific micro-organism, believed to cause the disease, is as yet uncertain. Roux and Yersin (*L'Union Médicale*, Paris; *Annual of the Universal Medical Sciences*, 1892) report that in the secretions from the mouths of fifty healthy children, living in a village near the

coast, where diphtheria was unknown, they found in 52 per cent a bacillus morphologically identical with the ordinary Klebs-Löffler bacillus and behaving in cultures exactly like the latter, excepting in the number of its colonies. This they believe to be the Klebs-Löffler bacillus in a non-virulent condition.

There can be no doubt that primary simple inflammation favors the production of diphtheria, but it is doubtful whether it is ever in itself capable of producing the disease. Infection may occur from another patient or from articles contaminated by him. Commonly its origin is referred to the use of certain drinking water or milk or the inhalation of emanations from sewers, or from damp, unhealthy cellars or decaying refuse. The most common predisposing cause, I believe, is the exposure of young children to the chilly atmosphere of our houses in the spring and fall months or during the warmer portions of winter, when fires are not considered necessary by adults.

Symptomatology.—After a period of incubation varying from one to eight days, the disease usually commences in young children with well-marked constitutional symptoms, such as headache, drowsiness, more or less fever, thirst, vomiting or diarrhœa, and stiffness of the neck at the angle of the jaw, with more or less soreness of the throat. In older children and adults, the invasion is more gradual. In from twelve to thirty-six hours from the first symptoms, the false membrane can usually be detected in the throat, and in some cases it is deposited in considerable quantities before the person is thought to be ill. The patient usually complains of a sensation of dryness and a desire to hawk and clear the throat, with some pain, especially upon deglutition. Exceptionally an erythematous eruption makes its appearance on the skin during the first few hours of the affection. The pulse is rapid, small, and feeble, and as the disease progresses it may be intermittent. Finally, it grows exceedingly feeble and slower than normal as death from exhaustion approaches. The temperature usually rises to 101° or 102° F. during the first hours of the attack, but with the deposit of false membrane it generally falls and may even become subnormal. After two to four days it may again rise, indicating in favorable cases suppuration and separation of the false membrane, or in others an extension of the disease to the larynx, lungs, kidneys, or other parts. In the later stages of the disease, sudden fall to the subnormal point is a serious symptom indicative of failing strength. The voice is often altered, weak, and hoarse, even before the larynx is affected, but when false membrane has extended to the glottis hoarseness becomes more pronounced or the voice may be entirely lost. With involvement of the larynx, dyspnœa appears, and it may steadily or suddenly increase, aggravated, however, from time to time, by spasms of the glottis. Respiration becomes noisy and stridulous, there is an irritating laryngeal cough, and with the spasms of the glottis all the symptoms of suffoca-

tion appear; the false membrane may be loosened, and fragments of considerable size are often expectorated. Sometimes complete casts of the trachea or bronchi are thrown off in this way. When the disease extends to the naso-pharynx and nostrils, there is obstruction of the nose and a fetid, sanious discharge, frequently accompanied in grave cases by epistaxis. The tongue is coated with thick, yellowish fur, and the breath has a peculiar odor most characteristic of the disease. In malignant cases this odor is so pronounced as to permeate the entire apartment. The tongue is coated from the first, and in unfavorable cases it becomes harsh and dry and covered with a thick, dark coat. The appetite is poor and in severe cases may be entirely lost; nausea and vomiting are not infrequent, particularly when the kidneys are involved. Swelling of the cervical glands occurs in most severe cases, especially at the angles of the jaw; the submaxillary and parotid glands are sometimes involved. The throat is at first deeply congested, but soon the false membrane is deposited, primarily upon one or both tonsils. In the beginning, this membrane is white in color, but it soon becomes yellowish, and with the advance of the disease grayish, brownish, or even almost black. It has the appearance of involving the mucous membrane and being slightly elevated above the surface. If the membrane is exfoliated or forcibly removed, an ulcerated, granular, and bleeding surface remains, which is again soon covered with false membrane. This membrane is firmly adherent to the surface, and cannot be removed by brushing with a swab of cotton, as can the mucus which collects in other forms of sore throat. With the laryngoscope, false membrane may be discovered in the naso-pharynx or the larynx. When the latter becomes obstructed, a sinking in of the softer portions of the chest is noticed with each inspiration, well marked above and below the clavicles, but especially at the lower part of the sternum. As the glottis becomes more and more obstructed, the skin is pallid and bathed in cold perspiration, the lips, ears, and extremities appear blue; the patient grows restless, throwing himself from side to side of the bed every few moments, and with the paroxysms of dyspnœa he throws his arms about and clutches at his throat in the vain effort to obtain more air. As the disease progresses, the signs of carbonic acid poisoning are more and more marked, the patient becomes listless and drowsy, and finally dies in a comatose condition; or he may be suddenly carried off by a spasm of the glottis, a general convulsion, or heart failure.

DIAGNOSIS.—Diphtheria may be confounded with simple catarrhal, or rheumatic pharyngitis; tonsillitis simple or follicular; erysipelas, scarlatina, and other constitutional diseases, or with simple membranous sore throat. The essential points in the diagnosis are the history, the rapid progress of the case, the appearance of firmly adherent whitish or yellowish gray membrane in the throat, and the condition of the urine.

In *catarrhal* or *rheumatic pharyngitis* the temperature is higher, the pain is greater, and there is no formation of false membrane.

In *erysipelas of the throat* the eruption is developed more slowly, and the history is entirely different. *Scarlatina* is developed more rapidly, the temperature rises early to 103° or 105° F. and remains so for several days; in diphtheria it seldom rises higher than 101° or 102° F. in the beginning. In scarlatina, after a short time a characteristic rash appears upon the skin; the appearance of the throat is not greatly different in the commencement, though the congestion is generally more uniform than in diphtheria, and in uncomplicated cases there is no false membrane.

In *tonsillitis* the temperature is much higher, the disease comes on more rapidly, there is more pain in the throat, and usually there is difficulty in opening the mouth which does not occur in diphtheria. In simple tonsillitis there is more swelling, but no deposit of inflammatory lymph. The history of follicular tonsillitis is essentially that of the simple form, but numerous yellowish points or spots appear upon the tonsils at the orifices of the lacunæ. These, however, differ from the appearance of diphtheritic membrane, in that they are more numerous, smaller, are not elevated above the surface of the mucous membrane, are confined to the tonsil in the majority of cases, and never found upon the palate.

Simple membranous sore throat, if seen at the beginning of the attack when the vesicles first appear, is not very likely to be mistaken for diphtheria; but if the patient does not come under observation until two or three days later, the diagnosis may be difficult or even impossible, especially if diphtheria is prevalent at the same time. In most cases of membranous sore throat the patient complains of much more pain and the false membrane is more easily detached and is much thinner than in diphtheria. In some cases a herpetic eruption in the throat and on the lips reveals the true nature of the disease.

In *phlegmonous* or *erysipelatous sore throat* the patient suffers more pain, the temperature is higher, and the tissues are very œdematous and livid, the invasion and course of the disease are different, and diphtheritic membrane is absent.

Prognosis.—The prognosis is always grave, for no matter how mild the case in its commencement, it is impossible to predict what the complications may be before it has run its course; and although the large majority of cases recover, it is never safe to make a favorable prognosis without warning the friends of possible danger. In fatal cases death occasionally occurs within twenty-four hours after the first appearance of the disease, and in the majority the fatal termination is within five days; but in some the struggle for life continues five or six weeks before the patient succumbs. In favorable cases convalescence is usually established about the end of the third week, but especially where complications have existed, the duration may be much longer. As a rule, the younger the patient the greater the danger. Among the symptoms and signs indicative of gravity are deposits of membrane in the nose,

pharynx, or intestines; extreme pain in the ears or throat, purpuric spots on the skin, epistaxis, and other hemorrhages, persistent anorexia, vomiting, diarrhœa, and suppression of the urine. Asthenia, a typhoid condition, or signs of heart failure are often precursors of death. When the larynx is involved, it is probable that without surgical interference the mortality reaches ninety-five per cent, and with it about sixty per cent. Patients not infrequently die suddenly of heart failure, and often the pulse becomes weak and intermittent on the slightest effort, and clearly points to the necessity of relieving the heart from all undue exertion in order to save the patient's life.

As the case progresses toward recovery, the appetite returns, the temperature diminishes, difficulty with respiration disappears, and articulation again is normal; however, the difficulty in swallowing often becomes greater, from exposure of ulcerated surfaces which cause more pain on deglutition, or from paresis of the deglutitory muscles. Not infrequently paralytic symptoms follow the attack closely, about the end of the third week, but, except in cases where the respiratory or circulatory centres are involved, recovery usually occurs, though it may be delayed for several weeks or even months. Owing to danger from the sequelæ, especially heart failure, we can never fully relieve the anxiety of friends until our patient has been well for about three weeks.

TREATMENT.—There are few diseases in which the methods of treatment recommended are more numerous, a fact which is explained by the inutility of a great majority of the means adopted. So much depends upon the nature of the epidemic, the condition of the patient when first attacked, and his surroundings, that it is very difficult to arrive at accurate conclusions regarding the effects of remedies. During the earlier portion of many epidemics a large proportion of those attacked die, and therefore whatever remedies have been used seem to be fruitless; whereas in the latter part of the same epidemic a large majority of the cases recover, no matter what treatment is employed, and the remedies in use at the time get the credit. Many physicians have favorite prescriptions, on which they place great reliance until called upon to treat serious cases; then, unfortunately, all methods often fail and the physician comes to believe that little can be accomplished by treatment. The methods to be adopted are: first, prophylactic; second, dietetic; third, local; fourth, internal or general; fifth, operative.

Prophylaxis is of prime importance in relation to diphtheria. The most useful measures consist of thorough ventilation and proper drainage, pure water supply, proper clothing, and proper heating of living apartments, and as far as possible protection especially of children, from the contagium. It must be remembered that sometimes the specific poison may be carried from one to another by domestic animals, or in the clothing, or about the person of one who has been visiting the sick or attending funerals. As the disease is generally prevalent during the cool

and damper portions of the year, when the need of fires is not appreciated by adults, it is of special importance that children be cared for at this time, that they have proper clothing, and that a suitable temperature of the house be maintained. It has appeared to me that during the spring and fall months children are much more liable to catch cold and consequently to have diphtheria, in the house with a temperature of about 65° to 68° F. than when the temperature is even colder. An effort should be made to maintain the temperature of the house as nearly as possible at 70° F., and children should not be allowed to run about in their night clothing morning and evening or to stand about while dressing with the temperature at from 55° to 65° F., as it is liable to be. They need to be carefully protected at night from exposure due to kicking off the bedding. If the disease has made its appearance in a household, other children of the family must be prevented from all intercourse with the patient, and the sick one should be given an airy, comfortable room, which may be freely ventilated without exposing the patient to draughts. Daniel R. Brower, of Chicago, advocates an excellent method of ventilation during an attack of this disease, consisting of changing the patient two or three times a day from one room to another, the vacated room being thoroughly ventilated in the interim. It is a useful precaution to hang over the door of the sick-room sheets kept moistened with carbolic acid to prevent contamination of the air of the house during the necessary opening of the door. The temperature of the sick room should be kept at from 70° to 75° F., and in all cases an abundant supply of fresh air provided. All utensils or clothing used in the room should be disinfected or destroyed, and finally the room should be thoroughly fumigated before it is again used.

Grancher, of Paris (*Revue d'Hygiene et de Police sanitaire*, December, 1890; *Annual Universal Medical Sciences*, 1892), expresses the opinion that in nearly all instances diphtheria is propagated by infected clothing or furniture. He states that in a diphtheritic ward in Paris, among 1,741 admitted were 153 that did not have diphtheria at the time, yet none of them contracted it. The means of prophylaxis employed in this ward were: a metallic screen about the bed; disinfection of articles used by the patient by boiling in about a six per cent solution of sodium carbonate; disinfection of the bedding and clothing by heat, and of the walls and furniture by washing with a solution of mercury bichloride. Attendants and doctors wear blouses that are disinfected by heat daily and wash themselves in a bichloride solution or in a five per cent solution of carbolic acid.

Ice taken frequently in the mouth tends to relieve thirst and reduce congestion. When children will not take this, Lennox Browne (Diseases of the Throat, 2d ed.) recommends the use of frozen milk or frozen beef tea. Of nutritious drinks, milk is the most important; beef tea and the various broths may be given in addition when the child will

take them, and these may be supplemented by rice water or barley water; the latter is sometimes taken more readily if flavored with lemon juice. As soon as the appetite becomes impaired, these liquid nutrients must be given at regular intervals, and in as great a quantity as the patient can be induced to take. To a child ten years of age as much as half a pint of milk or its equivalent should if possible be given, every third hour night and day. Sometimes with children it is necessary to withhold water in order that they may take the liquid nourishment.

Fontaine, acting on the principle that germs cannot exist in acid solutions, recommends frequent drinks or gargles acidulated with citric acid. On the same principle, pineapple juice has lately been highly recommended, particularly by the laity. When patients cannot take food, or when it will not be retained by the stomach, nutritive enemata become necessary; in this case the various preparations of peptonized meat are exceedingly useful.

Alcoholic stimulation is of great importance, and is usually recommended early in the attack, but I doubt its value at this time. The form in which it is administered is of little importance, so long as it is acceptable to the patient; whisky or brandy is most commonly used, but children will generally take much more readily alcohol diluted with two parts of syrup of tolu, given in as much water as desired.

The early continued application of cold externally is often of the greatest service; for this purpose the throat may be fitted with a coil of rubber or metallic tubing through which a current of ice water is kept constantly passing, or the ice bag may be used. When the latter is employed, the ice should be broken into small pieces and changed about once an hour; the bag should not be more than half filled, so that it may be accurately applied to the surface. When the false membrane begins to separate, hot applications have seemed more beneficial than cold, and occasionally, even in the early part of the attack, the patient so seriously objects to the cold that hot applications may be used instead, the effect being much the same providing the application is continuous and as hot as can be borne.

Topical Treatment.—A variety of substances have been used with the hope of removing the false membrane. The simplest of these is steam, applied either with the croup tent or the steam atomizer. This may be impregnated with the time honored lime water, or with various other substances according to the fancy of the physician. There can be no doubt that lime water is capable of dissolving the false membrane when the latter is immersed in it for a sufficient length of time, but probably it has very little influence upon the membrane in the throat. Liquor potassa, one part to four of water, may be used with equally good results. Mackenzie (Diseases of the Throat and Nose) highly recommended lactic acid applied freely with a brush or pledget of lint. He did not so state, but left us to infer that it was applied in full strength. He

classed it as among the most reliable solvents of diphtheritic membrane. Lennox Browne recommends a solution of lactic acid to be applied every two or three hours by the nurse in from one to six parts of water, and to be used pure once or twice a day by the surgeon. Trypsin, papain, and resorcin have all been recommended for their supposed solvent effects. Tannic acid, alum, and sulphur have been used in the form of powder by many physicians, but are of doubtful utility. Various local antiseptic applications are useful when they can be made without too much objection by the patient; but I believe that whatever is used should be so mild as to cause but little pain, otherwise it is apt to do more harm than good. Of these, mercury bichloride, carbolic acid, potassium permanganate, sodium chlorate, glycerole of borax, chloral, and the tincture of iron are most efficient. The first is used in the proportion of 1 to 4,000 of water, or even as strong as 1 to 1,000, but this is too strong for ordinary use. Carbolic acid is used in the strength of from one to five per cent; the latter is especially recommended by Oertel (Ziemssen's Cyclopædia, English translation, Vol. II.). Potassium permanganate may be used in the strength of gr. v. ad ℥ i., the liquor sodæ chloratæ four drachms to ten ounces, or potassium chlorate a saturated solution. Hugh Hemming, of Kimbolton, England, advocates the syrup of chloral, gr. xxv. ad ℥ i., applied every one or two hours. Sulphurous acid properly diluted is also beneficial. Hydrogen peroxide has been highly recommended as a spray either in its full strength (Marchand's) as obtained from the druggist, or diluted according to the degree of smarting produced. Pure alcohol is used by some as a gargle or spray, with apparent advantage. Tincture of myrrh has also been extolled as a local application. Tincture of the chloride of iron may be used either in the form of a spray or by means of a swab.

G. V. Black, of Jacksonville, Ill. (*Dental Review*, March 15th, 1889, p. 128), has shown that the officinal cinnamon water, although harmless to the patient, is one of the most efficacious antiseptics; and Roux and Yersin (*Annales de Gynécologie et d'Obstétrique*, September, 1889; Paris) have demonstrated that the toxicity of cultures of diphtheritic bacilli is greatly diminished by the addition of carbolic acid, borax, or boric acid; I have, therefore, been induced to try as a local application a saturated solution of boric acid in cinnamon water. This is neither painful, unpleasant, nor dangerous, and has seemed to me more efficient than other local remedies which I have employed. Any of these applications may be of more or less value when the patient does not rebel against their use; if a contest becomes necessary every time the remedy is applied, it will probably do more harm than good. The tincture of iron, when administered internally frequently and in comparatively large doses as recommended below, has all of the local influence that is usually necessary, and obviates the necessity of sprays or gargles.

When the diphtheritic process extends to the nose, the nares should be

washed three or four times daily with a saturated solution of boric acid or some mild alkaline wash, which should always be used warm. The washing may often be accomplished by an atomizer. Whenever it is necessary to employ a syringe, the patient should be placed face downward so that the fluid will not run into the throat and cause strangling. After the washing, a powder consisting of iodol, sugar of milk, and papain—equal parts, may be freely blown into the nose.

Internal Treatment.—Physicians generally are agreed that the treatment of diphtheria should be supporting and stimulating from the beginning. With this in view, iron, quinine, strychnine, and alcoholic stimulants have been employed for generations, and they still hold the first place with a majority of the profession. No internal remedy has seemed to be more effective than tincture of the chloride of iron given in frequent and comparatively large doses, amounting to about one minim of the medicine for each year of the child's age administered every one or two hours, according to the severity of the case. I usually combine it with a small quantity of glycerin and sufficient syrup of tolu to make one drachm, and direct the patient to take it without dilution, providing it does not cause smarting. As the throat becomes more sensitive, the remedy is diluted sufficiently to avoid much discomfort. To prevent any irritation of the stomach, it is well for the patient to take a drink of water before the medicine is given, and as much more as desired five minutes afterward. Quinine may be given at the same time, preferably in pills or capsules; otherwise the patient may become so disgusted as to refuse it altogether. Alcoholic stimulants should be given freely when the pulse becomes weak and the vitality diminished. If there is a tendency to heart failure, no remedy is of greater value than nux vomica in some form. Strychnine may be given, but the tincture of nux vomica has seemed to me more effectual, and it should be given in comparatively large doses, sometimes as much as half a minim for each year of the child's age, being required every one or two hours. Within the past few years mercury bichloride has been largely used in the treatment of this disease with apparent success, and other preparations of mercury are recommended by various authors. Pilocarpine is advised by Oertel, who believes that it hastens separation of the membrane but its depressing effect upon the heart is a serious objection to its use. Among other remedies which have received the sanction of good authority are cubebs, copaiba, potassium chlorate, the sulpho-carbolates, sodium and potassium sulphites, salicylic acid, the salicylates, and potassium, sodium and ammonium benzoates. Indeed, there are few remedies of any potency in any disease that have not been tried for this affection, and which have not, for a time at least, received unmerited praise.

When the disease extends to the larynx, remedies calculated to remove the membrane or to prevent spasm of the muscles have been recommended. For this purpose emetics are chiefly employed; among

those in common use are alum, ipecacuanha, tartar emetic, zinc sulphate, copper sulphate, apomorphine, and turpeth mineral. Of these, ipecacuanha and alum are the simplest and safest, though the turpeth mineral is largely employed, and copper sulphate is highly recommended by good authorities. These, however, should only be employed early in the attack. I fully indorse the ancient belief that in this condition mercurials have considerable power in preventing the deposit of membrane, and removing that which has already been formed. I prefer the mild chloride of mercury, administered in doses of about half a grain for each year of the child's age, every one or two hours until it acts upon the bowels. The frequency of the dose is then gradually diminished, and, as soon as dyspnœa has been relieved, the drug is withdrawn. It is surprising how slight its effects are upon the bowels in this condition; a child two years of age will frequently take twenty to forty grains of calomel without serious disturbance of the bowels. I have never seen any ill effects from its use in this way, and I believe it can do no harm. As obstruction of the glottis increases, the lips and finger nails become blue, there is recession of the softer portion of the chest walls during inspiration, with labored and stertorous respiration, and other signs of approaching suffocation. At this time operative measures should not be delayed. The operation to be preferred depends somewhat upon the age of the child and its surroundings. Other things being equal, in children under five years of age, I decidedly prefer intubation by O'Dwyer's method. In older children, intubation is not quite as satisfactory as tracheotomy, still it has been found useful in many cases, particularly where the graver operation will not be permitted; therefore I would advise that it be tried first; it does not preclude the subsequent performance of tracheotomy. These operations are described under the treatment of membranous croup.

CHAPTER XX.

DISEASES OF THE FAUCES.—*Continued.*

ACUTE FOLLICULAR PHARYNGITIS.

Acute follicular pharyngitis is an acute inflammation of the follicles in the mucous membrane of the pharynx, occurring most frequently in cold and damp climates, and in young or middle-aged people. Those suffering from a rheumatic diathesis are peculiarly prone to it.

Anatomical and Pathological Characteristics.—As a result of the inflammation, the mucous follicles become closed and finally distended by their altered secretions, in some cases the distention becoming so great that the follicle is ruptured and a small ulcer results.

Etiology.—The most frequent causes are: exposure to inclemency of the weather; the abuse of tobacco; and excessive use of the voice in badly ventilated rooms or out of doors, especially in the night air. The inhalation of irritating particles of dust or of smoke is an occasional cause.

Symptomatology.—Mild cases begin with malaise, which may last for a few days, the patient complaining in the mean time of some little fever and more or less discomfort in the throat. Early in the attack, the patient usually experiences dryness, smarting, or pricking sensations. In severe cases pain and swelling are excessive and the constitutional symptoms very pronounced, the fever running up several degrees. There is often a slight hacking cough, with expectoration of a small amount of glairy, tenacious mucus. Hoarseness is present in most instances, due to extension of the inflammation to the larynx. Upon examination of the throat, the mucous membrane is found congested; and in patches, corresponding to the follicles, there is swelling and deeper congestion. Several of these swollen follicles may be visible, especially just back of the posterior pillars of the fauces. Some are round, others oval, and all more or less elevated above the surface. Some with yellowish summits look like pustules. At other points where rupture of the follicles and escape of their contents has occurred, small ulcers are visible, and remain for a few days. Where the contents of a follicle are retained for a number of days, they become somewhat cheesy.

Diagnosis.—Acute follicular pharyngitis is apt to be mistaken for simple acute sore throat. The essential points in the differential diagnosis are the round or oval follicles more or less elevated above the surface, accompanied by pustules or small ulcers.

Prognosis.—The disease usually terminates in resolution within a few days. In most cases, however, there is a tendency to recurrence, and the attack may be repeated many times. I have seen one patient who has had an attack every three or four weeks during the last two years. Nearly always there is some disease of the nasal passages or of the naso-pharynx associated with this predisposition to acute follicular pharyngitis.

Treatment.—In cases where the portal circulation is sluggish, the administration of salines and an occasional mercurial cathartic will work much benefit. In lieu of mercurials, the mineral acids, especially hydrochloric, will be found useful as hepatic stimulants. Many of these patients are troubled with poor digestion, which may be best relieved by the use of bitter tonics. Quinine is useful, more especially in ultra-malarial districts, but under ordinary conditions I have found hydrastine muriate and extract of nux vomica more efficient; but whatever bitter tonics are prescribed, the doses should be small. The local treatment, which has the prestige of antiquity, consists of the application of solutions of silver nitrate in strength of from gr. xxx. to cxx. ad ℥ i. It should be made with an absorbent-cotton swab or large brush, saturated with the solution, but not so wet that drops fall from it. The tongue should be depressed as far as possible, and the application made quickly from the lower part of the pharynx upward, by which procedure the whole pharynx can be treated at once. Applications of silver nitrate often cause strangling, even if applied only to the pharynx; they taste badly and cause prolonged smarting if used in strength sufficient to be of value. For these reasons I seldom employ this remedy, and I have an impression that it is of no more therapeutic value than less disagreeable agents. In these cases the astringent and sedative spray containing morphine, carbolic acid and tannic acid (Form. 93) has not been disappointing. In obstinate cases some authors recommend the actual cautery, in the form of a small wire with a little bulbous end, which is heated and touched to the inflamed follicles. This results in a more acute inflammation for a short time, followed by thorough resolution. The galvano-cautery is much more easily applied than the actual cautery, and is to be recommended when needed. In cauterizing, not more than two or at most three small spots should be touched at a time, otherwise too much inflammation will be caused. The cautery is not often needed in acute cases.

CHRONIC FOLLICULAR PHARYNGITIS.

Synonyms.—Granular sore throat, clergyman's sore throat, chronic pharyngitis, sometimes known as hospital sore throat.

The disease is a chronic inflammation of the pharyngeal mucous membrane, the brunt of which is expended upon the follicles. It is

characterized by hypertrophy of the mucous membrane and irregular plastic exudation upon it, occurring in patches, especially about the follicles. It is most marked in damp and chilly climates, occurs most often in those of delicate constitution, and is perhaps the most frequent of all chronic affections of the fauces or throat. Three varieties of the disease have been described: the hypertrophic, the most common; the atrophic, not very frequent; and the exudative, which is rare. Lennox Browne does not recognize an exudative form, but I have seen several well marked cases.

Anatomical and Pathological Characteristics.—In the hypertrophic variety the mucous membrane of the pharynx is studded with swollen follicles varying from two or three to ten or twelve in number. These are red or yellowish red in color, oval or round in shape and elevated one to three millimetres above the surrounding surface. Those of a yellowish red color sometimes appear like small blisters, with gelatinous contents. Often two or three of these follicles are grouped closely together or united; this is much more frequent at the angles of the pharynx just back of the posterior pillars, where they often form long red welts. One or more of the superficial veins are usually enlarged, sometimes to a diameter of one or two millimetres, and they occasionally seem to terminate in the enlarged follicles. Where the inflammation has existed for a long time, it finally results in more or less atrophy. Some of the enlarged follicles may remain, but the mucous membrane between them looks thin and whitish and sometimes seems to be covered with muco-pus; an appearance due to the atrophied whitened tissue shining through the secretions. In the hypertrophic form, the bulk of the enlarged follicles has been found microscopically to be made up of swollen epithelial cells. In the exudative form, yellowish spots will be seen at the mouths of some of the follicles, similar to the yellow spots seen in chronic follicular tonsillitis, due to cheesy secretions from these diseased glands, mingled with viscid mucus.

Etiology.—The disease may be caused by the constant inhalation of vitiated atmosphere, by frequent exposures to cold or damp, and by the use of tobacco—particularly, there is reason to believe, by excessive smoking. Occasionally it seems to have been caused by the inhalation of acrid fumes, as for example, those to which tinsmiths are exposed. Overuse of the voice, particularly in badly ventilated rooms or in the open air, is evidently a frequent cause. The ingestion of spices is possibly an occasional cause of the disease. It has been attributed also to digestive disturbances, with which it is frequently associated. The most common cause is obstruction of the nasal passages by swelling of the turbinated bodies, polypi, and deflection or exostosis of the septum. As a result of such obstruction, normal nasal respiration gives place to mouth-breathing, which by rarefaction of air in the naso-pharynx with each inspiration, finally causes congestion of the throat, and if prolonged terminates in

disease of its mucous membrane. That the affection is hereditary in some instances there can be no doubt. It is claimed that the arthritic, rheumatic, and scrofulous diatheses favor the production of this disease. The frequent recurrence of acute attacks is apparently the cause in some instances. Chronic follicular pharyngitis is sometimes found following one of the eruptive diseases. It is favored by chronic alcoholism, and exposure to prolonged dry heat is a not very uncommon cause. Mental depression, portal congestion, and torpor of the liver may be put down as among the rare causes.

Symptomatology.—Usually there is at first passive congestion, which may run into the chronic condition of inflammation without greatly attracting the patient's attention. The first complaint is liable to be of slight discomfort in the throat, which may be a feeling of simple dryness, or some peculiar sensation, or may amount to actual pain. Patients usually speak of dryness or pricking sensations in the fauces, sometimes of a hair, or lump, or burning pain, which may be continuous or only occur at periods during the day. Pronounced instances of this character are more prone to occur in the exudative variety of the disease. Partial deafness sometimes occurs, and it may even become complete. This is due to an extension of the inflammatory process into and along the Eustachian tubes. The giving way of the voice is usually, however, the first thing which admonishes the patient to seek medical advice. When the voice is used more or less continuously for half or three-quarters of an hour, the person becomes fatigued, and the enunciation is likely to fail. Although hoarseness is not a constant feature, yet nearly all patients are troubled with it to a greater or less extent upon slight exposure or free use of the voice. Short of hoarseness, the expression of the voice will be found feeble or muffled, and the singing voice is generally lost. A few patients may even suffer from complete aphonia as a result of the extension of the disease to the larynx. All the symptoms are variable, and are apt to change in the same patient; they are generally intensified during the cold and changeable seasons, while an improvement occurs in the summer. In nearly all cases, careful investigation will lead to the discovery that there is oral respiration. Many patients, who affirm that they breathe perfectly, will be found to breathe with the mouth open, particularly during the latter portion of the night. The constitutional effects of follicular pharyngitis depend upon the impeded nasal respiration, or upon the digestive disturbances which may be a causative factor of the disease. The frequent hawking attempt to clear the throat is often one of the most noticeable symptoms of this affection, and is due to the uncomfortable sensation produced by the tenacious mucus adhering to the palate or pharynx. In a few cases there is severe cough, particularly in the morning, and mucous pellets are expectorated early in the day, more especially when the disease has extended to the larynx. In some cases there is muco-purulent expectoration, and occasionally the sputum is streaked with blood; this, however,

is of no consequence in the diagnosis or prognosis, though it is often alarming to the patient. In many cases the secretions which form in the naso-pharynx and nose gradually find their way downward and backward into the pharynx, or even into the larynx, and may be seen adhering to the posterior pharyngeal wall as thick, dry or moist scabs, or they may hang in stringy masses from the edge of the palate. There will usually be found a considerable amount of mucus in the naso-pharynx, and some adhering to the mucous membrane of the larynx, where it may cause cough. Commonly there is a coated tongue, together with other evidences of digestive derangement. Where pain is experienced, it may be during the act of swallowing, but in some cases the discomfort may be relieved by deglutition, and not reappear until an hour or so after eating. Liquids are easily swallowed by some patients, but solids cause pain; with others the opposite is true; while to still others neither will cause any discomfort. Upon examination of the throat, the surface (Fig. 89) will be found congested and swollen in patches, the blood vessels in many cases enlarged, and the follicles of abnormal development. About the latter there is usually a narrow zone of congestion. At the base of the tongue diseased follicles similar to those upon the pharyngeal wall may be observed. In the exudative type of the affection, two or three yellowish points similar to those of chronic follicular tonsillitis may be seen at some part of the pharynx. Small ulcers are described by Cohen and others as being present occasionally, though I have never seen them. The tonsils are often involved, in either chronic follicular inflammation or simple hypertrophy. The palate may be relaxed and the uvula elongated; and the larynx is not infrequently the seat of more or less congestion, more particularly the posterior ends of the vocal cords, especially after using the voice. Examination of the naso-pharynx will reveal congestion of its mucous membrane, with, generally, abundant secretion. Often there is submucous thickening at the sides of the vomer, which may appear grayish white and slightly nodular, and is sometimes sufficiently large to almost occlude the posterior nares. Such obstruction may also result from hypertrophy of the posterior ends of the turbinated bodies. When the secretion is scanty and the mucous membrane dry and thin, white atrophied tissue is seen between the follicles —a condition known as *pharyngitis sicca*, or *atrophic follicular pharyngitis*. Sometimes the entire pharyngeal wall will be found covered with dried secretions.

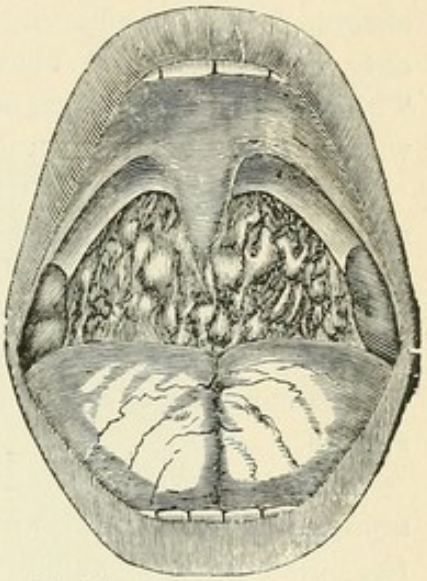

FIG. 89.—CHRONIC FOLLICULAR PHARYNGITIS (COHEN).

Diagnosis.—Syphilis is the only disease with which the affection is likely to be confounded. When there is simple congestion, with very slight enlargement of the follicles, it may be difficult or impossible to distinguish it from some cases of syphilitic sore throat, but in the latter there are usually either the mucous patches of the secondary stage or the ulcers or scars of the tertiary period, the presence of which renders the diagnosis plain. The remote possibility of mistaking the ulcer of chronic follicular pharyngitis—which is very rare—for that of syphilis may be remembered. Chronic follicular pharyngitis may possibly be confounded with *tubercular sore throat*, but in this the ulcers are superficial and irregular, and the edges not distinctly marked; whereas in chronic follicular pharyngitis they occur, if at all, but rarely, and then only as small, round ulcers where distended follicles have ruptured. The presence or absence of the constitutional evidences of tuberculosis will have great weight in determining the true nature of the disease.

Prognosis.—Chronic follicular pharyngitis may continue for years unless efficiently treated. In many cases the inflammation gradually extends to the ear, or to the larynx, giving rise to deafness, or to loss of voice. Again, the hypertrophic form of the disease may terminate in the atrophic, which is far more troublesome to the patient and very difficult to cure. The exudative form of the affection is generally more obstinate.

Treatment.—The old adage that an ounce of prevention is worth a pound of cure could well be applied in this disease, were it not that the opportunity is generally lacking to the physician, inasmuch as the patient does not present himself soon enough. A caution should be given, however, regarding those exposures already mentioned which are known to exert a damaging influence upon the parts, for they not only cause the disease, but favor its continuation. Faulty digestion and elimination should be corrected. In many cases a course of diuretics and bitter tonics is indicated. Arsenious acid is often of special service. Those predisposed to rheumatism must have appropriate constitutional treatment. Locally, silver nitrate is an old time remedy, but one which I rarely recommend. It may be applied in strong solution or in the solid stick, but, if the latter, only a small area should be treated at one sitting. I have had excellent results from powdered hydrastine (Form. 174) by insufflation into the naso-pharynx in cases presenting several enlarged follicles of a deep pink color, providing the surrounding mucous membrane is moist, and the secretion—except in the naso-pharynx—is not excessive. The powder remains in the naso-pharynx several hours, gradually working down the pharynx and thereby prolonging the effect. At first only a small quantity should be used, in order to ascertain the susceptibility of the patient, since in some cases the remedy applied in this way causes severe pain. Ordinarily it produces no discomfort.

In mild cases, and often in those more severe, local astringents are

desirable, and troches of krameria, either simple or compound (Form. 38 and 41), will be most conveniently used by the patient. Sprays to the oro-pharynx of copper sulphate in solution of ten or twenty grains ad ℥ i., zinc chloride or zinc sulphate in the same proportion, or mercury bichloride gr. ss. ad ℥ i. are also useful. Somewhat weaker solutions of the same may be used for the naso-pharynx, which in nearly all instances requires treatment; indeed, it is often more important to medicate the naso-

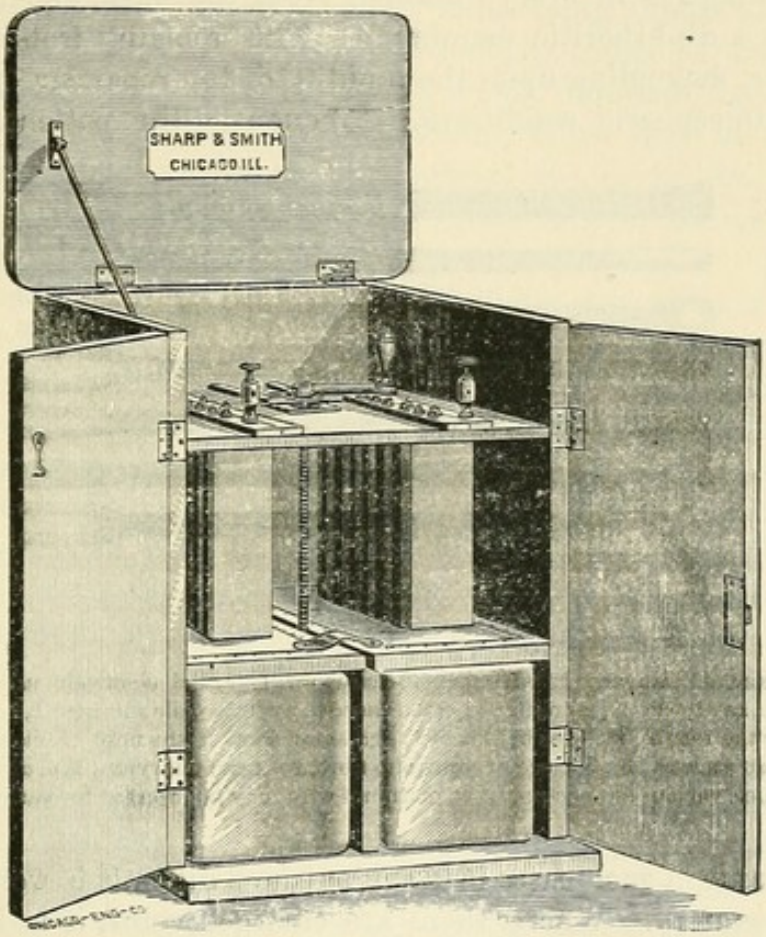

FIG. 90.—INGALS' MODIFICATION OF SHURLY'S BATTERY. This has two large cells. The elements consist of large zinc and carbon plates, which may be depressed to any desired depth by the screw shown in the centre. Thus the current may be accurately regulated. The cautery battery here shown I have used for years with much satisfaction, though for the past two years I have more commonly employed a storage battery so connected that I can easily charge it from the Edison current. It is somewhat more convenient, when working well, than the battery here shown, but more expensive and less reliable.

pharynx than the other parts. When the follicles are much enlarged, the above treatment will not be sufficient, and there will be no great relief until they are cured. To accomplish this, they may be cauterized with nitric acid, chromic acid, or London paste, a small quantity being applied directly to the surface of the follicle, not to the surrounding membrane; only two or three of the follicles should be treated at each sitting. This procedure may be repeated every four or five days until all are removed. Sometimes it is well to split the follicle with a sharp knife, and then crowd into the incision the pointed end of a stick of silver nitrate.

Some are in favor of scraping off these follicles with a curette. The actual cautery may be employed—as recommended for acute follicular pharyngitis—but the galvano-cautery (Figs. 90 and 91) is the best means for getting rid of the hypertrophied follicles. In using it the electrode is applied cold, the current is then turned on for a second and the follicle destroyed. The next day after using the cautery, a whitish pellicle is observed about this cauterized point, which may extend for four or five millimetres in every direction from the burn, and appears very much like a diphtheritic membrane. This remains from five to even twelve days, depending upon the rapidity of the reparative process and, perhaps, atmospheric conditions. Frequently the patients retch and

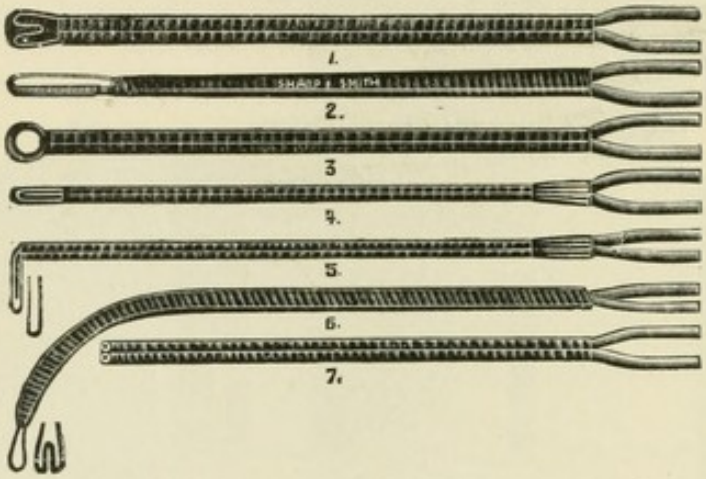

FIG. 91.—INGALS' CAUTERY ELECTRODES (2-5 size). 1, Guarded electrode used for superficial cauterization in hay fever; 2, knife-like electrode used in hypertrophic rhinitis; 3, 4, and 5, electrodes for cauterizing the tonsils, follicles in pharynx, and small spots in the nose; 5, electrode for base of tongue, or, when guarded by a piece of vulcanite fibre, for naso-pharynx; 5, 6, and 7, tubular electrodes, into which various shaped points of platinum wire may be inserted for various purposes.

gag easily, and in such cases it is evident how difficult it would be to use the actual cautery. Where there are enlarged veins, it is better to cut them off with silver nitrate or the galvano-cautery—the latter being much the more satisfactory in its action. Though the exudative form of the disease has been considered peculiarly obstinate, it has, in my experience, proved less stubborn than some other forms, when treated by the galvano-cautery in the manner just described. Cases of simple chronic congestion without enlargement of the follicles are most difficult to cure. In these all sources of irritation must be avoided, and the patient should make applications to the pharynx of some mild astringent two or three times daily. Sometimes such patients will find it necessary to remove to a different climate before relief is found, but ordinarily it is not well to advise such a course, for the climatic influence is very uncertain.

ACUTE FOLLICULAR GLOSSITIS.

Acute follicular glossitis is an inflammation of the follicles at the base of the tongue, in which severe pain is caused by an attempt at deglutition. Its causes are probably not unlike those of acute follicular pharyngitis, and its pathology is also similar.

SYMPTOMATOLOGY.—Pain is felt not only in the throat, but radiating to the ears, and some patients speak of it as being almost altogether in the ears, or near the orifices of the Eustachian tubes. Upon examination of the parts, we may find several small, rounded elevations of a whitish hue somewhat resembling pustules, which may be distributed all over the base of the tongue, or confined to one or the other side, particularly to that portion of the base which is often hidden from view by contact with the external wall.

In some cases, instead of these small follicles, one or more superficial ulcers are to be found. I have seen one at least a centimetre in diameter, where small ulcers had coalesced after rupture of several follicles. These ulcers are more apt to be found at the side of the base of the tongue, where they may escape notice except upon careful inspection.

DIAGNOSIS.—The disease is liable to be mistaken for inflammation in the naso-pharynx, because the patient often refers the pain to that locality. The diagnosis will be made by a careful laryngoscopic inspection of the base of the tongue, particularly of its sides, which must be exposed by crowding the organ over with a spatula.

PROGNOSIS.—Left to itself, the condition lasts a week or ten days.

TREATMENT.—The most satisfactory treatment consists in the application of a sixty grain solution of silver nitrate to the follicles or superficial ulcers. The rapidity with which the affection may be cured by this method is sometimes surprising. I recollect one case especially, where an ulcer a centimetre in diameter was found, in which the pain was relieved within a few minutes after the first application, and in forty-eight hours the ulcer practically healed.

CHRONIC FOLLICULAR GLOSSITIS.

Chronic follicular glossitis is not infrequently associated with chronic tonsillitis, and is characterized by chronic inflammation of the follicles at the base of the tongue, which become more or less filled with secretion producing numerous yellowish white spots similar to diseased follicles in the tonsils, and attended by various uncomfortable sensations referred either to the tonsils or, more accurately, to the base of the tongue. The nature of the affection is essentially the same as that of chronic follicular inflammation of the tonsils, and it is apparently dependent upon like causes.

Symptomatology.—The principal symptoms of which the patient complains are sensations of pricking or of a foreign body in the throat, which may be present continuously or only a part of the time, and which may or may not be aggravated by the act of deglutition.

Diagnosis.—The diagnosis is made by an examination of the base of the tongue with the laryngeal mirror, without which it is seldom possible to see the diseased follicles.

Prognosis.—The affection tends to run on for many months or years, during which time the patient is much annoyed by offensive breath and by harassing fears of tuberculosis or cancer.

Treatment.—When due to a rheumatic diathesis, or to disturbance of the digestive organs, the treatment suited to these disorders is indicated.

Locally, astringent troches as represented by the troches of krameria (Form. 38 and 41) are sometimes beneficial, and applications of more active astringents, of stimulants, or of strong solutions of silver nitrate sometimes prove curative. A more efficient method, and one which finally must be the resort in most cases, is cauterization with the galvanocautery. This is usually followed by the most satisfactory results. Two or three follicles should be cauterized at each sitting, by a small electrode, which should be passed to the bottom of each, and the operation should not be repeated until two or three days after all soreness from the previous cauterization has disappeared. This treatment should be continued until all of the diseased follicles have been dealt with and a complete cure may be confidently predicted.

SCROFULOUS SORE THROAT.

Scrofulous sore throat is a chronic inflammation, sometimes observed in scrofulous children, which in the simple form has the appearance of ordinary catarrhal inflammation; when more pronounced, it resembles the inflammation of tuberculosis or syphilis. In many instances it consists of simple inflammatory thickening of the mucous membrane of the fauces and naso-pharynx or palate, but in the more advanced conditions—which, indeed, are the only ones rightly classed under this head—ulceration occurs. This at first superficial and always indolent, finally becomes extensive, sometimes spreading over a large portion of the pharynx or involving the palate, and causing perforation, or even destruction of the uvula with considerable portions of the velum.

Etiology.—J. Solis Cohen (Diseases of the Throat) believes that most of these are cases of simple chronic inflammation occurring in those of inherited syphilitic taint, while others regard it as a manifestation of lupus. Still others ascribe some of the cases to tuberculosis or the rheumatic or arthritic diathesis. Whatever the remote cause, it is certain that a low form of inflammation, with ulceration, occurs in chil-

dren presenting what was formerly known as the scrofulous diathesis; and it is more than possible that, in most of these, hereditary syphilis or tuberculosis could be traced if an accurate history could be obtained.

SYMPTOMATOLOGY.—There are no positive symptoms or signs of this affection, but usually the child is pale and less vigorous than other children of the same age and surroundings; there is sometimes a tendency to clear the throat of secretions frequently, but usually this is not a pronounced symptom, and even when extensive ulceration has taken place the patient does not complain of pain. Difficulty in deglutition or alteration of the voice may be caused by partial destruction of the soft palate or extensive ulceration of the pharynx. Sometimes a history of inherited syphilis or tuberculosis can be obtained, and upon examination of the fauces more or less extensive ulceration will be found. These ulcers are at first superficial, but later are deep, with bevelled edges, indolent surface, and slight discharge.

DIAGNOSIS.—Scrofulous sore throat is to be distinguished from lupus, tuberculosis, and syphilis.

External manifestations which may at once decide the diagnosis, nearly always attend *lupus*. Upon the base and about the edges of the ulcer are red nodules, which do not appear in the scrofulous ulceration.

Scrofulous sore throat is distinguished from *tuberculosis* by the comparative absence of pain, by a well marked instead of an indistinct border, by the absence of fever and other evidences of tuberculosis.

Scrofulous sore throat is distinguished from *syphilitic* ulceration of the throat by the absence of a syphilitic history and the general signs of the disease, by the age of the patient, slow progress of the ulceration, slight discharge and bevelling of its edges, which do not have the punched-out appearance common in syphilis.

Scrofulous sore throat and lupus of the pharynx present the following points of difference:

SCROFULOUS SORE THROAT.	LUPUS OF THE PHARYNX.
Generally seen in children. Usually evidences of constitutional disturbance.	Generally in young adults. Usually associated with disease of the face.
Ulcers superficial or deep, with bevelled edges, indolent base, and slight discharge; no cicatrices.	Congested, irregular nodules about edges or on base of ulcers, which are usually extending in some places, while healing at some other part of their border; usually old cicatrices.

Scrofulous sore throat and syphilitic sore throat can be differentiated as follows:

SCROFULOUS SORE THROAT.	SYPHILITIC SORE THROAT.
Generally seen in children. Ulcer indolent and usually has a bevelled edge not indurated or undermined.	Generally seen in adults. Ulcer sharp cut, indurated, sometimes undermined.

The differential diagnosis of tubercular sore throat and scrofulous sore throat, will be further considered under the head of acute tubercular sore throat.

Prognosis.—If left to itself, the ulceration gradually extends, and may continue for many months; I have seen cases which had lasted for over a year. With improvement of the general condition and appropriate local treatment, healing may be expected within a short time.

Treatment.—Good hygienic surroundings and tonics are most important. Calcium iodide and chloride internally in moderate doses are beneficial, and cod-liver oil is generally recommended. The local treatment consists of frequent cauterization or stimulation by less active agents. In practice, the thorough application of strong tincture of iodine to the ulcer two or three times a week has given best satisfaction. Under its influence and the general treatment, healing soon begins, and an ulcer an inch in diameter may be expected to heal within six or eight weeks.

ACUTE TUBERCULAR SORE THROAT.

Acute tubercular sore throat is a rare affection occurring in about one per cent of all cases of tuberculosis of the respiratory tract (Browne, Diseases of the Throat, third edition). It runs a rapid course, being characterized by ulceration and great pain and the constitutional symptoms of tuberculosis.

Anatomical and Pathological Characteristics.—At first there appear numerous small, gray granulations grouped in patches beneath the epithelium, and if abundant, closely resembling the mucous patches of syphilis, but they lack the inflammatory areolæ which are found about the latter. These granulations are said to bleed easily when touched, but this has not been my experience. They may be located upon the palate and the pharynx, and late in the disease may be found on the epiglottis and in the larynx. As the affection progresses they lose their transparency, become hidden in a purulent or pultaceous covering, and finally undergo ulceration. These ulcerations are shallow, have no well marked borders, but rather a worm eaten, irregular edge, and bleed easily when touched.

Etiology.—The cause is the same as that of tuberculosis in other localities.

Symptomatology.—Usually there are evidences of primary pulmonary or laryngeal phthisis. The consumptive appearance, persistent fever, rapid pulse, cough with or without expectoration, anorexia, and other symptoms of tuberculosis are apt to be marked, but the pharyngeal lesions may be independent of laryngeal or pulmonary disease, these subsequently supervening. The one prominent, sometimes the first, symptom of tubercular sore throat is intense pain, sometimes experienced upon phonation and upon attempts at deglutition. It becomes agonizing,

largely preventing the taking of food, with consequent speedy loss of strength and rapid advance of the disease. An early examination may reveal congestion of the pharynx similar to that found in simple inflammation, but in most cases the mucous membrane presents a characteristic grayish pallor with numerous semi-transparent granulations which speedily give place to ulceration. The tubercular ulcer is superficial, with irregular ill defined borders, which are not undermined, and it is sometimes surrounded by a faint blush, though usually there is no areola of hyperæmia. The floor presents indolent, gray granulations, and scanty secretions.

In exceptional cases the tubercular ulcer has a sharply defined border, which may be slightly thickened and congested; it has a depth of about one and one-half millimetres, and its base is covered with a grayish white coating presenting an appearance about midway between that of the ordinary superficial ulcer described above and the deep ulceration of syphilis.

DIAGNOSIS.—Tubercular sore throat may be mistaken for syphilitic or scrofulous sore throat.

Syphilitic sore throat is not accompanied by the excessive pain, the fever, and the constitutional symptoms of the tubercular affection; and instead of the marked anæmia of the mucous membrane and small gray granulations, or shallow irregular ulcers with ill defined, pale borders, and scanty, grayish, viscid secretion, it is characterized by the large, sharply defined inflammatory ulcers of the secondary stage, or the deep ulcers of the tertiary form with raised and often undermined edges, granular floor, and profuse purulent secretion. As also noted by Lennox Browne (*op. cit.*), the enlargement of the parotid, submaxillary, and cervical glands, both superficial and deep, so commonly observed in the tubercular affection, is relatively infrequent in the latter part of the secondary, and in the tertiary stage of syphilis.

From syphilitic sore throat, tubercular sore throat may be distinguished as follows:

TUBERCULAR SORE THROAT.	SYPHILITIC SORE THROAT.
No syphilitic history. Generally in adults.	Syphilitic history. If hereditary, it may appear in children; otherwise in adults.
Marked constitutional symptoms.	Constitutional symptoms may be marked.
Fever, rapid emaciation.	Usually no fever.
Severe local pain.	Frequently no pain.
Aphonia, dysphagia.	Hoarseness, but usually no aphonia or dysphagia.
Ulcer usually superficial, with grayish, worm eaten appearance and rapidly progressive.	Ulcer sharp cut, with areola of reddened, thickened tissue about it, sometimes undermined edge.
Short duration.	May progress rapidly but usually relatively longer in duration.

Scrofulous sore throat, unlike the tubercular, occurs in children instead of young adults, and lacks the severe pain, the fever, and the irregular, superficial, poorly defined ulcers of the latter affection.

Between tubercular sore throat and scrofulous sore throat the following are the chief points of difference:

Tubercular sore throat.	Scrofulous sore throat.
Rarely seen in children. Ulcer superficial, with poorly defined borders.	Generally seen in children. Ulcer deep, with sharply defined edges.
Hectic fever. Considerable cough.	No fever. Little or no cough.
Rapid emaciation.	Slow physical change.
Severe pain, frequently the first symptom.	But little or no pain.
Dyspnœa, dysphonia or aphonia, dysphagia.	No dysphonia, aphonia, or dysphagia.
Pulmonary tuberculosis usually present.	No signs of pulmonary tuberculosis.

Prognosis.—Tubercular sore throat usually runs its course in from six to twelve weeks, and nearly always terminates fatally. In exceptional instances the duration is as much as six months, and in extremely rare cases recovery may occur, or the disease may progress slowly, the patient under favorable conditions living for several years before succumbing to the constitutional disease. Death is caused commonly by asthenia.

Treatment.—The treatment recommended by Krause and Herying, by thorough curetting the ulcers, followed by the application of lactic acid, with occasional use of the galvano-cautery, has effected a few cures (Gleitsmann, *New York Medical Journal*, 1891), and similar results have been attained by the use of lactic acid alone in solutions varying in strength from twenty to seventy-five per cent. Sedative applications are of much benefit, chief among which are steam impregnated with belladonna, hyoscyamus, stramonium, or opium, as recommended (Form. 56, 57, and 59). Sajous (Diseases of the Nose and Throat) recommends a ten per cent solution of cocaine applied often enough to relieve pain; but the evil effects of this drug are so pronounced that extreme caution should be used in its employment. Painting the throat with solutions of silver nitrate as advised by some, has usually proven more hurtful than otherwise. I have found most satisfactory, for relieving pain, a spray of morphine, carbolic acid, and tannic acid (Form. 93). This may be used by the patient also, diluted, with one or more parts of water, according to the amount of smarting occasioned. Troches of morphine or lactucarium, or althea (Form. 25, 29, and 36) are sometimes efficient in relieving the distress, but the good effect of opiates is usually counteracted by the excessive dryness which they cause. When dysphagia becomes extreme, the feeding bottle may be used, as recommended by Delavan (Transactions of the Ninth American Laryngological Association) or

nutritive enemata may be employed, but in well marked cases all that we can hope for is to render the patient as comfortable as possible.

SYPHILITIC SORE THROAT.

Syphilis may affect the fauces in any of its three stages, but the earliest manifestation is seldom seen in the throat, though the secondary and tertiary forms are common. The chancre or primary lesion of syphilis, when present in the mouth, is similar to that which may occur in other parts, and lasts for five or six weeks; in the secondary stage the erythematous or mucous patches, and in the tertiary stage gummata or deep ulcers, are characteristic. When the disease is inherited, the secondary symptoms usually occur within two to six weeks after birth; the tertiary, in early childhood or at any time before the sixteenth year.

Anatomical and Pathological Characteristics.—When chancre occurs in the throat, it is nearly always located on one tonsil. In the secondary affection, usually at first the fauces present a uniform dull red erythema; this in part gradually fades away, leaving erythematous patches which tend to symmetrical arrangement upon the two sides of the palate or pillars of the fauces, and sometimes upon the pharyngeal wall. These patches are separated from healthy tissue by a distinct line of demarcation. Mucous patches (also termed mucous tubercles or broad condylomata) when occurring in infants, are usually found in the upper part of the pharynx and on the fauces; but in adults on the pillars of the fauces, or the velum palati and the sides and base of the tongue. They are circular or elliptical in form, slightly elevated, at first of a deep red, later of a grayish white color, and, as a rule, symmetrically situated on each side of the throat. These subsequently become the seat of superficial ulcers; their borders are distinctly marked and surrounded by an areola of hyperæmia, slightly elevated, and from three to five millimetres in width. Occasionally deep and rapidly extending ulceration follows; these ulcers are two or three millimetres in depth, with a light pinkish or grayish surface, and have sharply defined but not indurated edges. In the tertiary stage, ulcerations are deep and usually preceded by gummata. A gumma, situated as a rule under the mucous membrane, is at first small varying from three to eight millimetres in diameter, and causes no disturbance, but as it increases in size the mucous membrane covering it becomes congested, and finally, as the gumma softens, a yellowish spot appears at the surface, soon to be followed by ulceration.

Two varieties of ulceration occur in this stage, the superficial and the perforating. The former is most frequently found on the velum, but is also seen upon the pillars of the fauces and tonsils; often having a depth of one or two millimetres. The ulcers have irregular, sharply defined borders and secrete foul, dirty pus, which when cleared away

reveals a floor pale and smooth, with here and there fungoid granulations. Fissures sometimes extend from the edges into the surrounding tissue. Deep ulcers situated on any part of the fauces or pharynx are commonly from three to five millimetres in depth with clear-cut edges, often undermined and indurated. Ulcers of the third stage, whether sequelæ of gummata or not, are apt to extend rapidly, destroying all tissue in continuity, not excepting cartilage and bone. Frequently perforation of the palate occurs (Fig. 92) as if by magic, sometimes as the result of a gumma, which in the palate occurs preferably upon its upper surface. Such ulceration may destroy a considerable portion of the velum within ten or fifteen days.

Etiology.—Syphilis, whether inherited or acquired, is probably due to a specific virus, not yet identified.

Symptomatology.—The primary affection usually causes no symptoms, in the throat unless phagedenic ulceration occurs, giving rise to pain and fever. In the secondary stage, there is dryness of the throat,

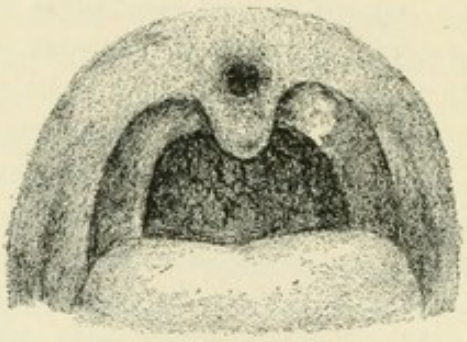

Fig. 92.—Perforating Ulcer of Palate, Syphilitic.

with more or less soreness and occasionally a slight febrile reaction. In some cases, owing to the location of the ulcer, there is great pain upon deglutition. Papillary eruptions upon the skin usually appear at this time. The tertiary form sometimes develops insidiously, and may have produced great mischief without having caused the patient much discomfort. In other cases, owing to the location of the ulcer, severe pain is experienced, especially on deglutition. In such cases constitutional symptoms are then apt to be pronounced, and after a few weeks the patient may present much the same symptoms, with fever and emaciation, as one suffering from advanced tuberculosis.

Diagnosis.—The primary affection is apt to escape observation, but careful examination of the throat may discover a small ulcer situated on an indurated base surrounded by a slightly œdematous, elevated mucous membrane. If this is associated with a suspicious history, and remains obstinate to all treatment for four or five weeks, we may be nearly certain of our diagnosis.

The secondary affection, in the beginning, is liable to be mistaken for *catarrhal sore throat*, but after three or four days the development

of symmetrical, erythematous patches distinctly outlined, or the grayish elevated mucous patches or superficial ulcers, with areolæ of inflammation, will at once suggest the true nature of the disease. However, even then it is possible to confound the affection with *simple membranous* or *herpetic sore throat;* but the specific history, if it can be obtained, or, if not, the progress of the case for the next few days, will settle the diagnosis. The superficial ulceration of this stage should not be confounded with acute tubercular sore throat, if the history, constitutional symptoms, and appearance of the ulcer are taken into account.

The tertiary stage is liable to be mistaken for *scrofulous* or *tubercular sore throat*, the distinctive features of which were pointed out in considering these diseases. The characteristic features of tertiary syphilitic ulceration of the throat are: commonly absence or insignificance of pain and of constitutional symptoms; also the edges of the ulcer are sharp cut, indurated, and sometimes undermined, and the process is rapid.

In a very rare form of diphtheroid syphilitic ulceration of the throat I have seen three cases that have been mistaken for diphtheria.

Prognosis.—The primary disease continues five or six weeks, and then terminates spontaneously. The secondary affection usually comes on in from six to twelve weeks after inoculation, and, as a rule, disappears in from six to eight weeks, or sooner under proper treatment; but sometimes renewed eruptions make their appearance from time to time for several months. The gummata of the tertiary stage sometimes disappear as they came, but usually soften and ulcerate, the ulcers spreading rapidly for two or three weeks afterward; subsequently they may continue to progress more slowly for several months if left to themselves. The primary affection makes little impression on the general health; the secondary is seldom dangerous to life, but the tertiary is often grave. The ulceration in the latter may perforate the hard palate and destroy large portions of the soft tissues, and may sometimes cause death by erosions of a large blood vessel or by narrowing of the air passages. Cicatrization after ulceration frequently narrows or completely closes the opening to the naso-pharynx or causes stenosis of the larynx, interfering with respiration and phonation. Destruction of the palate interferes with phonation, and with deglutition by allowing fluid to regurgitate through the nose. Adhesion of the base of the tongue to the pharyngeal wall sometimes seriously interferes with both respiration and deglutition. In one case which has come under my observation, an opening was left only two or three millimetres in width by six or eight in length. Under appropriate treatment the majority of cases can be relieved and the disease checked, but sometimes, in spite of everything, it goes on or the exacerbations frequently recur until death results.

Treatment.—For the primary affection cauterization is recommended by some, while others favor a negative course. Even for the

secondary lesions some are in favor of confining the treatment in the majority of cases to local measures. Mackenzie (Diseases of the Throat and Nose, Vol. I.) seldom uses constitutional remedies in the secondary stage, relying mainly upon local applications of the zinc chloride gr. xx., ad ℥ i. for the erythematous eruption, or the tincture of iodine for mucous patches, but he recommends mercurials for the inherited syphilis and in obstinate cases of the acquired affection. Sajous (Diseases of the Nose and Throat) advises for the secondary affection local applications of silver nitrate, iodoform, and tincture of the chloride of iron. For the secondary affection, I usually employ a spray of zinc chloride gr. xxx. ad ℥ i. two or three times a week, directing the patient to use at home the same remedy twice daily in the form of spray gr. x. ad ℥ i. For the mucous patches I sometimes rely upon these applications, and at others I use the strong tincture of iodine or a solution of copper sulphate gr. xx. ad ℥ i., having the patient use the spray at home as just recommended. Usually small doses of mercury bichloride and potassium iodide are administered after each meal, and in many cases ferruginous or bitter tonics are given before eating, depending upon the patient's general condition. For the ulcers of tertiary syphilis the strong tincture of iodine is the most efficient application, though occasionally the sulphate of copper, as recommended above, will be found useful. Much, I believe, depends upon the manner of applying the tincture of iodine. The ulcer should be touched repeatedly at each sitting (four to eight times), and a minute allowed between each application for the parts to dry. When the application is completed the surface of the ulcer should appear dry and glazed and of a dark brown color. These treatments should be repeated daily for ten to fifteen days and subsequently less frequently until the parts are healed. At the same time the patient should be given the iodides of sodium and potassium in doses of from 5 to 10 grains each three or four times a day. Under this treatment even large chronic ulcers may be expected to heal in from two to four weeks. If there is a tendency to closure of the entrance to the naso-pharynx, or other vicious adhesions are forming, bougies should be passed frequently until complete cicatrization has occurred; but this should not be attempted until the reparative process has been fully established. It is especially important to be faithful in dilatation just as the last vestiges of the ulcer are disappearing, for at this time contraction takes place with wonderful rapidity.

Syphilitic sore throat in infants, is a congenital manifestation of syphilis usually characterized by ulceration, the favorite seat of which is the palate, naso-pharynx, or posterior pharyngeal wall. According to J. N. Mackenzie, of Baltimore, nearly fifty per cent of the cases occur within the first year of life, and as many as thirty-three per cent within the first six months. In some, however, the development is delayed until near the age of puberty.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS. — Mucous patches are rare, this stage having probably been passed in intra-uterine life; when found, these patches are apt to be located in the upper portion of the pharynx. Ulceration is more commonly present, its favorite seat in order of frequency being the fauces, naso-pharynx, posterior pharyngeal wall, nasal fossæ, septum, tongue, and finally the gums. The ulcers present the appearance of tertiary syphilis in adults, already described, and are peculiarly prone to attack the bones and cartilages.

ETIOLOGY.—The affection is either inherited during the intra-uterine life or contracted during parturition.

SYMPTOMATOLOGY.—This condition of the throat is usually associated with syphilitic lesions in the nose, giving rise to embarrassment of the nasal respiration and difficulty in nursing. This in a short time is followed by a serous discharge from the nose, that becomes thick and purulent, sometimes sanguinolent within a few days. The lips are frequently excoriated, and specific fissures, pustules, and ulcers develop upon the alæ of the nose, the lips, and angles of the mouth, extending outward upon the cheek. Ulceration of the pharynx also may seriously interfere with deglutition.

DIAGNOSIS.—The disease is distinguished from *simple catarrhal inflammation* by the profuse discharge from the nose, the obstruction to nasal respiration, the occurrence of pustules and ulcers upon the lips, and the peculiar ulceration in the pharynx.

PROGNOSIS.—When occurring within the first year of life the disease is nearly always fatal. Older children may recover, but are apt to be left with disfigurement of the nose and partial destruction of the palate with consequent interference with the voice and respiration. Often deafness results. The later the appearance of the disease, the better the chance of cure; but it is apt to break out anew from time to time.

TREATMENT.—The treatment is essentially the same as for adults, though children bear mercurials better. Local applications should be so mild as to cause but little pain.

CHAPTER XXI.

DISEASES OF THE FAUCES.—*Continued.*

DISEASES OF THE UVULA.

ACUTE INFLAMMATION AND ŒDEMA OF THE UVULA.

Acute œdematous inflammation of the uvula is a rare affection except as associated with pharyngitis or tonsillitis. It usually causes but little pain, but is attended by some discomfort in eating and by frequent desire to swallow. The uvula when œdematous sometimes becomes so large as to interfere with respiration, and if it be long enough to touch the base of the tongue or epiglottis it causes an irritating throat cough. The affection is not difficult of recognition.

Treatment.—The proper treatment consists in the application of astringent sprays or the use of astringent troches or gargles, and, if the œdema is great, a few punctures may be made near the lower end of the uvula to allow the serum to escape, but the organ should not be cut off during the acute inflammation unless it seriously interferes with respiration or deglutition, and then only a part ought to be removed. If the punctures are not sufficient to allow the serum to escape, the removal of a small bit of mucous membrane from the tip of the organ is generally effectual.

CHRONIC INFLAMMATION AND ELONGATION OF THE UVULA.

Elongation, though sometimes occurring without chronic inflammation, is generally associated with it. It is apparently due to the same causes as chronic pharyngitis or tonsillitis. Sometimes it takes place without any appreciable cause. In health the uvula is from one-fourth to three-eighths of an inch in length. Sometimes when diseased, it may become three-fourths of an inch in length without causing inconvenience; but in other patients, even moderate elongation causes frequent desire to clear the throat, with expectoration of small masses of mucus, and an irritating cough which occasionally becomes so excessive as to interfere with the patient's rest, and in rare instances, by this means, to bring on symptoms similar to those of serious pulmonary disease. An elongated uvula sometimes causes spasmodic attacks of retching and vomiting and occasionally reflex spasm of the glottis. The symptoms are usually

worse when the patient lies down. In a few cases it gives rise to pain and fatigue after using the voice, and more rarely to hoarseness.

DIAGNOSIS.—Elongation of the uvula may be easily detected by inspection.

TREATMENT.—When all other causes of the symptoms have been excluded, the superfluous part of the organ should be removed by the uvulatome, scissors (Fig. 93), or the nasal snare (Fig. 208). Various uvulatomes have been devised for the purpose, but they are not better than the scissors shown in Fig. 93, which are simple and well suited to the purpose. The nasal snare will be found much more convenient. By it, abscission can be done more accurately, and excessive bleeding is less likely to occur. The snare for this purpose is armed with No. 5 steel wire, a loop just large enough to easily enclose the tip of the uvula is formed, the physician depresses the tongue with one hand, and with the other slips the snare under the tip of the uvula, carrying it up to within from one-half to three-eighths of an inch of its base. If

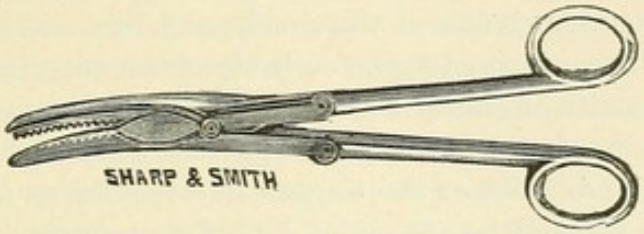

FIG. 93.—SCISSORS FOR AMPUTATING THE UVULA (½ size).

the uvula appears swollen at the time, less should be removed than otherwise, and it is best never to make it shorter than normal. The wire is tightened down until the tissue is secured, then the tongue depressor is removed, and the physician, seizing the cross bar of the snare with his left hand, suddenly draws upon the wire with the combined strength of the fingers of both hands, cutting through the tissue as quickly as by a knife. After the operation, the patient should be supplied with troches of althea to use as often as desired to soothe the pain, and a one per cent gargle of carbolic acid may be advantageously used several times daily until the wound has healed. In a few instances alarming hemorrhage has taken place after cutting off the uvula.

MALFORMATIONS AND NEW GROWTHS OF THE UVULA.

The uvula may be asymmetrical or absent, but the most frequent malformation is bifurcation. This requires no treatment unless the organ is also elongated, when a portion should be removed.

Papillary growths are not infrequently found on the uvula, and if large, by their mechanical effects they may give rise to the same symptoms as elongation. They are easily diagnosticated, and may be readily removed by the snare.

Malignant growths rarely, if ever, first attack the uvula, though it may be involved by extension of the disease from the tonsils and palate. The organ is often involved in syphilitic inflammation and ulceration, but these cases require no special consideration, as they were sufficiently described in speaking of diseases of the adjacent parts.

LEUCOPLAKIA BUCCALIS.

Synonyms.—Leucoplakia buccalis et lingualis, ichthyosis linguæ.

Leucoplakia buccalis is a chronic affection of the buccal mucous membrane, characterized by thickening of the epithelium and the formation of white, opaline, elevated patches, which usually become fissured and painful, and, after continuing for a long time, are inclined to terminate in epithelioma. The disease is very rare, occurring almost invariably in men over forty years of age.

Anatomical and Pathological Characteristics.—The patches are limited to the buccal cavity, and are generally found on the dorsum of the tongue or inner surface of the cheeks and lips, but seldom, if ever, on the lower surface of the tongue or back of the anterior pillars of the fauces. They consist of one or more small, irregular or oval spots which may become confluent. A considerable portion of the tongue alone may be involved, or the dorsum of the tongue, buccal mucous membrane, and the gums, one or all may be affected. The first appearance of the white patch is preceded by hyperæmia, and subsequently in the early stages a hyperæmic areola is found about its borders. Before long the patch itself becomes thickened, sometimes to the extent of six or eight millimetres, and the epithelium which has become hard and dry may be easily removed, or in spots it may be spontaneously exfoliated, leaving the appearance of an ulcer. The surface of the patch is marked by numerous fine lines or furrows which by intersecting each other divide it into small polygonal spaces. Some of these lines may extend as deep fissures down through the thickened epithelium, involving the submucous tissue in a painful excoriation. In cases of long standing, the papillæ may be much enlarged, giving the surface a warty appearance. Under the microscope, the epithelium is found greatly thickened, the papillæ enlarged and flattened, and the blood vessels dilated, with an accumulation of leucocytes about their walls. The superficial layer of the mucous corium is infiltrated with embryonic cells, and the deep layer is involved in vascular alterations.

Etiology.—Excessive tobacco smoking is ranked as one of the most frequent causes of the disease, but it is probable that prolonged irritation of any character may have a similar effect on those predisposed to it. Thus, highly spiced food and alcoholics seem to excite it in some instances; and the occurrence of the affection in several members of the same family led Bazin to believe that it is often the result of constitu-

tional syphilis. It is also attributed to the arthritic or dartrous diathesis.

Symptomatology.—The clinical history of the disease is not definitely known, because generally it has been discovered accidentally and found to have existed for some months or years before it has come under the physician's observation. This is due to the fact that at first the affection causes no inconvenience. The small patch which first appears gradually increases in size and at length stiffness occurs or painful fissures form which first attract the patient's attention. Ultimately, in the majority of cases, epithelioma results and runs its usual course. Sometimes the affection remains stationary for months, or under the influence of some irritant it may rapidly progress, but it may again become dormant if the irritant is removed. Cases associated with syphilis or that have developed into epithelioma are attended by much swelling of the parts, and sometimes deep ulceration, which may erode the vessels and cause severe hemorrhage. In these, the lymphatic glands soon become involved, a sign not observed in the earlier stages of idiopathic leucoplakia. Often the first symptom is merely an uneasy sensation, but in others the mucous membrane early becomes more or less painfully sensitive to spices, hot food or drinks, alcoholics, or tobacco. With the occurrence of fissures, pain may become more intense and almost constant, although in some it is present only at intervals. There are no constitutional symptoms until epithelioma is developed. Late in the disease, speaking, mastication, and swallowing usually become difficult, especially when epithelioma occurs. In such cases also profuse salivation is often a very annoying symptom.

Diagnosis.—Leucoplakia may be misinterpreted for what Guinaud has termed the professional patches found in glass blowers, for smokers' patches, mercurial patches, psoriasis linguæ, syphilitic patches, and epithelioma unconnected with leucoplakia. The *professional patches* occur only in old glass blowers, particularly bottle-makers, and are found symmetrically upon both sides of the mouth, on the lateral surface of the gums, and around Steno's duct. *Smoker's patches* are more irregular than those of leucoplakia, and are commonly located near the commissures of the lips, but not upon the dorsum of the tongue or the inner side of the cheek. Again, the epithelium covering their surfaces is thin and closely adherent, so that it cannot be removed, as in the disease under consideration. *Mercurial patches* are not so thick as those of leucoplakia, are never quite white, and are found on all parts of the tongue, but particularly where it is pressed against the teeth. In *psoriasis linguæ* which sometimes accompanies psoriasis of the skin, the patches of epithelium assume a white, opaque appearance and after a day or two they are thrown off, the epithelium being speedily restored; but soon other patches appear and go through a like course until after a time a large part of the dorsum of the tongue may become denuded and of a uniform red color,

with crescentic markings or depressions entirely unlike the appearance of leucoplakia. *Syphilitic patches* are not so white as those of leucoplakia; they are usually round or oval and more regular in form, seldom occurring on the cheek, but found principally upon the tip or margin of the tongue and often on its lower surface, which is never invaded by leucoplakia. The syphilitic patches are thinner than the patches of leucoplakia, and the lymphatic glands are much sooner involved. The pain is more severe in leucoplakia than in the syphilitic disease, and anti-syphilitic treatment causes no improvement, but on the contrary may aggravate the affection. When syphilis and leucoplakia coexist, the diagnosis is difficult. *Cancer* arising without previous leucoplakia is distinguished from the latter by its history; the induration of the tissues and the final ulceration are not preceded by the chronic white patch, but are attended by more constant pain, with profuse salivation and a very offensive odor.

PROGNOSIS.—The duration of the disease varies from a few months to several years. The majority of cases ultimately terminate in epithelioma, which runs its course to a fatal issue.

TREATMENT.—All sources of irritation, particularly the use of tobacco, alcoholic stimulants and strong condiments, should be at once removed. If the digestive organs are deranged, they should receive proper attention. Aside from these measures, most authors believe treatment to be of little or no avail. Arsenious acid, the alkalies, mercury, and the iodides have been recommended, though in the absence of syphilis the latter seem to be injurious. For local application various caustics, such as silver nitrate, zinc chloride, tincture of iodine, and the solution of mercury nitrate have been recommended, but none of them seem of any value except in cases complicated by syphilis. On the contrary, soothing applications seem to have been the most beneficial, though giving only temporary relief. I have succeeded in curing one well-marked case by repeated careful applications of the galvano-cautery, made to a small spot at each sitting and in such manner as not to destroy the healthy tissue beneath.

For a more complete exposition of this subject the student is referred to my paper, Leucoplakia Buccalis, etc., in the Transactions of the American Laryngological Association for 1885, page 57.

ACUTE TONSILLITIS.

Synonyms.—Amygdalitis, cynanche tonsillaris, quinsy.

The tonsils, which are located between the pillars of the fauces, are, in the normal condition, scarcely visible and never large enough to project beyond the edges of the anterior pillars. They are essentially lymphatic glands, but their function is unknown. It is believed by some that they absorb a portion of the starchy foods, which their secretions are capable of converting into sugar, but this is certainly an unimportant function.

Upon the free surface of these glands are the orifices of from twelve to eighteen lacunæ or crypts which are lined with a continuation or pouch of the mucous membrane and surrounded by numerous spherical and lymphoid follicles. These, together with softer lymphoid tissue, constitute the substance of the tonsil, and are the parts more or less involved in the disease under consideration. Acute tonsillitis is most prevalent in humid climates and during the spring and winter months. It is more frequently observed between the ages of fifteen and thirty years, especially in subjects of the rheumatic diathesis. It is peculiarly prone to attack those patients in whom the tonsils are hypertrophied; and those who have once suffered from it are liable to repeated attacks. It is only occasionally witnessed in young children or the aged.

Anatomical and Pathological Characteristics.—The inflammation may attack the mucous membrane covering the surface of the tonsils, it may be mainly confined to the follicles, or it may involve the whole substance of the gland, with or without the peritonsillar connective tissue. It is frequently confined to one side, but in many cases, when the disease has nearly run its course in one gland, the other will become likewise affected. The mucous membrane covering the tonsil, the pillars of the fauces, and a portion or all of the pharynx is red and swollen. The uvula is generally swollen and elongated, and is frequently seen adhering to the affected tonsil. In the follicular variety of the disease, the orifices of the crypts may become occluded and the lacunæ distended by the changed secretion, in which event rupture may finally occur, with a discharge of the contents, or, on the other hand, the pent up secretions may become the centre of a suppurative process leading to a tonsillar abscess.

Etiology.—The disease is usually attributable to exposure, the rheumatic diathesis, or chronic enlargement of the glands. Among the occasional causes of the attack are: errors of diet, suppression of the menses, a strumous constitution, and heredity. Higston Fox (Transactions of the Medical Society of London, Vol. IX, p. 255) believes that, where both glands are simultaneously involved, the disease is almost invariably of septic origin. The follicular variety of the disease is thought by some authors frequently to result from diphtheria. This view, however, does not accord with the experience of the great majority of physicians, though undoubtedly a few cases are of diphtheritic character.

Symptomatology.—Most patients give a history of previous similar attacks. The disease is usually preceded by malaise for several hours and attended by aching of the back and limbs, and is often ushered in by a slight chill and fever. This is speedily followed by sensations referable to the throat, with swelling of the glands and more or less pain and difficulty in moving the jaw. In the later stages of severe cases there may be great depression, cold perspiration, insomnia, restlessness, and sometimes delirium. The patients are usually worse during

the night, and experience most pain early in the morning on account of the dryness of the throat. In the inception of the attack there are usually sensations of dryness or pricking in the parts, soon followed by pain, which is aggravated by deglutition and after a time becomes very severe, even on attempts at swallowing the saliva. This pain is referred to the region surrounding the angle of the jaw, and radiates toward the ears. Occasionally there is severe headache, which is aggravated by movements of the head. Owing to the tumefaction, the patient is frequently unable to open his mouth more than half an inch; partial deafness is common; and the senses of taste and smell are sometimes obtunded. The face becomes puffy and swollen, the skin hot, the pulse rapid, and the temperature may rise to 103°, 104° or 105° F. A high temperature is more to be expected in children or in persons suffering their first attack. Articulation is difficult and enunciation muffled. The swollen glands may seriously interfere with nasal and oral respiration, so much so that patients frequently fear suffocation, which indeed in extremely rare cases, is an actual danger. There is little or no cough, but the patient is frequently impelled to clear the throat of a thick, viscid secretion which causes much discomfort. The tongue is coated with a yellowish white fur, while the breath is very offensive. There is increased thirst, and usually loss of appetite. Even when there is a desire for food, the patient can seldom take it on account of the painful deglutition, while attempts at swallowing fluids oftentimes result in their regurgitation through the nose. The bowels are nearly always constipated. Upon examination of the fauces, the congestion and swelling of the parts will be readily distinguished. It is often desirable to make the examination with the aid of a laryngoscopic reflector, for the patient is unable to open the mouth sufficiently to permit a thorough inspection with ordinary illumination. In the follicular type of the disease, the orifices of the crypts may be filled with a yellowish white secretion which causes round or oval patches from four to eight millimetres in diameter. In exceptional instances a rash has been observed upon the skin.

Diagnosis.—Acute tonsillitis is to be distinguished from scarlatina, diphtheria, phlegmonous tonsillitis, and syphilis. The essential points in the diagnosis are the history, swelling of the parts, difficulty in opening the mouth, and severe pain on deglutition.

In children, *scarlatina* is usually ushered in by vomiting, which is not the case with tonsillitis. The fever is often higher, is always more persistent, and after a few hours a bright red rash appears upon the surface of the body. Usually the congestion of the fauces is much more diffuse in scarlatina than in tonsillitis, and the swelling of the parts is much less. The peculiar appearance of the tongue in scarlatina is not observed in tonsillitis.

Acute tonsillitis may be distinguished from scarlatina as follows:

ACUTE TONSILLITIS.	SCARLATINA.
Inflammation and swelling of tonsils. But little redness of pharynx or palate.	General redness of fauces, sometimes appearing in patches, sometimes little or no swelling of tonsils.
Pain about angle of jaw, often referred to the ears.	Pain, usually confined to the throat, until late in the disease.
Difficulty in opening the mouth.	No difficulty in opening mouth.
Tongue coated yellow.	Strawberry red tongue.
Usually no eruption on skin.	Characteristic rash on skin.

The fever is at first commonly lower in *diphtheria* than in tonsillitis, there is no difficulty in opening the mouth, and usually there is but little pain. Upon examination of the fauces, there is found a thick, grayish white membrane uniformly covering a large portion of the throat or confined to one or two patches upon the tonsils. These patches are much larger than the yellowish masses seen at the orifices of the crypts, and are less numerous, and they appear to be laid upon the mucous membrane instead of being beneath it or even with its surface. In cases of bilateral follicular tonsillitis, the disease is frequently septic, and paralysis of the pharyngeal muscles may follow, very closely simulating that of diphtheria. Probably some of these are truly diphtheritic in character.

Acute follicular tonsillitis and diphtheria present the following differential points of diagnosis:

ACUTE FOLLICULAR TONSILLITIS.	DIPHTHERIA.
Tonsils inflamed, enlarged.	Tonsils not always enlarged.
Whitish or yellowish deposit at orifices of crypts.	Thick, grayish white membrane on fauces or tonsils, or possibly confined to one tonsil, much larger than the deposit of tonsillitis.
High fever.	Oftentimes subnormal temperature.
Difficulty in opening mouth.	No difficulty in opening mouth.

Phlegmonous tonsillitis is more likely than acute tonsillitis, to be confined to one side of the throat. The swelling and pain are greater, the difficulty of opening the mouth is more pronounced, and after four or five days rigors indicate the formation of considerable pus, while fluctuation may occasionally be detected, especially if one finger is placed on the tonsil and the other behind the angle of the jaw externally.

We can usually readily distinguish *syphilitic sore throat* from acute tonsillitis, but there are cases in which a diagnosis is attended with much difficulty. In specific sore throat, there is generally little or no fever, and ordinarily but little pain; the redness and swelling of the parts usually occur in symmetrical patches upon both sides; and the congestion is seldom of that bright red character seen in tonsillitis. In the

secondary disease superficial ulceration and mucous patches, with possible eruptions upon the skin, and in the tertiary form, deep ulceration with moderate congestion, a peculiar swelling, together with the history and other symptoms, will usually enable the physician to make the diagnosis easily.

From syphilitic sore throat the disease is distinguished by the following points of difference:

Acute tonsillitis.	Syphilitic sore throat.
No specific history. Inflammation and swelling. Parts bright red.	Syphilitic history. Comparatively little inflammation or swelling.
Often collection of yellowish secretions in follicles.	Mucous patches usually symmetrical.
High fever, acute pain.	But little fever or pain.
Difficulty in opening mouth.	Usually no difficulty in moving jaw.

Prognosis.—There is very little danger to life from the disease, although death has been known to occur in a few instances. The affection often terminates in chronic hypertrophy of the glands, and not infrequently a simple inflammation eventuates in suppuration. It is usually the forerunner of other similar attacks, and is occasionally immediately preceded or followed by acute articular rheumatism. It often terminates in four or five days; sometimes, however, it lasts ten days or two weeks, and in exceptional cases as long as three weeks.

Treatment.—Persons subject to tonsillitis should avoid all exposure likely to excite the inflammation, and should be careful to keep the digestive organs in perfect condition, attending especially to regularity of the bowels. Guaiacum has been highly recommended for aborting the disease. It is given in the form of troches, each containing two or three grains, every two hours during the beginning of the attack, or the ammoniated tincture in doses of a drachm every fourth hour may be administered in milk. Although this remedy has the sanction of high authority, I must admit having seen very little, if any, benefit from its use. Brushing the tonsils with a sixty grain solution of silver nitrate will cut short the attack in probably about one in four cases. Aconite, opium, and belladonna given in small doses, frequently repeated, have the power of speedily abbreviating the disease in some instances. Aconite may be given in doses of half a minim of the tincture every fifteen minutes until sweating or other constitutional effects are produced; and thereafter less frequently, about once an hour for four or five hours, and still later once in two, three, or four hours, according to the febrile symptoms. The tincture of opium may be given in doses of one minim every fifteen minutes at first until the patient experiences relief from the sensations in the throat, and subsequently once in from two to four hours, according to its influence upon the pain. Tincture of belladonna may be given in a similar way in doses of a half-minim. By

some of these measures the disease may frequently be aborted; but it will be found that a remedy which acts well in one person will often be entirely inefficient in another. In the beginning, constipation should be relieved by the employment of a mercurial or saline cathartic.

Ice held continuously in the mouth, or applied externally by means of ice bags, will frequently check the commencing inflammation. Frequent gargling with strong solutions of potassium chlorate and nitrate, in water as hot as can be borne, is very beneficial after the disease is fairly established. For this purpose it is my custom to order one part of the chlorate and two parts of the nitrate, and direct the patient to use a heaping teaspoonful of this in half a teacup of hot water every half hour. Gargling with a one-half per cent to two per cent solution of carbolic acid is also useful in many cases. A one per cent solution of salicylic acid is also recommended. Lemonade may be taken frequently to clear the throat of the tenacious mucus. Dobell's solution is also an excellent mouth wash for this purpose. Whenever there is evidence of a rheumatic habit, guaiacum is indicated and may be advantageously combined with small doses of opium and medium doses of the potassium bromide, which relieve the pain and lessen congestion. If, in spite of these various remedies, the inflammation progresses and the tonsils become much swollen and painful, scarification, deep incisions, or four or five simple punctures will often give great relief. In making an incision, the bistoury should be passed with its back toward the outer portion of the tonsil and the cut made toward the median line. Where the gland is very large, two or three of these cuts should be made. When the patient is subject to frequent attacks and the tonsils remain large after the inflammation has subsided, removal of the glands should be advised. There are some patients who suffer from recurring attacks of acute tonsillitis in whom the glands subside after each inflammation so that during the period of health they appear but little if any larger than normal. In such cases it has been recommended that the glands be removed during the period of an acute inflammation, while they are considerably enlarged. The main objection to this procedure is the excessive hemorrhage which sometimes follows. These cases may be very satisfactorily treated by repeated punctures with the galvano-cautery. In carrying out this treatment two or three punctures should be made at each sitting, this not to be repeated until two or three days after the soreness occasioned by the last cauterization has subsided. The treatment is necessarily protracted, as ten or a dozen cauterizations will usually be found necessary. In some of these cases I have obtained excellent results by passing a vulsella forceps through the fenestra of the tonsillitome, seizing the gland, drawing it well out, and then cutting it off with the latter instrument.

PHLEGMONOUS TONSILLITIS.

Synonyms.—Suppurative tonsillitis, abscess of the tonsils, quinsy, phlegmonous sore throat.

Phlegmonous tonsillitis is a suppurative inflammation of the tonsil and peritonsillar tissue, characterized by the formation of a circumscribed abscess. It occurs most frequently in children or young adults; seldom before the tenth year of age, and not commonly after the thirtieth year. Persons who have had it once are much more liable to attacks than others; and those having chronic enlargement of the tonsils are peculiarly subject to this variety of inflammation.

Anatomical and Pathological Characteristics.—The inflammation attacks the mucous membrane, the glandular, or the peritonsillar tissue—sometimes part and sometimes all of the tissues—and frequently extends down to the sheaths of the muscles. Sometimes the muscles themselves are involved, but usually the force of the attack is expended upon the connective tissue about the gland. The swelling is nearly always unilateral, and the abscess which forms is, I think in at least four-fifths of the cases, outside of the gland itself.

Etiology.—The causes of the disease are the same as those of acute tonsillitis, with the addition usually of some debilitating circumstance which has rendered the patient peculiarly susceptible to suppurative inflammation.

Symptomatology.—Inquiry into the history of such a case frequently reveals that the person has had kindred attacks several times during the previous two or three years. The local and constitutional symptoms in these cases are essentially the same as those of ordinary acute tonsillitis of the severer grade. Superadded to these we nearly always find rigors at the time suppuration takes place, and sometimes a peculiar, sharp pain is associated with the formation of the abscess. Swelling of the part is excessive, so great in some instances, even though confined to one side, as to fill the whole fauces. As the disease progresses, the spot at which an opening is about to take place may be distinguished. This is at first more livid than the surrounding tissue, and after a time it becomes yellowish and slightly prominent, and finally the tissue gives way and pus escapes.

Diagnosis.—The disease is to be differentiated from the same affections that are liable to be mistaken for acute tonsillitis. It is not always easy to distinguish it from acute inflammation of the glands without suppuration. The essential points in the diagnosis are the sharp pain and rigors at the time of suppuration, and the occurrence of fluctuation, occasionally to be detected by palpation. However, in many cases the tissues are so tense that palpation will not give distinct fluctuation even though considerable pus be present. Then an exploring needle must be employed.

PROGNOSIS.—We expect suppuration to occur from the third to the sixth day. If the case is left to itself, the abscess will usually open spontaneously about the tenth day, and the patient will so far recover as to be out of doors within three or four days after the abscess has been evacuated. So far as life is concerned, the prognosis is favorable. There have been, however, a few exceptions to this rule. Convalescence is usually very rapid, though sometimes the inflammation is followed by some paralysis of the muscles of the fauces, which may last several weeks. Paralysis of the palate causing indistinctness of speech, and regurgitation of fluids through the nose when the patient attempts to swallow, is the most prominent of these manifestations. In rare instances typhoid symptoms supervene upon the acute inflammation.

TREATMENT.—Early in the attack the disease may be aborted as in acute tonsillitis—in about one case out of four—by the application to the inflamed gland, once or twice a day, of a sixty grain solution of silver nitrate, two or three applications usually being sufficient. If the case is seen early, I would advise this treatment, for, even if it does not succeed, it is not harmful. Care should be exercised that none of the solution drops into the lower pharynx or the larynx, where it would be likely to cause spasm of the glottis. Guaiacum has been highly recommended as a specific for this disease, used in the form of troches, or the ammoniated tincture as already recommended for simple tonsillitis; but it is useless to continue with it longer than forty-eight hours. My personal experience with this remedy has been unsatisfactory; I have never seen an attack aborted by it, though some have apparently been shortened. If abortive measures prove unavailing, we seek to conduct the inflammation to a speedy resolution. For this purpose, aconite, opium, and anti-rheumatic remedies are of chief value. Tincture of aconite or tincture of opium should be given in minim or half-minim doses once in fifteen to thirty minutes until the patient is relieved or the constitutional effects of the remedy appear; afterward once an hour for a few doses, and subsequently less frequently as the symptoms subside. Ordinarily eight or ten doses must be given close together, and as many more once an hour. In most of these cases, after the first twenty-four hours, sodium salicylate gr. viiss., with potassium bromide gr. x., every fourth to sixth hour, are especially beneficial. Local applications are valuable in the onset of the disease, ice being the best remedy. It may be held in the throat constantly, or may be applied in ice bags externally, or cold applications may be made by means of the Leiter coil. Some patients, however, are made uncomfortable by cold; in such we recommend gargling once an hour of the solution hot as can be of potassium nitrate and chlorate, recommended for acute tonsillitis. Usually in the first stage of the disease cold applications are to be recommended, and after the second day hot applications. Many of the patients are constipated; this is best overcome by saline cathartics. Scarification of the tonsils will

sometimes give great relief, even before suppuration has taken place. Pus should be evacuated as soon as discovered. Pain from the incisions may be in great part prevented by a few applications of a ten per cent spray of cocaine. Some patients think that if the tonsils are cut they are more liable to subsequent attacks, but there is no foundation for such belief.

HYPERTROPHY OF THE TONSILS.

Synonym.—Chronic tonsillitis. This includes chronic follicular tonsillitis.

Hypertrophy of the tonsils is an affection characterized either by a collection of secretions in the crypts of the gland and consequent irritation, with or without hypertrophy of the parenchyma known as—chronic follicular tonsillitis, or by simple hypertrophy of the glandular tissue with but little involvement of the lacunæ. About two-thirds of the cases occur in boys. It is most frequent in youth or in young adults, but it is also very common in children, and is congenital in rare instances. The tendency to the disease diminishes with advancing years. The hypertrophied tonsil presents a yellowish-pink or dusky red color; it varies in size from a large almond to a large walnut, and may weigh from one to three drachms. At times the gland is very friable; again it is firm, cutting with a creaking sound, owing to increase in the connective tissue. Some of the lacunæ may be filled with an extremely offensive secretion of yellowish color and cheesy consistency. When the follicles are involved, with but little hypertrophy of the glandular tissue, this secretion will be found in several of them.

Etiology.—The disease is most frequently the result of repeated acute attacks of inflammation of the gland, especially when occurring in subjects of a strumous or rheumatic diathesis. But the starting point often seems to have been an attack of diphtheria, scarlatina, or measles. Again it has also been attributed to chronic follicular pharyngitis and to acquired syphilis, while occasionally it is supposed to be of hereditary origin. The view has been advanced that follicular disease of the tonsil is caused by bacterial development in the lacunæ, but as many varieties are found in such cases and as bacteria are always present in decaying organic substances and associated with dead tissue, their presence here is not sufficient reason for believing that they cause the disease.

Symptomatology.—Sometimes there is the history of a hereditary tendency to the disease, and usually a history of noisy or snoring respiration with altered voice, and frequent acute attacks of tonsillitis. In children particularly, partial deafness is a frequent symptom. In rare cases the senses of smell, taste, and sight are said to be affected. Pain is seldom present, except when the lacunæ become much distended by the secretions, but the patient often experiences more or less discomfort in deglutition, and sometimes complains of a sense as of a for-

eign body in the throat. Where the glands are large, particularly in children, the open mouth, dull eye and stupid appearance are almost characteristic of the disease. The voice is usually thick, as though the patient had something in the mouth when speaking; it may be husky or hoarse, or may possess a guttural or nasal quality. Some of these patients are easily fatigued by speaking for any length of time. Respiration is obstructed in proportion to the enlargement of the glands. This is more especially noticeable during sleep, when the respiratory movements are often painful to behold. As a result of poor aëration of the blood, there is frequently great deterioration in the general health.

There is but rarely actual danger of suffocation, though serious symptoms pointing in this direction are occasionally observed. Cough is not usually present, but it may sometimes occur in severe paroxysms. In many patients there is a frequent desire to clear the throat of mucus. I have seen children who have coughed much at night, especially during the winter, in whom the cough has been immediately and permanently relieved by removing the enlarged tonsils. Continued difficult breathing in children may cause deformity of the elastic chest walls, which take the form of the pigeon breast, or the pyriform chest in which the upper part is prominent and the lower contracted. These distortions only occur when the tonsils are extremely large, and possibly when the bony and cartilaginous structures are unusually soft. Impairment of the special senses and the obstruction of respiration with its sequences, commonly attributed to hypertrophy of the tonsils, are probably the result, in most cases, of associated hypertrophy of the pharyngeal tonsil. The enlarged glands may sometimes be evident externally, at the angles of the jaw, and occasionally the cervical glands are also enlarged. Upon examination of the throat the appearance of the tonsils already described may be seen at once.

DIAGNOSIS.—There can be no difficulty in making the diagnosis if the throat is inspected, except in rare instances where the anterior pillars of the fauces are adherent to the tonsils and hide them from view. In such cases the occurrence of retching usually rolls the glands out so that they can be readily seen; but if this does not occur, palpation, with one finger on the tonsil and the other externally, will readily detect the enlargement.

PROGNOSIS.—The disease may be expected to extend over several years; but when occurring in childhood, spontaneous recovery not infrequently occurs at puberty. In young adults, the trouble usually subsides by the thirtieth year. There is little danger from the disease excepting that it may impair the general health or the special senses, as already indicated. Persons with these glands hypertrophied are subject to frequent attacks of acute tonsillitis, and it is probably a fact that in them the throat affections of scarlatina and diphtheria are more dangerous than in those whose glands are normal.

TREATMENT.—In young children where the glands are soft, the re-

peated application of powdered alum or other astringents, or the use of counter irritation at the angle of the jaw, or the internal administration of the syrup of the iodide of iron, or some other preparation of iodine, will occasionally cure the disease, but this manner of treatment is too uncertain to be recommended excepting where the patient will tolerate no other. Enlarged tonsils may sometimes be reduced by repeated injections, into the substance of the gland, of iodine, ergot, or carbolic acid; or by electrolysis, by the galvano-cautery, or by cauterization with chromic acid or other caustics. The galvano-cautery is especially useful in the treatment of chronic follicular tonsillitis. It is highly recommended by

FIG. 94.—MATHIEU'S TONSILLITOME (2-5 size), with fenestra at right angles to handle.

C. H. Knight, of New York, and others for reduction of hypertrophy in these glands, but it is a tedious process; usually from ten to twenty or thirty sittings will be required before the desired end is accomplished, and each of these will cause but little less discomfort than excision, yet the method is to be recommended where there is danger of bleeding, where the disease is mainly confined to the follicles, and in some cases where the chronically inflamed gland is not sufficiently large to be removed by other means. Electrolysis may be useful in some instances, but it is tedious and not very satisfactory. Enucleation of the whole gland by the finger has been recommended, but its accomplishment is difficult unless the mucous membrane has been first

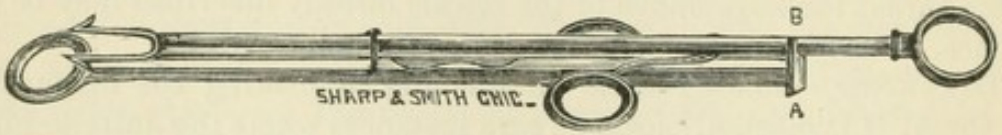

FIG. 95.—THE SAME AS FIG. 94, fenestra placed obliquely.

cut around at the base, and even then there is unnecessary bruising of the surrounding tissues. In adults, the quickest, easiest, and altogether most satisfactory procedure is removal by means of the tonsillitome, which is far preferable to the old method by means of the forceps and bistoury, because of the rapidity of the operation and the small danger of bleeding. Many varieties of the tonsillitome are used, but Fahnestock's, also known as Mathieu's (Figs. 94 and 95), has proved most satisfactory. It is suitable for all cases, and will sometimes engage a gland which cannot be secured by other varieties of the instrument. In performing the operation, the patient is to be placed in a good light, and an assistant should make pressure behind the angle of the jaw with the finger so as to crowd the gland well into view. The operator should then depress the tongue, encircle the tonsil with the ring of the tonsilli-

tome, press the instrument firmly down to the base of the gland and cut it off with a single movement. The other may be removed in the same way a few minutes later. The glands may first be partially anæsthetized by a spray of cocaine, but the operation is not usually very painful without it, and cocaine is somewhat objectionable as it tends to increase the bleeding, which sometimes comes on two or three hours later. It is well to have the patient use frequently a gargle of a solution of one and one-half per cent of carbolic acid, until the wound has healed. Some recommend that only a slice be removed from the tonsil, with the hope that the remainder will atrophy; but the entire gland is diseased and, if any considerable part of it is allowed to remain, the patient is almost sure to suffer from a recurrence of the growth, or at least from repeated attacks of acute inflammation; therefore it is better, when possible, that the whole gland be removed. There are some cases of chronic inflammation of the tonsil in which the gland becomes large only during the acute exacerbations. These may be treated by the galvano-cautery or, as recommended by Lennox Browne, the gland may

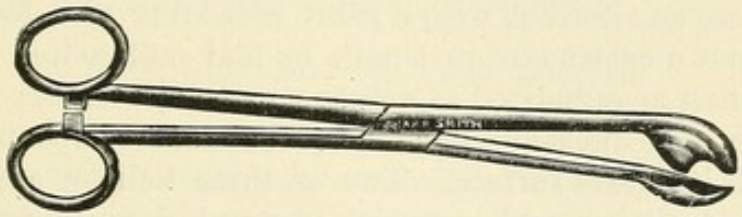

FIG. 96.—INGALS' TONSIL FORCEPS (2-5 size).

be removed during an acute attack of inflammation, notwithstanding the increased danger of hemorrhage. In such cases I have obtained very gratifying results by using a vulsella forceps and the tonsillitome, as indicated under acute tonsillitis.

In adults, as a rule, écrasement is a less satisfactory operation than excision by the tonsillitome; but for young children it is much preferable, because it may be done under the anæsthetic influence of chloroform with much less shock to the friends, and with but little fright to the child, and also because it is nearly or completely bloodless. My method of performing this operation is to give the patient chloroform, place him in the prone position, seize the enlarged gland with the tonsil forceps (Fig. 96) which I have had constructed for this purpose, and then slip over the forceps and down over the gland the steel wire loop of the snare which is used for removing nasal polypi. As the loop is drawn tight, it slips under the blades of the forceps and either cuts the gland close to its base, or better yet, by sliding beneath, completely removes it. During the operation the child's mouth is kept open by a gag. I have found it preferable to remove the undermost gland while the patient is lying upon one side of the face, then turning him over to remove the other. In seizing the gland, the forceps should be carried back to the pharyngeal wall, opened out, and

then drawn forward until they strike the anterior pillar. At the same time, pressure is made externally behind the angle of the jaw, the forceps are crowded down, the blades engage the upper and lower portion of the gland, grasping it firmly, and the handles are locked. The snare is then slipped over the forceps and the gland cut off and removed. This may often be done without the loss of a drachm of blood. To avoid removing the uvula at the same time considerable care is necessary that it be not caught in the forceps or snare with the tonsil. Where the anterior pillar of the fauces is adherent to the gland it should first be separated by a blunt hook and the finger. A strong uvula holder similar to that shown in Fig. 84, though less bent at the hook and with a larger handle, answers well for this purpose. Treatment of follicular tonsillitis is unpromising by the ordinary methods, yet the disease may sometimes be cured by inserting into the follicles, one after another (two or three at each sitting), a small quantity of silver nitrate or chromic acid, the retained secretions having first been squeezed out. Treatment by means of the galvano-cautery is usually very satisfactory, and in using this instrument there is no necessity of first squeezing the secretions out of the follicles. I use an electrode with a point consisting of a loop of platinum wire about a centimetre in length by four millimetres in breadth. The tonsil is first anæsthetized as well as may be by cocaine; the point is then passed into the diseased follicle, heated, and moved about for a second so as to touch its entire surface. Two or three follicles are treated in this way at each sitting, and excepting in rare instances a few days later these points will be found to be completely cured. From five to a dozen sittings may be required to cure cases of this kind. The treatment should not be repeated for five or six days; that is, till two or three days after any soreness occasioned by the preceding cauterization has disappeared.

Excessive bleeding is not common after tonsillotomy, but a few cases of alarming hemorrhage have occurred, and there is a possibility of death from this cause. Though the danger of this is so small as hardly to merit consideration, yet we should always be prepared to check any undue hemorrhage as speedily as possible. The methods which have been found most effective for this purpose are: the sucking of ice, rubbing powdered alum upon the cut surface, compression of the stump of the tonsil by the finger or thumb or by means of a sponge saturated with a strong solution of tannin or of iron persulphate, which may be applied by the finger, or by one blade of a pair of forceps the other being pressed against the external parts. Mackenzie recommended a mixture of two drachms of gallic to six of tannic acid, and enough water to make an ounce, which is to be gradually sipped, instead of being used as a gargle. This will prove efficient in nearly every case. In two such cases I have resorted to the galvano-cautery, once with perfect success, but in the other I was obliged later to use compression by

means of cotton saturated with persulphate of iron. Hot water and various other substances have also been used successfully; but in the most severe hemorrhage that ever occurred in my experience, after all other methods had failed, the bleeding stopped as soon as fainting occurred, and did not reappear. This harmonizes with the suggestion made by D. Bryson Delavan, of New York, who recommends that in excessive hemorrhage after tonsillotomy the limbs and arms be corded so as to retain as much blood in them as possible, and that fainting be encouraged; he having observed that, in all serious cases, as soon as this took place the bleeding stopped. When advising removal of the tonsils, we are often asked as to its probable effect upon the voice, and occasionally as to its influence upon the generative organs. To the first we may answer positively that it will improve the voice if it alters it in any way; to the second, we may answer that there is no reason for believing that the tonsils have any influence whatever upon the generative organs, though the statement of Chassaignac indicates his belief that hypertrophy of the tonsils tends to arrest growth of these parts, and removal of the tonsils favors their development.

CONCRETIONS IN THE TONSIL.

Synonym.—Calculus of the tonsil.

Concretions in the tonsil consist usually of a collection in the lacunæ of desiccated secretions from the follicles, by which the gland may be much enlarged or inflammation excited. Some of these are hard and others soft. The hard consist of the phosphate and carbonate of lime; the soft, of the *débris* of the epithelial cells, cholesterin, pus cells, and bacteria, with more or less chalk. This latter condition was considered under the head of chronic follicular tonsillitis.

Etiology.—The affection is due to inflammation of the lacunæ.

Symptomatology.—There is usually a pricking sensation in the tonsil, with sometimes a little difficulty in swallowing. The gland is swollen, and upon inspection we find a yellowish white spot where the mucous membrane is distended by the mass, or some portion of the calculus may be seen and felt protruding from the surface. By touching the mass with a probe, we can readily determine whether it is hard or soft.

Prognosis.—Where small, the concretions are frequently expelled spontaneously. Their persistence predisposes to hypertrophy of the tonsils and acute or phlegmonous tonsillitis.

Treatment.—Remove the concretion, and if necessary cauterize the empty crypt.

MYCOSIS OF THE TONSILS.

Mycosis of the throat is a parasitic disease of the tonsils and upper portions of the throat, characterized by yellowish white deposits resembling in some cases those of chronic follicular tonsillitis.

Anatomical and Pathological Characteristics.—The deposit usually occurs in numerous small, yellowish or yellowish white patches from two to five millimetres in diameter. These are found sometimes within the crypts of the tonsil or more frequently close to their orifices, but are not uncommonly seen upon the pillars of the fauces or the pharynx, and often in considerable numbers upon the base of the tongue. The deposit may in some cases be so soft as to be easily scraped off, but in other instances it is quite hard. Sometimes it is so prominent as to become almost pedunculated, and often it presents a papillary or warty appearance. According to Delavan, scrapings from the diseased part, when examined microscopically, show the presence of granular matter, pus corpuscles, leucocytes, cholesterin, and, most important of all, the *leptothrix buccalis* (Reference Handbook of Medical Sciences, Vol. VII). This organism attacks mainly the outer layers of epithelium, but sometimes extends deeply into the mucosa, which explains the difficulty, in certain instances, of its removal by swabbing or scraping.

Etiology.—The causes of the affection are not definitely understood, but it is said frequently to arise from carious teeth, where the leptothrix finds a congenial soil.

Symptomatology.—Frequently mycosis gives rise to no inconvenience and is only discovered by accident; but in other cases pricking sensations and other symptoms similar to those of chronic follicular tonsillitis are experienced.

Diagnosis.—The affection is liable to be mistaken for acute or chronic follicular tonsillitis or glossitis, upon which, indeed, it may be engrafted. From the *acute* affections, it may readily be distinguished by the absence of congestion and swelling of the parts and febrile symptoms, and by its prolonged course. From *chronic* follicular affections of these parts, it is to be distinguished by the position and appearance of the deposits, and by a microscopic examination, which in this disease reveals a large number of the micro-organisms already referred to. The deposit in mycosis is either soft or hard; and it occurs, as a rule, in smaller masses than that of chronic follicular inflammation; although in many cases it is found within the crypts, on careful inspection it will be observed in some places clinging to the surface of the mucous membrane at the orifice of the crypts or even remote from them. The wart like and sometimes pedunculated appearance which obtains with some of the masses is never found in follicular tonsillitis or glossitis. The foreign

products are usually smaller and much more numerous in mycosis than in either of the diseases just named.

Mycosis may be differentiated from acute follicular tonsillitis as follows:

MYCOSIS.	ACUTE FOLLICULAR TONSILLITIS.
No inflammation or swelling.	Inflammation and swelling.
Absence of febrile symptoms.	Fever.
Prolonged course.	Brief history.
Deposit soft or hard and in small masses; may be found either at orifices of crypts or remote from them.	Collection of soft, yellowish secretions in the lacunæ.

From chronic follicular tonsillitis, mycosis is to be distinguished by the following characteristics:

MYCOSIS.	CHRONIC FOLLICULAR TONSILLITIS.
Often history of carious teeth only.	Often history of strumous diathesis, or of diphtheria, scarlatina, or measles.
Tonsils usually of normal size.	Tonsils usually enlarged.
Deposit in small masses; found on mucous membrane, and may be remote from orifices of crypts. They often appear like decolorized warty growths, firmly attached to the mucous membrane and standing out two or three millimetres from the surface.	Deposit within the lacunæ, often in large masses, not adherent to the mucous membrane.

PROGNOSIS.—The affection, if left to itself, is of long continuance, and, if the masses are scraped off, they tend to recur speedily, though spontaneous recovery sometimes takes place.

TREATMENT.—The usual forms of treatment advised for chronic affections of the throat have little or no influence upon mycosis, and, in order to eradicate it, thorough and radical measures must be adopted. Delavan recommends frequent applications to the throat of gargles or sprays containing either mercury bichloride gr. i. ad ℥ iv. or sodium biborate gr. xx. to xl. ad ℥ i.; but especially scraping off the deposit with a sharp curette and then applying the galvano-cautery to the site of the growth. I have seen no benefit from local applications of an antiseptic, stimulant, or caustic character, excepting the treatment by the galvano-cautery which has proven very efficient, and it has not been found necessary to scrape the part before its application. Cocaine is first applied, and then the masses are each carefully touched by the galvano-cautery point, four or five being treated at each sitting, and the process repeated once in four or five days until all the growths have been destroyed. There is but little tendency to recurrence of any of the masses which have been thoroughly treated by the galvano-cautery. Carious teeth should, of course, receive proper attention.

TUBERCULAR ULCERATION OF THE TONSILS.

Tubercular ulceration of the tonsils is extremely rare as a primary lesion, but is not uncommon as a concomitant of advanced tuberculosis.

Anatomical and Pathological Characteristics.—Usually the surface of the tonsil is pale and more or less covered with a viscid, yellowish gray secretion, beneath which the tissues appear eroded or worm eaten by irregular superficial ulcers, which may by extension involve the pharyngeal wall or larynx. The borders of these superficial ulcers are not sharply defined, but irregular, and there is little or no swelling of the surrounding parts. Sometimes, however, the ulcers are much deeper, and exceptionally the edges may be sharp cut and elevated, everted, or according to some authors even undermined, but these latter appearances are extremely rare. Sometimes the parts are slightly more congested than the surrounding tissue. In the deep ulceration which I have seen, the borders have been clearly cut, but never undermined as in syphilis nor indurated as in malignant disease. The surface has presented a pale, granulated appearance, bleeding easily upon being touched. Microscopical examinations of scrapings from the parts show a small amount of fibrous tissue, epithelial and pus cells, with abundance of granular matter, and occasionally giant cells, but the bacillus tuberculosis cannot often be detected.

Symptomatology.—In all the cases which have come under my observation, painful deglutition has been the most prominent symptom, and in the major number this has been severe. Usually, even though the tubercular process is slight in other organs, the constitutional symptoms are very pronounced. The pulse is rapid, the temperature rises two or three degrees every day, the strength fails, night sweats are common, and the appetite is usually poor. Cough and expectoration may, however, be absent or but slightly troublesome if the lesion is confined to the faucial region. As the disease progresses, constitutional symptoms become more and more marked and the evidences of tuberculosis in other organs rapidly develop.

Diagnosis.—The disease may be confounded with syphilis or cancer. The essential points in the diagnosis are: painful deglutition, the constitutional symptoms, and the comparative absence of induration.

It is distinguished from *syphilis* by the absence of a specific history, by the pain upon deglutition, which is usually much more severe than in syphilitic ulceration, and by the pronounced constitutional symptoms. Again, when the ulcer is superficial, its worm eaten and irregular appearance, with the pallor of the adjacent surface and absence of induration, are distinguishing features; and when the ulceration is deep, the slight induration, if any, the irregular border of the ulcer—neither everted nor undermined and seldom sharply cut—and its comparatively

light color and granular, easily bleeding surface, will serve to distinguish it from the specific affection. Anti-syphilitic treatment, when vigorously pushed, usually causes rapid improvement in the specific disease, whereas it aggravates the tubercular affection.

Tubercular ulceration of the tonsil is to be distinguished from syphilitic ulceration by the following characteristics:

TUBERCULAR ULCERATION OF TONSIL.	SYPHILITIC ULCERATION OF TONSIL.
Little, if any, swelling.	Syphilitic history; induration.
Ulcer is usually superficial, not sharply defined, but may be deep and irregular.	Ulcer may be superficial or deep, edges well defined, may be undermined and everted: indurated base.
Pain, fever, rapid pulse, usually evidences of tuberculosis in other organs.	Usually little or no pain or fever, with normal pulse.

The deep tubercular ulcer is distinguished from *cancer* of the tonsils by the comparative absence of induration, which is usually pronounced in cancer even for several weeks or months before ulceration takes place; by the appearance of the edges of the ulcer, which are not everted in tuberculosis, and by the character of the surface of the ulcer, which is much cleaner in the tubercular disease than in cancer. The superficial ulcer of tuberculosis does not resemble the ulceration of malignant disease, and is not at all likely to be confounded with it. Pain usually occurs earlier in cancer than in tuberculosis, and is of a lancinating character and present for some weeks before ulceration takes place. In the early stages, constitutional symptoms are more marked in tuberculosis than in cancer, and the peculiar cachexia which develops in the later stages of carcinoma is not apparent in tuberculosis.

From cancer of the tonsil tubercular ulceration may be distinguished as follows:

TUBERCULAR ULCERATION OF TONSIL.	CANCER OF THE TONSIL.
Little, if any, swelling, with pallor instead of congestion of parts.	Parts swollen, indurated, and congested.
Usually ulcer is superficial and irregular, not sharply defined; whitish secretions.	Ulceration deep with abrupt borders and reddish or grayish white surface, fetid yellowish secretions, and fungous granulations.
Pain does not occur until after ulceration has commenced, and then is experienced especially on swallowing.	Pain marked before, as well as after, ulceration, and often sharp even when throat is at rest.
Fever, rapid pulse.	During first few months little if any fever or acceleration of pulse.
Usually no enlargement of cervical glands.	Enlarged cervical glands comparatively early in the disease.
Generally associated with pulmonary tuberculosis	Usually marked cachexia.

PROGNOSIS.—When the disease occurs primarily in the tonsil, many cases may be cured if taken early and given thorough and energetic

treatment; but when it develops subsequent to tuberculosis in other organs, little more than temporary relief of the disease can be hoped for.

Treatment.—Where the ulceration is secondary to general tuberculosis, constitutional treatment is of the most value. When the disease is primary, destruction of the affected tissues by scraping, and the application of lactic acid, or the galvano-cautery will occasionally be followed by perfect recovery. The part should be anæsthetized by cocaine, and it may then be scraped with the curette, and subsequently the lactic acid may be applied; but some cases do quite as well if the acid is thoroughly applied without previous scraping. Lactic acid is used for this purpose in strength varying from thirty per cent to one hundred per cent, and must be applied daily, and with thoroughness, for three or four days, and afterward less frequently for two or three weeks until the ulcer heals. As a rule, when the strong acid is employed, previous curetting is unnecessary. If the ulcer is not large and does not readily yield to the lactic acid treatment, the surface should be touched with the galvano-cautery, and subsequently lactic acid may be employed. For temporary relief, the parts may be sprayed with a two to four per cent solution of cocaine two or three times daily, or, in place of this, with a solution of morphine, or, better yet, the solution of morphine, carbolic acid, and tannic acid (Form. 93) recommended for tubercular laryngitis. Whatever local measures are adopted, all sources of irritation, especially tobacco smoking, should be removed. Constitutional treatment will be of the utmost importance.

CANCER OF THE TONSIL.

Cancer of the tonsil is a comparatively rare affection; but seven cases have come under my observation within the last five years, one being of the melanotic variety. One or both tonsils may be the seat of the disease which commences as a tumor in the substance of the tonsil and gradually and steadily extends, involving not only the whole gland, but the surrounding tissues. Ulceration usually occurs within five or six months from the commencement. The affection is attended by more or less constant pain, especially upon deglutition. This is frequently lancinating in character and radiates toward the ear. A pronounced cachexia is developed in some instances, during the later portion of the disease.

Diagnosis.—Cancer is to be distinguished from *hypertrophy of the tonsil* by the history, age of the patient, and course of the disease. Hypertrophy of the tonsil is a disease of early life, seldom observed after the thirtieth year, whereas cancer usually occurs after the age of forty. Hypertrophy of the tonsil is not attended by pain or constitutional symptoms, and is not followed by ulceration; furthermore unlike the malignant disease, it may last for years without seriously affecting the patient's general health.

Cancer is to be distinguished from hypertrophy of the tonsil as follows:

CANCER OF TONSIL.	HYPERTROPHY OF TONSIL.
Generally seen in those past middle life. Induration of surrounding tissues and congestion. Unilateral.	Generally seen in children and young adults. Hypertrophy with but little if any redness. Generally bilateral.
Late ulceration with reddish or grayish white surface, fetid secretions, fungous granulations.	No ulceration. Whitish deposit found in the lacunæ, no peculiar secretion.
Severe pain. Usually characteristic cachexia.	No pain. Frequently open mouth, dull eye, and stupid appearance, but no cachexia.

Cancer of the tonsil and syphilitic ulceration of the tonsil present the following differential diagnostic points:

CANCER OF TONSIL.	SYPHILITIC ULCERATION OF TONSIL.
Much swelling and induration, membrane darkly congested. Unilateral.	Comparatively little swelling and induration. Usually bilateral.
Late, ulceration with reddish or grayish white surface, profuse fetid secretions and fungous granulations.	Syphilitic history. Ulcer may be superficial or deep and undermined with indurated base and everted edges.
Lancinating pain, frequently marked before as well as after ulceration.	Little or no pain.
Usually marked cachexia.	No peculiar cachexia.

Cancer of the tonsil is distinguished from tubercular ulceration by the signs pointed out in considering the latter affection.

PROGNOSIS.—The disease usually runs its course in four to eight months, and probably is always fatal.

TREATMENT.—If seen early, the tumor should be removed by snare or galvano-cautery écraseur if possible; or later, if the growth is so large as seriously to interfere with respiration and deglutition, a similar procedure, though giving no hope of cure, may happily be followed by development of the tumor in some other direction less immediately dangerous or distressing. I have seen two cases in which removal of the cancerous tonsil was followed by perfect cicatrization and no subsequent trouble in the fauces, whereby the patient was saved from much of the distress which would otherwise have attended the later stage of the disease. Recently I have succeeded in retarding the growth for several months by frequent injections into the substance of the tumor of six to ten minims of a twenty-five to fifty per cent solution of lactic acid. After ulceration has taken place, surgical procedures are not likely to be of benefit, but detergent and antiseptic gargles and sprays may give temporary relief. The spray of carbolic and tannic acids with morphine (Form. 93) may be employed with no little satisfaction.

CHAPTER XXII.

DISEASES OF THE PHARYNX.

FOREIGN BODIES IN THE PHARYNX.

Foreign bodies of great variety have been found lodged or impacted in the pharynx, the most frequent being pieces of meat, fragments of bone, bristles, false teeth, buttons, coins, and needles or pins. Some people in whom there is impaired sensibility of the mucous membrane are specially predisposed to such lodgements. Large bodies generally lodge at the lower part of the pharynx or in the valleculæ between the base of the tongue and the epiglottis. Small or sharp pointed bodies may become fixed at any part of the throat, but they are more apt to lodge in the crypt of a tonsil or in the depressions between the gland and the pillars of the fauces.

Symptomatology.—Large bodies, unless speedily removed, may cause suffocation, but this usually ensues only when the substance has become impacted in the larynx or œsophagus. Hard or sharp substances cause pricking sensations or more or less severe pain, especially on deglutition, and, if they remain, inflammation and swelling soon follow. Even after the body has been extracted or has passed into the stomach the patient often complains of similar sensations for some time. Ulceration and even abscess may follow if the occluding substance remains for any length of time.

Diagnosis.—The diagnosis must be based upon the history given, and a careful inspection of the part; but it is to be remembered that sensations of pricking or actual pain are often felt even after the source of the trouble has been removed. Hysterical women especially, often insist for weeks or months that the foreign body remains, in spite of all assurances to the contrary. It is to be remembered also, that small bodies may actually remain for a long time in the crypt of a tonsil, or in the valleculæ, escaping observation.

Prognosis.—Occasionally immediate death from suffocation is caused by impaction of a foreign body in the pharynx. A fatal issue may likewise result from perforation of large arteries or other vital parts by ulceration, but often the body is either swallowed or expelled by the patient's own efforts. In many instances these substances remain several weeks, giving the patient much discomfort but not endangering life.

Treatment.—The foreign body should be removed as soon as practi-

cable. Unless seen at once, a most thorough and painstaking examination should be made, with the parts well under the influence of cocaine, and if nothing is found, a pledget of cotton should be brushed over every part with the hope of removing or bringing into view the possibly hidden object. Two bodies, especially in the case of fish bones, are not infrequently present in the same case; therefore if the unusual sensations persist, another examination should be made. As a rule, when the substance has been removed, the sensations disappear within a few hours, but sometimes they continue for a long time, usually as the result of an injury or small ulceration produced by the object. Generally such lesions yield speedily to the application of astringents or silver nitrate.

RETRO-PHARYNGEAL ABSCESS.

Retro-pharyngeal abscess, is a circumscribed suppuration of the submucous tissues of the pharynx, giving rise to swelling, in consequence of which there is interference with respiration and deglutition. The affection occurs most frequently in infants, having been observed even in the new born; but as a result of syphilis it is comparatively common in adults.

Anatomical and Pathological Characteristics.—The abscess may be located in the posterior wall of the naso-pharynx, the oro-pharynx, or the laryngo-pharynx. It may be developed near the median line, but in about three cases out of four it is confined to one side. The loose attachment of the mucous membrane by cellular tissue to the muscles beneath favors the formation of an abscess in this locality and allows pus to burrow easily in any direction, though it is inclined to gravitate downward. It sometimes extends even to the mediastinum.

I recollect one case in which the sinus, left after the abscess had opened, could be traced from the lower part of the oro-pharynx downward and backward ten inches.

The tumor formed by an abscess has a broad base, and the surface is smooth and usually not much discolored, especially when occurring in feeble children; though in adults an abscess resulting from syphilis, is often considerably congested.

Etiology.—The affection in children is usually idiopathic; yet if the ultimate cause could be traced, it would probably be found to depend in most instances upon an inherited scrofulous or syphilitic diathesis.

The exciting cause is often exposure to cold or to the prolonged heat of summer. It may follow simple acute pharyngitis, scarlatina, erysipelas, or tonsillitis. In adults it most commonly follows syphilitic disease of the cervical vertebræ. Some cases follow wounds inflicted by swallowing pins, bones, and other foreign substances. It is said to have followed stricture of the œsophagus, owing to the mechanical irritation attending forced deglutition.

SYMPTOMATOLOGY.—The affection usually comes on somewhat slowly, being first indicated by stiffness of the neck, with deep seated pain, which is referred to the palate when the abscess is far up, but is commonly felt deeper and may extend over the entire throat. Dyspnœa and dysphagia generally arise from mechanical obstruction, a result of the swelling. In children, convulsive symptoms often occur. According to Bokai, idiopathic abscess may develop in forty-eight hours, and secondary abscess in from seven to ten days; while that form proceeding from diseased bone is still more chronic in its course. Primarily, the patient usually experiences slight chilly sensations, but occasionally distinct rigors, with headache and slight rise of temperature. The pulse is usually weak and compressible, the head is thrown backward or inclined to one side, and sometimes there is painful tumefaction of the sides or front of the neck. If the abscess is located in the naso-pharynx, it interferes only with nasal respiration; if in the oro-pharynx, it does not affect respiration unless of large size. If, however, the disease should be situated in the laryngo-pharynx, a comparatively small abscess, by crowding the mucous membrane forward over the larynx, may speedily cause severe dyspnœa subject to frequent exacerbations and accompanied by cough and stertorous breathing. Abscess in the naso-pharynx gives the voice a nasal twang, and in the laryngo-pharynx may cause hoarseness or complete aphonia. Deglutition may be seriously disturbed by large abscesses in the naso-pharynx. Those located in the oro-pharynx or laryngo-pharynx are frequently attended by choking from the passage of fluids into the larynx. Abscesses in the naso-pharynx may escape observation on inspection, but ordinarily a dusky red tumor is visible which is doughy to the touch, yet somewhat elastic, but late in the affection may yield distinct fluctuation and have the appearance of pointing.

DIAGNOSIS.—A differentiation is here to be made from croup, œdema of the glottis, foreign bodies in the larynx, and cerebral or digestive disorders causing convulsions. Retro-pharyngeal abscess is distinguished from *œdema of the glottis* by inspection, which reveals the pharyngeal instead of laryngeal swelling; furthermore, by lifting the glottis, the dyspnœa is relieved in an abscess situated very low, but not in œdema.

Retro-pharyngeal abscess may be diagnosticated from œdema of the glottis by the following points of difference:

RETRO-PHARYNGEAL ABSCESS.	ŒDEMA OF THE GLOTTIS.
Pharyngeal swelling.	Laryngeal swelling.
May be located in oro-pharynx or laryngo-pharynx.	Located at glottis.
Lifting larynx relieves dyspnœa.	Lifting larynx does not relieve dyspnœa.
May interfere with nasal or obstruct laryngeal respiration.	Does not interfere with nasal respiration.
Rather insidious in its development.	Comes on suddenly.
Comparatively long duration.	Short duration.

Loss of voice or extreme hoarseness, symptoms not present in retro-pharyngeal abscess, attend *croup;* in croup there is no swelling or dysphagia, both of which are marked in retro-pharyngeal abscess.

It may be distinguished from *foreign bodies in the larynx* by the history and signs found by inspection and palpation, together with the quality of the voice.

Between retro-pharyngeal abscess and foreign bodies in the larynx, the following are the chief points of difference:

RETRO-PHARYNGEAL ABSCESS.	FOREIGN BODIES IN THE LARYNX.
Inspection reveals a tumor in the oro-pharynx or laryngo-pharynx. Rather slow development.	History of accident. Inspection and palpation may reveal presence of foreign body. Sudden obstruction to respiration or deglutition.
No hoarseness.	Voice usually much altered or lost.

It can only be diagnosed from *convulsive disorders* by a careful examination of the parts and detection of the tumor.

PROGNOSIS.—The affection usually terminates in recovery, idiopathic cases convalescing in from three to five days, and secondary cases in from seven to ten days, though fatal results are not infequent. Abscess due to spondylitis may last from three weeks to several months, and usually proves fatal in the end. In favorable cases the abscess opens spontaneously, unless sooner relieved, and with the escape of pus the more violent symptoms at once subside. Pus may burrow into the areolar tissue of the neck or into the ary-epiglottic folds and obstruct respiration even to suffocation; or it may escape into the larynx, with a similar result. Pus burrowing into the mediastinum may be discharged into the œsophagus or pleural cavity, an accident which is serious in either instance. Death has been known to result from ulceration of the internal carotid artery.

TREATMENT.—If the case is seen early, the abscess may sometimes be aborted by the continued sucking of ice, or by cold applications to the neck. When pus forms, it must be evacuated as soon as discovered. The incision should be made as near to the median line as possible, in order to avoid injury to the internal carotid artery; and as soon as the opening is made the patient's head should be thrown quickly forward to prevent the passage of pus into the larynx; or, better still, the operation may be done with the patient lying upon the abdomen, with the face extending slightly over the edge of the table. An ordinary bistoury, guarded to within a quarter of an inch of its point by a wrapping of cloth or adhesive plaster, is a good instrument for the purpose.

Tonics and supporting treatment are necessary; the syrup of the iodide of iron being a most useful remedy. The phosphates of iron and quinine, or the compound syrup of hypophosphites, may be given with benefit. Cod-liver oil is generally recommended, but should not be given unless it thoroughly agrees with the stomach. In children when

there is a tendency to convulsions, potassium bromide should be administered freely in the early stage.

TUMORS OF THE PHARYNX.

Non-malignant tumors, especially of the papillary variety, are comparatively frequent on the pillars of the fauces, tonsils, or posterior wall of the pharynx. These usually vary in size from three to ten millimetres in diameter. Large fibrous (Fig. 97) and fatty tumors are also sometimes seen. Small tumors cause but little inconvenience, except occasionally a troublesome cough or sensation as of a lump in the throat during the act of swallowing. When coming in contact with the epi-

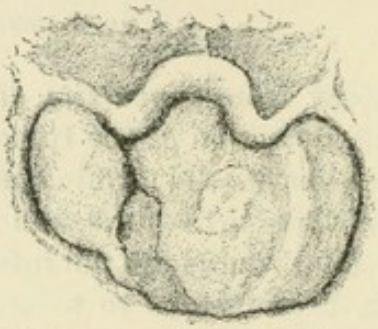

FIG. 97.—FIBROMA OF LARYNGO-PHARYNX. This was a large fibrous growth attached to the lower portion of the pharynx by a pedicle about half an inch in diameter. It was removed by the steel wire snare shown in speaking of nasal polypi. The base was subsequently cauterized with the galvano-cautery. No recurrence.

glottis or larynx, large growths may interfere with respiration and deglutition.

TREATMENT.—Small growths may be readily removed by the forceps, snare, or galvano-cautery. Large formations, if pedunculated, may be removed by the ordinary snare, the galvano-cautery écraseur, or écraseurs of other forms. In cases of large or vascular growths, care must be taken not to cause suffocation during their removal, and sometimes preliminary tracheotomy may be necessary.

CANCER OF THE PHARYNX.

Cancer is rare in the upper portion but not so infrequent at the lower part of the pharynx, where it joins the œsophagus.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—Cancers of the laryngo-pharynx usually first attack the posterior wall, and passing around the sides subsequently invade the larynx. They are more commonly of the epitheliomatous variety, but those of the pharyngo-oral cavity are very often of the scirrhous form.

SYMPTOMATOLOGY.—When the disease occurs in the pharyngo-oral space, it usually causes constant pain, often radiating toward the ear, and

is greatly aggravated by deglutition, especially after ulceration begins. The voice is indistinct, and there is profuse fetid expectoration.

When the tumor is situated in the lower part of the pharynx, it is not usually painful, although there may be difficulty in swallowing, and as the disease advances respiration becomes embarrassed. Cancer at the lower portion of the pharynx usually commences on the posterior wall near the level of the arytenoid cartilages, but gradually extends until it involves the larynx, causing tumefaction, hoarseness, and dyspnœa.

Scirrhous growth in the upper pharynx makes its appearance as a hard, imperfectly circumscribed mass beneath the mucous membrane, which in the early stages remains of normal appearance. As the disease progresses, induration extends and may involve the palate, pillars of the fauces, and even the posterior nares. Ulceration follows and extends over all the affected tissue, the ulcer presenting a reddish or grayish white surface covered with fetid secretion and here and there fungous granulations. The cervical glands at the angles of the jaw are usually involved, comparatively early in the disease. Cancer at the lower part of the pharynx usually appears first as a grayish white, fungous vegetation covered with secretion and surrounded by a zone of red and swollen mucous membrane. As it progresses, extensive ulceration may occur, and all the surrounding tissues may become indurated, but the cervical glands are not usually much enlarged.

Diagnosis.—Cancer of the pharynx is not apt to be mistaken for anything excepting syphilitic disease or fibrous tumors.

We may generally readily distinguish *fibrous growths* by their pedunculated form, firm consistence, and by absence of pain and ulceration.

As a rule, *syphilis* can be distinguished by the history, the less amount of pain, the presence of old cicatrices, or by the results of medication. Under the influence of potassium iodide given freely, the syphilitic patient usually increases in weight and improves in general health, whereas in a person suffering from cancer, although this treatment may appear to be beneficial for a few days, the weight does not increase, and it is soon apparent that the general condition is growing worse.

Treatment.—Palliative measures only can be adopted. Opiates, when well borne, may be given internally in sufficient quantities to relieve pain. The spray of morphine, carbolic acid, and tannic acid (Form. 93) will be found beneficial from its property of mitigating the pain, modifying the offensive odor of the discharge, and exerting some restraining influence upon the ulceration or subjacent inflammation. More than this cannot be accomplished in the present state of our knowledge. When deglutition becomes difficult, food may be administered per rectum or by the œsophageal tube.

NEUROSES OF THE PHARYNX.

ANÆSTHESIA OF THE PHARYNX.

Anæsthesia of the pharynx, a rare affection, is characterized by the patient's inability to feel the bolus of food, some portions of which are liable to remain in the pharynx and subsequently to be drawn into the larynx during inspiration.

Etiology.—Transient local anæsthesia is produced by the internal administration of morphine or the bromides in large quantity, or by local or general anæsthetics. As found in practice, this affection is usually a sequel of diphtheria or the result of progressive bulbar paralysis. It sometimes occurs in hysteria, and is present in some cases of typhus fever, cholera, and the general paralysis of the insane. It also occasionally attends epilepsy. Owing to the liability of portions of food to be drawn into the larynx, patients come to dread taking anything but liquids or semi-solids.

Prognosis.—Following diphtheria, or when associated with hysteria or acute disease, the prognosis is favorable, but in other instances recovery cannot be expected.

Treatment.—When well marked, food should be given through the œsophageal tube. In remediable cases, tonics and galvanism are indicated, but especially the internal administration of strychnine in large doses. When faithfully followed out, promising results may be expected. Strychnine should be given in small but steadily increasing doses and carried to the point of physiological toleration indicated by mild muscular spasms. The dose should then be diminished, but may be again increased, after a few days, to an amount just short of that which caused the spasms; this dose may be continued with benefit for days or weeks.

HYPERÆSTHESIA OF THE PHARYNX.

Hyperæsthesia of the pharynx is of common occurrence, but can hardly be called a disease. It is often associated with acute inflammation of the pharynx, and is frequently found in persons given to the excessive use of tobacco or alcoholic stimulants. It may be produced by elongation of the uvula, and it is one of the manifestations of hysteria, but it is also sometimes present in persons otherwise in perfect health. In marked cases the sensitiveness may be so great as to interfere somewhat with deglutition of solids, so that patients prefer to take liquid or semi-solid food; but usually the condition causes no inconvenience excepting when the physician attempts to examine the fauces or introduce the throat mirror. Hyperæsthesia attending inflammation may be relieved by sedative troches of slippery elm, althea, lactucarium, or opium.

The internal administration of from ten to twenty grain doses of potassium bromide three or four times daily, and the inhalation from a steam atomizer of a solution of the same, gr. xx.–xxx. ad ℥ i., will also be found beneficial; a five per cent solution of carbolic acid will also give a good result, and may sometimes be particularly beneficial when ulceration is present. To relieve the hyperæsthesia which interferes with laryngoscopic examination, the sucking of ice for fifteen or twenty minutes will often answer an excellent purpose, but it may usually be accomplished more speedily by spraying the pharynx five or six times with a ten per cent solution of cocaine.

PARÆSTHESIA OF THE PHARYNX.

Paræsthesia of the pharynx, a common affection, is characterized chiefly by the presence of sensations of heat or cold, pricking, or swelling; or the patient may imagine he feels in the throat some foreign substance like a hair, bit of straw, bristle, or sliver of toothpick.

ETIOLOGY.—The affection often follows removal of foreign bodies from the fauces, but not infrequently it occurs in hysterical women without definite exciting causes; it is often associated with a varicose condition of the veins or enlargement of glands at the base of the tongue, or with follicular pharyngitis. It is sometimes kept up by a small ulcer which may have been caused by injury from a foreign body. The principal objective conditions found are, enlargement of the follicles in the pharynx or of the glands or veins at the base of the tongue.

PROGNOSIS.—The patient should always be assured that it is not a serious disorder, for frequently he is tormented with fears of cancer; but he must also be told that the condition, in spite of all treatment, may remain for many months, though it is likely eventually to subside.

TREATMENT.—Enlarged follicles upon the pharyngeal wall, or enlarged glands or veins at the base of the tongue, should be destroyed with the galvano-cautery. If this does not relieve the sensations, the application two or three times daily of a spray of morphine, carbolic acid, and tannic acid (Form. 93), and the internal administration of the bromides, with nerve tonics, is likely to be most beneficial. The sensations are frequently associated with rheumatism; under such conditions, anti-rheumatic remedies should be administered.

NEURALGIA OF THE PHARYNX may be characterized by the same symptoms as paræsthesia, but more commonly by actual pain. It is often due to the same conditions as neuralgia in other portions of the body and frequently results from the rheumatic diathesis, when it might properly be termed chronic rheumatic sore throat. The treatment consists of applications of sedative, astringent, or stimulating sprays to the throat, combined with the internal administration of potassium bromide and nerve tonics.

SPASM OF THE PHARYNX.

Spasm of the pharynx is a rare affection except as associated with acute inflammation of the fauces or hydrophobia, and it is usually of the tonic variety. The affection is sometimes associated with spasm of the œsophagus, and is characterized by sudden ejectment of fluid upon attempted deglutition.

ETIOLOGY.—Pharyngeal spasm may be due to acute pharyngitis, tetanus, hydrophobia, or certain disorders of the brain. It is occasionally a reflex phenomenon occurring in the course of chronic pharyngitis, and in a mild form may result from swallowing food which is imperfectly masticated. It may be purely a neurosis, as observed in hysterical persons.

SYMPTOMATOLOGY.—The spasm is marked by sudden ejectment of food on attempted deglutition. It may occur only at certain times of the day; the patient perhaps being able to eat breakfast and dinner easily, but at supper he may find that he is unable to swallow. Sometimes it occurs only after taking certain kinds of food. It may come at the beginning of the meal, or later after considerable food has been taken; it is always a source of great distress to both the patient and his friends. Often, while eating naturally, the patient is suddenly compelled to rush from the table, or, without warning, the food is forcibly ejected from his mouth.

DIAGNOSIS.—The affection is to be distinguished from stricture or paralysis of the œsophagus and from paralysis of the pharynx.

Solid or liquid foods are swallowed with more or less difficulty in *stricture of the œsophagus*, according to the degree of stenosis, but the bolus is not, as a rule, thrown out forcibly, though sometimes this occurs. In such cases persistent difficulty in the passage of an œsophageal bougie will settle the diagnosis.

Dysphagia is present in *paralysis of the pharynx* or *œsophagus*, but the food is not suddenly expelled from the mouth. In the spasmodic affection, according to Lennox Browne (Diseases of the Throat, second edition), an important diagnostic sign in protracted cases is obtained by placing the fingers over the masseter and temporal regions during mastication, when it will be found that the muscles are more or less atrophied from want of use, a condition not obtained in the disease under consideration.

PROGNOSIS.—The affection may last for weeks or months, and is sometimes so serious a malady as to necessitate the administration of food per rectum.

TREATMENT.—The treatment consists in the administration of tonics and nerve sedatives, such, for example, as quinine, zinc valerianate, arsenious acid, potassium bromide, camphor monobromide, and asafœtida. If associated with spasm of the œsophagus, the occasional passage of an œsophageal bougie will usually be found most beneficial.

PARALYSIS OF THE PHARYNX.

Paralysis of one or more of the constrictor muscles of the pharynx may be unilateral or bilateral, partial or complete. It is characterized by dysphagia and the accumulation of saliva which the patient is unable to swallow and which therefore drips from the mouth.

ETIOLOGY.—The paralysis may be idiopathic, but the most common cause is disease of the medulla involving the origin of the vagus and glosso-pharyngeal nerves. It may also result from other cerebral diseases. It sometimes follows syphilis, cerebro-spinal meningitis, or sunstroke, or accompanies facial paralysis, or diphtheritic paralysis of the œsophagus. It sometimes occurs in the course of acute febrile diseases, and is then commonly one of the precursors of death.

SYMPTOMATOLOGY.—Among the most clearly characteristic symptoms is difficulty of swallowing, even of the saliva, which constantly collects and streams from the mouth. Liquids also are often taken with great difficulty on account of running into the trachea and exciting cough and spasm of the glottis. This is caused by associated paralysis of the depressors of the epiglottis. Deglutition is generally accompanied by contortions of the neck and face, from the efforts made to assist the passage of food. In chronic disease of the brain and spinal cord these symptoms sometimes occur long before the fatal termination. In the paralysis associated with facial paralysis, the uvula usually deviates toward the healthy side, and the palate scarcely moves on phonation. Paralysis of the pharynx following diphtheria usually comes on ten or fifteen days after convalescence begins, and is characterized by dysphagia, especially on attempts to swallow fluid, inability to expectorate, and a peculiar nasal timbre of the voice due to paresis of the palate, with non-closure of the passage to the naso-pharynx. The sense of taste is obtunded, and the velum is usually relaxed upon one side. Paralysis of the pharynx is frequently associated with paresis of the œsophagus, in which condition solids are swallowed more easily than fluids, and large boluses than small.

Paralysis of the pharynx is often one of the early symptoms of progressive bulbar paralysis. In this affection loss of motion is usually first manifested in the tongue, lips, and palate, causing at first indistinctness and slowness of speech, but later, difficulty in mastication and finally dysphagia, with more or less dyspnœa due to spasm of the glottis caused by entrance into the larynx of liquid or solid food. The voice is weak and often aphonic, and there is inability to pronounce the labials b, w, m, p, or the dentals f, d, v, n, and th.

DIAGNOSIS.—The diagnosis depends upon the history, symptoms, and signs just described. The continuous character of the paralysis distinguishes it from spasm of the pharynx.

PROGNOSIS.—When due to temporary causes, when following diph-

theria or other acute diseases, or when associated with facial paralysis, recovery may be expected; but cases dependent upon progressive bulbar paralysis always end in death.

TREATMENT.—If food cannot be swallowed, it must be administered by means of the œsophageal tube or per rectum. Internally iron, quinine, arsenious acid and strychnine, especially the latter, are indicated in most cases, and sometimes considerable benefit will be obtained by change of air and scene. Here, as in anæsthesia of the pharynx, the most pronounced benefit will usually be obtained from strychnine, in large and gradually increasing doses.

SCALDS AND BURNS OF THE PHARYNX.

Injuries by heat are not uncommon, especially among children of the poor, in whom they frequently follow inhalation of steam from the teapot. They are sometimes caused in adults, by the inhalation of steam, flame, or hot air, as in burning vessels or buildings. In such cases the tongue, palate, and often the nares and œsophagus are similarly affected.

SYMPTOMATOLOGY.—There is acute pain and distress in the throat, with quickened pulse and more or less fever. Usually the larynx is involved, and swelling and dyspnœa are speedy results. Cohen states that when smoke has been inhaled, the sputum is blackish in color for several days ("Diseases of the Throat"). Dysphagia is always present.

If seen early, the affected parts are of a whitish color due to burning of the mucous membrane, and shortly afterward patches of the membrane are found to be destroyed, and severe inflammation with marked swelling ensues.

DIAGNOSIS.—The diagnosis may be easily made from the history, symptoms, and appearance of the parts.

PROGNOSIS.—In many instances the accident is speedily fatal, and in all cases where the burn is at all severe the prognosis is very grave. If the patient lives long enough, sloughing and excessive suppuration occur, and vicious adhesions, together with chronic laryngitis and stenosis of the larynx and trachea, are apt to follow.

TREATMENT.—Cold compresses, with sucking of ice and soothing applications, should be employed, mucilaginous drinks being given if they can be swallowed. Nourishment must be given by enemata, when deglutition is impossible. If dyspnœa supervene, tracheotomy must be promptly performed to prevent suffocation. Unfortunately, however, in these cases the operation does not often prevent a fatal issue.

SWALLOWING THE TONGUE.

The so called swallowing the tongue is an extremely rare accident. Most of the cases recorded seem to have occurred in children suffering from whooping cough. A case which I reported to the American

Laryngological Society at its annual meeting, 1880, occurred in a lady suffering from hysteria. It was characterized by a spasmodic action of the hyo-glossus and probably also the stylo-glossus muscles, which drew the tongue into the pharynx in such a position as to prevent respiration. There was no cough. The accident may prove speedily fatal.

TREATMENT.—The tongue should at once be drawn forward to prevent suffocation. Subsequently the primary disease should receive appropriate treatment.

DISEASES OF THE VALLECULÆ AND PYRIFORM SINUSES.

Ulceration of the valleculæ at the base of the tongue, or of the pyriform sinuses of the larynx, occasionally occurs from injury in swallowing bits of bone or food, and sometimes from inflammation of the glandular structure. Ulcers in either position give rise to pricking sensations and pain upon deglutition, and those in the pyriform sinuses are attended also by cough. Upon inspection with the laryngoscope, the valleculæ are commonly found filled with secretions, which must be wiped away before the cause of the trouble can be discovered, and it is usually necessary to anæsthetize the parts thoroughly with cocaine in order to make a complete examination.

TREATMENT.—If foreign bodies are found, their removal usually gives prompt relief. If ulcers exist, they are generally speedily cured by touching them once or twice with a solution of silver nitrate, gr. lx. ad ℥ i.

CHAPTER XXIII.

DISEASES OF THE LARYNX.

ACUTE LARYNGITIS.

Synonyms.—Acute catarrhal laryngitis, cynanche laryngea, angina laryngea, angina epiglottidea, inflammation of the larynx.

Acute laryngitis is a simple catarrhal inflammation of the mucous membrane of the larynx, characterized by pain, dyspnœa, dysphonia or aphonia, stridulous breathing, and cough.

Anatomical and Pathological Characteristics.—In mild cases there is congestion with slight swelling of the mucous membrane, either uniformly or in patches; the latter are more commonly found at the posterior end of the vocal cords, the posterior commissure, or on the ventricular band. In more severe cases the mucous membrane is œdematous and deeply congested, the epiglottis is thickened and flaccid, the ary-epiglottic folds are swollen into thick, pyriform bodies, and the ventricular bands may be so swollen as to overlap and completely hide the cords.

Etiology.—Indoor occupation, malnutrition or defective excretion, and excessive use of alcoholic stimulants or tobacco, are among the principal predisposing causes. Certain diseases, as measles, scarlatina, and variola, also favor its occurrence. Among the exciting causes are exposure to irritating vapors or drugs, to wet and cold, or to draughts of air, also violent cough and excessive use of the voice, especially in the open air. It is also frequently due to extension of inflammation from the neighboring mucous membrane.

Symptomatology.—The affection usually comes on insidiously, preceded by a mild rhinitis, pharyngitis, or bronchitis, and is finally ushered in by slight rigors or chilly sensations. In severe cases there is sometimes a pronounced chill followed by rapid development of the symptoms. Sensations of dryness, roughness, or tickling in the larynx are early experienced, and these may be followed by pain, which is aggravated by coughing or speaking. As the disease progresses, there is a feeling of constriction, the tendency to cough and clear the throat becomes more pronounced, and the swelling may give rise to sensation as of a foreign body. The pain is aggravated by deglutition, and tenderness is usually elicited by palpation. At first respiration is not affected, but as soon as swelling occurs dyspnœa comes on, and in severe cases becomes very

distressing. The patient cannot lie down, is very restless and makes frantic efforts for breath. At the commencement of the attack, the face is flushed and the eyes are bright, but, as dyspnœa develops, the face becomes livid and anxious, or of an ashy hue, and the eyes protrude as in strangulation. The skin, which is at first hot, particularly in children, becomes cold and clammy; the pulse, at first full and bounding, grows weak and irregular, and the temperature rises to 102°, 103°, or 104° F. The voice, in the beginning hoarse and shrill, later may be weak or entirely lost. The cough, at first resonant and clear, becomes convulsive, brazen or croupy in character, and there is a slight expectoration of tenacious, glairy mucus until toward the end of the disease, when the secretions become muco-purulent in character, and profuse when the bronchi are also involved. Children suffering from acute laryngitis are prone to croupy attacks at night, probably due to the collection of secretions about the glottis. The tongue is usually white, furred, and red at the

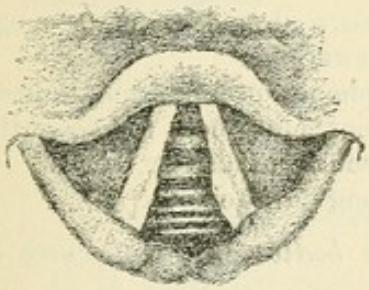

FIG. 98.—SUPERFICIAL ULCERS OF VOCAL CORDS. Herpetic; covered with a thin whitish false membrane.

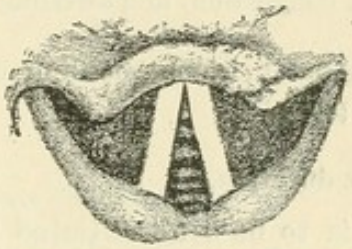

FIG. 99.—SUPERFICIAL ULCERATION OF EPIGLOTTIS. Herpetic; covered with a thin whitish false membrane.

tip. Upon laryngoscopic examination, the congestion and swelling are readily detected, and occasionally small erosions, particularly at the vocal processes, are observed. In rare instances, superficial ulcerations of an herpetic character are seen, though these are not apt to be associated with much congestion and swelling of the parts (Figs. 98 and 99). As a result of the swelling, there is frequently paresis of the arytenoideus or of the thyro-arytenoid muscles, giving rise to the gaping of the cords (Figs. 182, 183). Occasionally, even before hyperæmia occurs, the patient becomes hoarse, and upon examination paresis is found to be present.

A mild form of laryngitis frequently attends asthma or hay fever.

DIAGNOSIS.—The disease is to be distinguished from laryngismus stridulus, true croup, paralysis of the vocal cords, and foreign bodies in the larynx. The chief features in the diagnosis are hoarseness and dryness and pain in the larynx, with hyperæmia and swelling. It is distinguished from *laryngismus stridulus* by coming on more slowly and being attended by chills, fever, congestion, and swelling of the parts.

The following are the differential points peculiar to acute laryngitis and laryngismus stridulus:

Acute laryngitis.	Laryngismus stridulus.
Congestion and swelling of mucous membrane.	No congestion or swelling of mucous membrane.
Fever.	No fever.
Generally pain.	No pain.
Gradual accession, and of several days duration.	Sudden in its onset and short in duration. Attack usually at night; may not be repeated.

It is distinguished from *true croup* by the age of the patient and by the greater amount of pain, congestion, and swelling; by the scanty tenacious sputum and absence of false membrane. When occurring in young children, it is not always possible to make an accurate diagnosis.

Acute laryngitis is distinguished from *paralysis of the vocal cords* by the pain, congestion, and swelling, which are not present in the latter disease; and by the other points presented in the following table:

Acute laryngitis.	Paralysis of the vocal cords.
Pain, congestion, and swelling.	Entire absence of pain, congestion, and swelling.
Voice harsh; sometimes aphonia for a brief period.	Aphonia pronounced, especially if patient is fatigued; is present throughout course of disease.
Short duration.	Long duration.

It is to be differentiated from *foreign bodies in the larynx* by the history and by laryngoscopic examination.

Prognosis.—Mild cases usually pass off in four or five days, and others in most instances soon yield to suitable remedies; but occasionally the swelling and consequent obstruction of the glottis are so great as to cause death. Neglected cases, or those in which the patient again exposes himself before the inflammation has entirely subsided, are liable to end in chronic laryngitis.

Treatment.—Cold compresses renewed every half-hour or hour are found most effective in the beginning of the disease. If these fail, sedative vapors or inhalations of steam impregnated with opium, belladonna, or lupulin (Form. 55, 56, 57), together with large doses of potassium bromide and warm compresses, will be found more effective. The disease is sometimes aborted by the early administration of ten grain doses of Dover's powder or quinine, or small and frequently repeated doses of the tincture of aconite or opium, one minim every half-hour or hour for ten or twelve hours, or until the physiological effects are obtained, and subsequently less often. Saline cathartics to keep the bowels open are usually desirable unless the affection is aborted within twenty-four hours. In all cases in any degree severe, the patient should remain in the house in a warm, moist atmosphere, and refrain from using the voice. Toward the close of the disease, the application of mild astringent sprays (Form. 88, 90, 94) once or twice daily will be found very

beneficial. Sometimes compressed tablets of potassium chlorate are also useful. If œdema occurs so as seriously to impede the respiration, scarification or rupture of the swollen membrane is indicated, though the necessity for it may sometimes be removed by administration of the fluid extract of jaborandi, or its active principle pilocarpine, in sufficient quantity to excite profuse diaphoresis and salivation. Scarification is best practised by means of the guarded laryngeal lancet (Fig. 100). The mucous membrane may sometimes be ruptured by the finger nail, the edge of which has been roughened for the purpose. Severe cases may require intubation or tracheotomy. In children where there is doubt as to the diagnosis, the disease should be managed in the same way as true

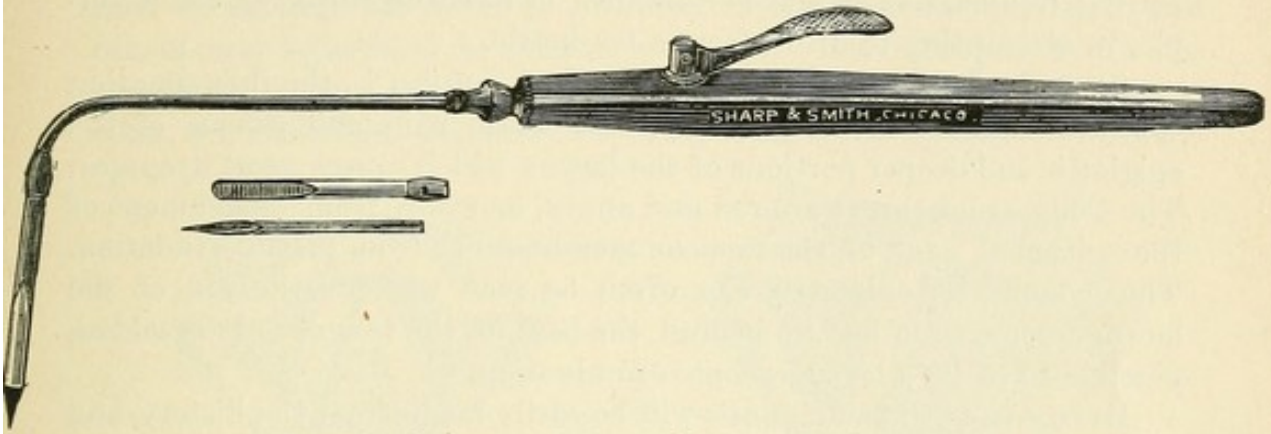

FIG. 100.—MACKENZIE'S LARYNGEAL LANCET (3-5 ordinary size).

croup. It is generally best in the beginning to give a free calomel purge and follow this by the treatment suitable for true croup, intubation or tracheotomy being performed as soon as there is serious interference with respiration.

SUBACUTE LARYNGITIS.

Subacute laryngitis is a mild form, usually present in what is known as an ordinary cold. It is characterized by dryness or tickling sensations in the larynx, with slight pain, hoarseness, and inclination to cough, with but little or no fever. The cough is laryngeal, hacking, and more or less paroxysmal, and the expectoration usually consists of a small amount of clear, tenacious mucus. The causes are the same as those of acute laryngitis, operating in a milder degree. Upon inspection of the larynx, more or less congestion is observed, but frequently none except along the edges of the vocal cords at their posterior extremities.

PROGNOSIS.—The prognosis is favorable, and often the only treatment needed is care as to exposure, and confinement to the house for one or two days. Even this precaution is neglected by most patients, yet the great majority recover within five or ten days.

TREATMENT.—Local and internal treatment suitable for mild cases of acute laryngitis are appropriate in the subacute form, and mild astrin-

gent sprays are especially indicated in the latter portion of the attack if the patient suffers from hoarseness, tickling in the larynx, or a tendency to cough. Unless the patient is careful not again to expose himself, there is great liability to recurrence of the attack, and, if this is repeated a few times, chronic laryngitis is the probable sequel.

TRAUMATIC LARYNGITIS.

Traumatic laryngitis may result from the irritation caused by foreign bodies, from the inhalation of irritating gases, or from mechanical injury in operations; but most commonly it occurs in children from swallowing boiling liquids, strong acids or alkalies, or inhaling steam, as, for example, in attempting to drink from a tea-kettle.

SYMPTOMATOLOGY.—After the accident causing it, the inflammation comes on almost instantaneously, with acute pain, and œdema of the epiglottis and deeper portions of the larynx which causes great dyspnœa. The tongue and throat are red and angry, or white from detachment of the epithelial layer of the mucous membrane or from plastic exudation. The œdematous epiglottis can often be seen without the aid of the laryngoscope, standing up behind the base of the tongue. It is seldom possible to make a laryngoscopic examination.

DIAGNOSIS.—The diagnosis will be easily made from the history, and from the appearance of the mouth and fauces.

PROGNOSIS.—The prognosis depends upon the extent of the injury, but is commonly grave, especially when the disease results from scalds or burns.

TREATMENT.—The affection can sometimes be aborted by painting the parts with a strong solution of silver nitrate. However, this application is not devoid of danger from spasm of the glottis. Full doses of jaborandi may be tried. Constant applications of ice to the neck, and the sucking of ice, should be practised; or, in its stead, hot applications or inhalations of steam. The parts usually become œdematous in spite of these measures, and then scarification or tracheotomy must be promptly performed.

CHRONIC LARYNGITIS.

Synonyms.—Chronic catarrh of the larynx, laryngitis chronica.

The chronic inflammation of the larynx indicated by more or less hoarseness and cough with a frequent inclination to clear the throat is most common in male adults.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—There is hyperæmia of the parts, which may be general or circumscribed, shading off gradually into the color of the surrounding tissue. Usually there is but little swelling, occasionally small blood vessels upon the epiglottis or the vocal cords are enlarged, and in rare instances nodular excrescences

are met with. Not infrequently slight erosions are noticed, particularly between the arytenoid cartilages, but often these consist simply of destruction of the epithelium and cannot be distinguished except by the absence of the peculiar glistening appearance characteristic of healthy mucous membrane. Exceptionally small ulcers occur upon the vocal cords at the vocal processes (Fig. 101).

In unusual instances hypertrophy of the soft tissues exists.

ETIOLOGY.—The disease is occasionally primary, but more frequently it is the result of repeated attacks of acute or subacute inflammation, and therefore is generally due to like causes. The excessive use of tobacco, chronic alcoholism, and the constant inhalation of irritating dust or particles of metal as observed in metal-grinders, millers, and others, may sometimes be classed as causes. Not infrequently the disease follows from over-use of the voice, especially in the open air, or when the individual is already suffering from acute or subacute inflam-

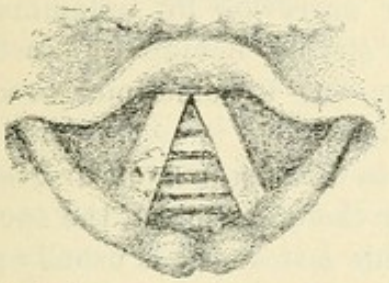

FIG. 101.—CATARRHAL ULCER OF THE VOCAL CORD.

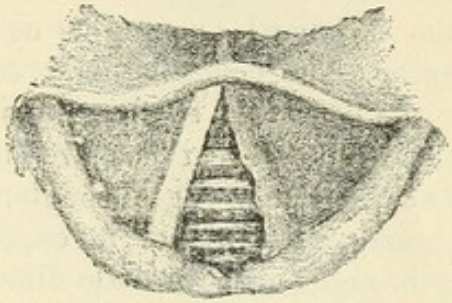

FIG. 102.—CHRONIC CATARRHAL LARYNGITIS WITH DEFORMITY.

mation of the organ. The disease sometimes is a sequel of measles, scarlatina or other eruptive fevers, and in rare instances it results from eczema.

All long continued affections of the larynx, as cancer, lupus, or polypoid growths, may finally set up chronic inflammation. Phthisis and syphilis are frequent causes.

SYMPTOMATOLOGY.—In some cases the symptoms are not marked, and the patient only complains of something wrong in the larynx, with hoarseness and more or less dryness of the throat, especially after exposure. These patients often expectorate small pellets of thickened mucus. Sometimes they are suddenly startled in the night or at other times with a sense of suffocation due to spasm of the glottis, and attended by a feeling as though a crumb of bread had dropped upon the vocal cords. In mild cases there are no constitutional symptoms, but in those more severe there may be emaciation, fever, and night sweats, as results of the disturbance caused by the frequent cough. Among the common sensations experienced, are pricking or burning in the throat and a frequent desire to clear it. Varying degrees of hoarseness are observed; in some this symptom is noticed during ordinary conversation, in others only when singing, and in still others the singing voice seems natural, al-

though the voice is very hoarse in its ordinary use. In others difficulty is noticed only on attempts at shouting. Sometimes early in the morning the patient is very hoarse, but after two or three hours the voice becomes nearly normal as a result of physiological stimulation of the circulation in the parts. In these cases, the voice usually again becomes hoarse after a few hours. In some instances taking of food greatly clears the voice. In some the tones are clear during quiet conversation, and hoarseness is only experienced after talking or singing for a half-hour or more. In nearly all cases, however, the voice eventually becomes continuously strained. Persons suffering from this disease commonly tire easily on attempting to talk for any length of time, and with the fatigue the voice usually becomes more and more harsh and unnatural.

The fatigue resulting from exertion of the parts may be confined to the larynx, or it may be general, so that even strong subjects suffering from laryngitis may become much exhausted after using the voice for half an hour. Respiration is not affected, barring those instances wherein the laryngeal opening is considerably narrowed by inflammatory changes. The cough usually consists of simple hemming efforts to clear the larynx of small pellets of mucus, but it sometimes becomes frequent and severe, especially during the night.

Two kinds of laryngeal cough may occur in this disease: one dry, harsh, and brassy, with little or no expectoration; the other moist, the sputum being brought up with little difficulty. This latter type is usually associated with chronic bronchitis, in which case the expectoration may be abundant. As a rule, the sputum consists of small masses of mucus, grayish in color from being more or less tinged with dust; after a time it may become yellowish or brownish. The tongue is usually thick and coated at its base with a yellowish pasty fur. The mucous membrane of the fauces and pharynx is generally relaxed and more or less congested, and in many instances enlarged follicles may be seen upon the pharyngeal wall or base of the tongue. The general health is not usually impaired, the appetite remains good, but constipation is common and occasionally there are symptoms of dyspepsia. The mucous membrane of the larynx is more or less red and slightly swollen either uniformly or in patches; the latter condition is more apt to be noticed on the vocal cords and the arytenoids, but may involve the ventricular bands or epiglottis.

Sometimes nodular excrescences exist, varying in size from one to five millimetres in diameter; these give the larynx a granular appearance. This is especially noticeable upon the vocal cords in the condition known as trachoma. In some cases slight erosions may be seen, being more apparent by the loss of that "peculiar sheen" which is seen upon the healthy mucous membrane than by a visible depression. This condition is most likely to occur on the inner surfaces of the arytenoid cartilages just above the posterior ends of the vocal cords. The

laryngeal mucous membrane is sometimes dry, but, as a rule, the secretions are somewhat increased. Often flakes of more or less discolored mucus may be seen adhering to the cords or slightly sticking them to each other, and in other instances a less tenacious and thinner secretion is seen in a very thin layer upon the cords and other portions of the larynx, or stretching between the vocal cords in respiration, but, as be-

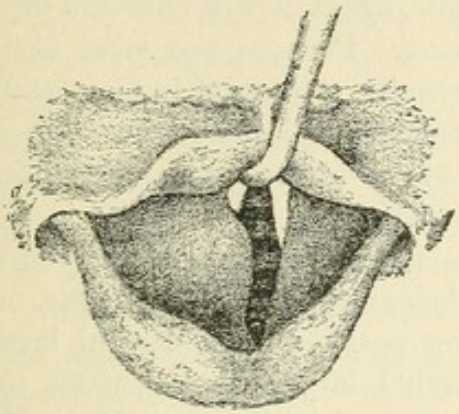

FIG. 103.—CHRONIC CATARRHAL LARYNGITIS.

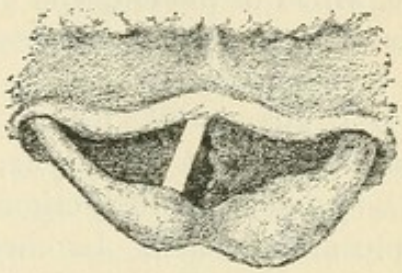

FIG. 104.—CATARRHAL LARYNGITIS WITH DEFORMITY SIMULATING CANCER.

fore mentioned, the secretion is never abundant if only the larynx is involved. In many examples of the disease the tracheal mucous membrane is also congested, and often secretions may be seen collected upon its surface. There is as a rule comparatively little thickening of the laryngeal tissues, excepting the vocal cords, which may be swollen to two or three times their normal size—but the epiglottis or one or both arytenoids may be thickened from twenty to fifty per cent.

In unusual instances all the soft parts are hypertrophied, and exceptionally the changes are so great as to simulate malignant disease, or aggravated forms of syphilitic laryngitis. It has been stated that the larynx sometimes appears to be dilated, but I have not seen this condition.

Subglottic hypertrophy, consisting of a grayish welt just below the vocal cord, is occasionally seen, and it is probable that the same condition

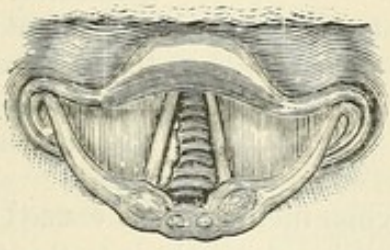

FIG. 105.—SLIGHT SUBGLOTTIC ŒDEMA IN A PHTHISICAL PATIENT.

at the outer portion of the under surface of the cord may account for some of those cases of hoarseness where the physical condition of the larynx appears nearly or quite normal. This condition might easily escape observation because of its location beneath the cord. Sluggish movement of the cords or want of proper approximation is not uncom-

monly the result of mechanical interference with contraction of the laryngeal muscles, or thickening and irregularities of the mucous membrane. The glands at the base of the tongue are quite often enlarged, and sometimes they seem to stand in a causative relation to the laryngitis. In some instances a varicose condition of the veins may be noticed in the same locality. The pharyngeal wall may be normal or it may be relaxed and studded with enlarged follicles, while, again, it will be found dry and glazed, or partially coated with secretion. Perhaps the most constant changes which accompany chronic laryngitis are found in the nasal cavities, which in the majority of cases are more or less obstructed by exostosis or enchondrosis of the septum, or by hypertrophy or swelling of the turbinated bodies.

DIAGNOSIS.—The disease may be mistaken for paralysis of the vocal cords, œdema of the larynx, tubercular or syphilitic laryngitis, or for cancer; a definite distinction only being possible after careful laryngoscopic examination. In chronic catarrhal laryngitis the parts nearly always remain of normal contour, and are but little swollen, though more or less congested; ulceration is rare.

Constant hoarseness is caused by *paralysis of the vocal cords*, and dysphonia is especially pronounced when the patient is fatigued; therefore the voice is usually better in the early morning than in the evening. In simple catarrhal inflammation, the hoarseness is generally worse early in the morning. In paralysis, there is no congestion or swelling, but there is marked loss of movement of one or both cords, in which respect it differs from laryngitis.

Chronic laryngitis is to be distinguished from paralysis of the vocal cords by the following characteristics:

CHRONIC CATARRHAL LARYNGITIS.	PARALYSIS OF THE VOCAL CORDS.
Parts slightly thickened. More or less congestion.	No swelling or congestion.
Slight loss of movement of cords.	Marked loss of movement of one or both cords.
Hoarseness usually most marked in the morning.	Constant hoarseness; usually less in the morning.
	Dysphonia especially pronounced when patient is fatigued.

Swelling of the mucous membrane is caused by *œdema of the larynx*, the parts generally appearing from three to five times as large as normal. The mucous membrane is usually pale and has a semi-transparent appearance. Sometimes it may be considerably congested, but in all cases it appears as though serum would flow out if the membrane were punctured. In these respects chronic laryngitis is quite different.

From œdema of the larynx, chronic laryngitis is to be distinguished as follows:

CHRONIC CATARRHAL LARYNGITIS.	ŒDEMA OF THE LARYNX.
Prolonged course; slight swelling of parts, with more or less redness of membrane.	Short duration; great swelling of parts, with change of color; membrane pale, semi-transparent.
Respiration normal.	Labored respiration.

Simple catarrhal inflammation is distinguished from *tubercular laryngitis* by the history, by the constitutional symptoms and by the color and contour of the parts. In the early stage of tubercular laryngitis there is frequently anæmia of the organ and sometimes of the soft palate, instead of congestion as in chronic catarrhal inflammation. In some cases, however, the color in the two diseases is not very dissimilar; but in the tubercular affection superficial or occasionally deep ulceration of the vocal cords and ventricular bands or of the posterior commissure, or the epiglottis, are soon discoverable, which are not observed in the simple catarrhal disease. In the later stage of most cases of tubercular laryngitis there is peculiar pyriform swelling of the arytenoids and ary-epiglottic folds, the parts being paler than in health, three or four times their ordinary thickness, and having an appearance of solidity instead of that of œdema. Ulceration is usually associated with this condition, or, if not present at first, it speedily follows. The loss of strength, rapid pulse, fever, emaciation, and night sweats of tubercular laryngitis are very seldom found in the simple catarrhal inflammation. In the tubercular affection pain is a common and distressing symptom, but it seldom occurs in the disease under consideration. Again, in the tubercular affection there are generally signs of disease in the apices of the lungs.

Simple catarrhal inflammation and *syphilitic laryngitis* cannot be distinguished in all instances, especially when there is simple redness with slight swelling, although usually the history of the case, the old cicatrices in the pharynx, with scars or deep ulcers in the larynx, and distortion and thickening of the organ, which has a peculiarly dense appearance as compared with œdema or tuberculosis, are sufficient to enable the physician to make an accurate diagnosis.

Between chronic catarrhal laryngitis and syphilitic laryngitis the following are the chief points of difference:

CHRONIC CATARRHAL LARYNGITIS.	SYPHILITIC LARYNGITIS.
No specific history.	Syphilitic history.
Normal contour of parts.	Sometimes distortion of parts by old cicatrices or thickening.
No evidences of ulceration, past or present.	Mucous patches, scars, or ulcers generally present.

We find *malignant disease of the larynx* usually attended by more or less pain and marked in the beginning by circumscribed congestion which is speedily followed by the development of a neoplasm, that

gradually advances, involving, as a rule, all of the tissues with which it comes in contact, causing distortion of the larynx, and finally undergoing deep ulceration. Catarrhal laryngitis never has this history, though I have seen a few cases in which the swelling and distortion of the parts were strongly suggestive of malignant disease. In such instances nothing but continued observation of the case for some time will enable the physician to make an accurate diagnosis.

The differential diagnosis of chronic catarrhal laryngitis and malignant disease of the larynx is as follows:

Chronic catarrhal laryngitis.	Malignant disease of larynx.
Moderate uniform congestion and thickening of parts.	Circumscribed redness and swelling; contour of parts much changed.
No pain.	Pronounced pain.
Hoarseness, but no dysphagia.	Aphonia and dysphagia.
No ulceration.	Eventually ulceration, with offensive discharge.

Prognosis.—The disease usually runs a very protracted course, lasting for months or years, though there is a strong tendency to improvement at times, with subsequent recurrence of the more pronounced symptoms. It very rarely, if ever, terminates fatally; yet there is some reason for believing that very protracted inflammation, after involving the trachea and bronchial tubes in greatly debilitated patients, may eventually terminate in consumption. The disease is not intractable if the exciting causes can be removed and the predisposing tendency corrected.

Treatment.—In every case of chronic laryngitis it is the first duty of the physician to remove the causes if possible. With this end in view, the excessive use of tobacco and alcoholic stimulants, and sometimes even the use of tea and coffee, should be interdicted and the condition of the digestive organs must be carefully regulated. The patient must avoid all exposure to damp and cold, or to the vitiated atmosphere of crowded rooms. He must avoid the inhalation of irritating dust and gases, and must keep the skin and other excretory organs in a healthy condition. The parts involved should be placed, as nearly as possible, at rest, especially during all acute exacerbations of the disease. Singing, shouting, and excessive use of the voice, especially in the open air, must be prohibited; and when there is much irritability of the parts, the patient should converse only in whispers. There are some cases, however, of a chronic low grade of inflammation that seem benefited by moderate use of the voice, which stimulates a flow of blood through the parts, and thus promotes absorption of inflammatory products. Usually prolonged systematic treatment, consisting of repeated applications of stimulating substances, will be necessary before the disease can be cured. The various substances used for this purpose may be applied in the form

of powders, sprays, or pigments according to the tolerance of the patient and the inclination of the physician. As a rule, sprays give the patient less inconvenience and are on the whole preferable, though occasionally powders answer an excellent purpose, and sometimes pigments, especially when applied by means of a cotton probang (Fig. 107), are very effectual. These applications should be made, when possible, every day for one or two weeks, until considerable acute congestion of the parts has been excited; then once in two days for a week or two, and after this less frequently, according to the improvement of the case. It is well

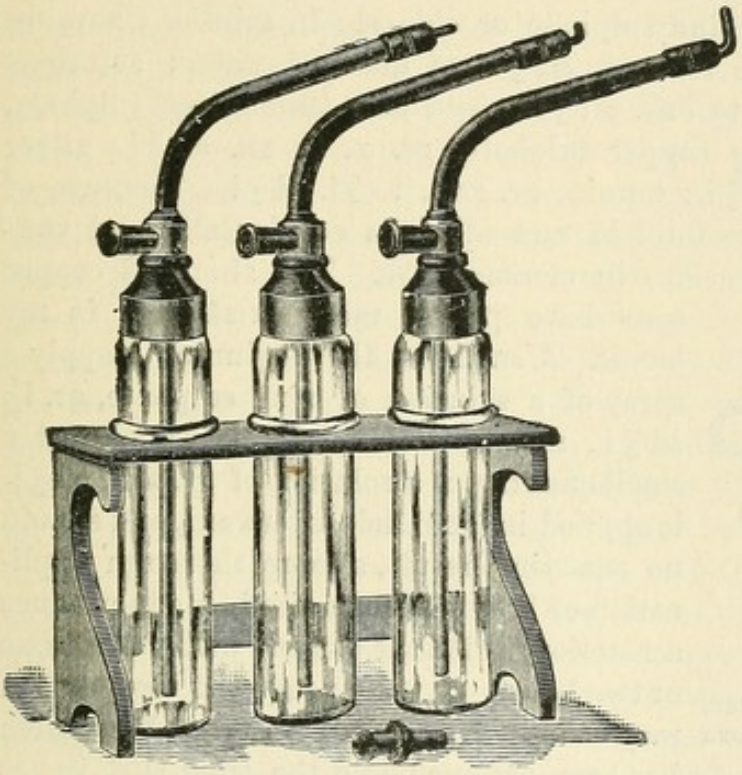

FIG. 106.

FIG. 107.

FIG. 106.—DAVIDSON'S ATOMIZERS, SET NO. 66, FOR OFFICE USE (1-3 size). For the specialist, to whom time is an object, it will be found preferable to have these bottles held by an open spring-clip to the edge of a shelf. The facility with which the tips may be changed to throw a spray in any direction makes each of these bottles equivalent to four of the atomizer tubes in common use. They may be used with the hard rubber attachment shown at bottom of cut but more conveniently with the Davidson cut-off.

FIG. 107.—INGALS' LARYNGEAL APPLICATOR (copper staff, 2-5 size). The cotton should be wound firmly upon the point, and to prevent the possibility of accident a thread should be tied about it with a slip-knot and wound about the staff up to the handle.

also to have the patient at the same time use weaker applications to the larynx by sprays or inhalation each morning and evening. It will be found that different larynges vary exceedingly in sensitiveness, so that an application which will cause no discomfort whatever in one may in another produce extreme pain. It is therefore necessary to try weak medication at first, and always to regulate the strength by the effect, which may be judged quite accurately by the sensations of the patient.

Applications which are made by the patient himself should never cause discomfort for more than twenty or thirty minutes. Those made

by the physician, if daily, should not cause smarting for more than an hour, and, if every second day, not more than two hours; in either case actual pain should not last more than ten or fifteen minutes. The particular remedy to be employed is, as a rule, a matter of little consequence, the object being merely to stimulate the mucous membrane; though it will be found that in some cases one substance will really work better than any other. In most instances a change from time to time will hasten recovery, for where a single agent is used for a long period the parts appear to become so accustomed to it that it has but little effect upon them. The topical remedies commonly employed in this disease consist of zinc sulphate or chloride in solutions varying in strength, from gr. ij. to xxx. ad ℥ i. of distilled water; solutions of iron chloride, ♏ lx. to cxx. ad ℥ i.; iron and ammonium sulphate, gr. v. to xxx. ad ℥ i., or copper sulphate, gr. x. to xx. ad ℥ i.; silver nitrate, gr. x. to ℥ ij. ad ℥ i.; tannin, gr. xxx. to ℥ i. ad ℥ i. Tincture of iodine or turpentine, the fluid extract of thuja occidentalis, and various other substances are also in common use. The zinc and copper salts have proved most satisfactory in my hands. Usually in the beginning I apply a spray of a solution of zinc sulphate, gr. ij. ad ℥ i., and if this causes no discomfort a small quantity of a solution of gr. xxx. ad ℥ i. is applied immediately afterward, and should no smarting result, a more thorough application of it is made, the aim being to produce a reaction which the patient will feel for one or two hours. At the next visit the solution may be modified according to the effect which has been obtained, and the time that it has been felt. Other remedies may be employed in the same manner. I usually make these applications in the form of spray with an air pressure of thirty or forty pounds to the inch. The swab I seldom use, and the brush not at all. I rarely employ tincture of iodine or silver nitrate, though sometimes they are of great benefit. The strong solutions of the latter recommended by some authors are in most cases objectionable, because of the spasm of the larynx and the great discomfort they cause, while their beneficial effects are seldom greater than those of milder applications. For use at home I give the patient weak solutions of similar astringents (Form. 88, 92, 94). These the patient applies cold with some suitable atomizer.

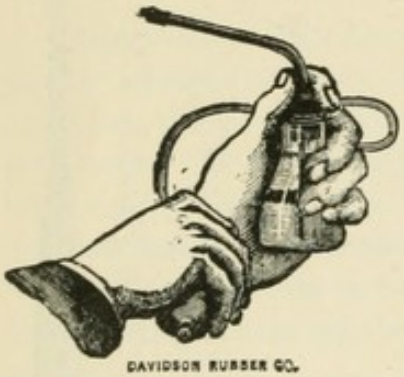

FIG. 108.—DAVIDSON'S ATOMIZER, No. 59, OLD STYLE, SCREW TOP, LONG TIP (¾ size).

Steam sprays seem to cause relaxation of the parts, which favors subsequent inflammation, and therefore they are not recommended. However, they may sometimes be used with more or less benefit at night or when the patient is not going out of doors for one or two hours. Lennox Browne particularly recommends such inhalations as benzoin,

phenol, creasote, or camphor. If these are used with warm water, the patient must not go out of doors for some time afterward. They may be employed in some of the lighter oils, as for example, liquid albolene, and applied by means of some of the various nebulizers or atomizers without the danger incident to the use of warm vapors.

The substances most commonly used in the larynx in the form of powder are bismuth, boric acid, iodoform, iodol, berberine muriate, gum benzoin, myrrh, alum, zinc sulphate, and silver nitrate. Boric acid and iodol or iodoform in equal parts constitute a very useful stimulant and antiseptic application in some cases. Boric acid alone is slightly more stimulating. Equal parts of gum benzoin, bismuth, and iodol or iodoform make an excellent powder, still more stimulating. Tannin, in the proportion of from two to ten per cent, with sugar of milk, is sometimes useful. One part of berberine muriate to two parts of acacia forms an excellent application for certain cases, especially where there is a relaxed condition of the mucous membrane and enlargement of the follicles. Equal parts of alum and sugar of milk answer well when a decided effect is desired. Silver nitrate I never employ in this way, though it is recommended by good authority. With most of these powders it is well to combine about five per cent of pulverized starch to prevent packing, and all of them should be thoroughly triturated. Stimulating or sedative troches will often be found beneficial; of the former, troches of ammonium compound, krameria compound, or benzoic acid compound are excellent examples (Forms. 41, 46, 48). Of the sedative troches we have lactucarium, terpin hydrate and cannabis compound (Forms. 29, 33), or morphine, antimony, and ipecac compound (Form. 32) are good examples. When cough is a troublesome feature, sprays of potassium bromide ʒ ss. to ʒ i. ad ℥ i. will often be found very useful.

Irritating cough may sometimes be readily relieved by a few light inhalations of chloroform; for this purpose a small bottle may be given the patient to carry in his pocket for use as needed. Aside from this local treatment, it will often be found of the greatest importance to cure coexisting disease of the pharynx, base of the tongue, or nasal cavities. Enlarged glands at the base of the tongue, or varicose veins, should be reduced by cauterization. Follicular enlargements on the pharyngeal wall must be cut down by the cautery, and hypertrophic rhinitis or exostoses of the septum must be met by proper surgical procedures. Other forms of inflammation or obstruction in the nares or pharynx must also be remedied, for the laryngeal disease can seldom be permanently cured while these affections remain. In some instances it will be found desirable to apply caustics, such as silver nitrate, chromic acid, or the galvano-cautery point to enlarged follicles in the larynx itself. In such cases the larynx should first be thoroughly anæsthetized by a twenty per cent or twenty five per cent solution of cocaine, and then the application should be made accurately to the parts diseased, and

to no other, care being taken that the cauterizations are never extensive or severe. After any of these operations the patient should apply cold compresses to the neck for from twelve to twenty-four hours, to prevent undue reaction.

TRACHOMA OF THE VOCAL CORDS.

Synonym.—Chorditis tuberosa.

Trachoma of the vocal cords is a chronic inflammation of the larynx, characterized by roughness or a granular appearance of the vocal bands, with some swelling, and more or less alteration of the voice. It is found most frequently in singers, but may occur in others. I have seen one case in the person of a farmer who used his voice very little in singing.

Anatomical and Pathological Characteristics.—The disease appears to consist of hypertrophy of the connective tissue, which results in a nodular or granular thickening of the cord.

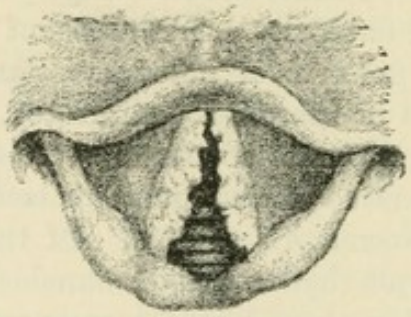

Fig. 109.—Trachoma of Vocal Cords (extreme).

Etiology.—No special causes of the affection are known, aside from repeated over-use of the voice especially when the larynx is congested.

Symptomatology.—The symptoms are those of chronic laryngitis, *i.e.*, hoarseness or aphonia, with more or less cough and expectoration. Upon laryngoscopic examination, the cords are found congested and thickened, and presenting a nodular appearance (Fig. 109) of the surface, with unevenness of the edges.

Diagnosis.—The diagnosis will be based upon a history of chronic laryngitis, with the physical appearances just mentioned.

Prognosis.—The duration may be months or years, but prolonged rest and judicious treatment will usually promote a cure.

Treatment.—The treatment consists of the application of mild caustics or mineral astringents in the same manner as recommended for chronic laryngitis. By this course, persistently carried out, a cure may usually be effected. Owing to the obstinacy of this affection, Carlo Labus, of Milan, has recommended flaying of the vocal cords, or, in other words, stripping off of their hypertrophied mucous membrane by means of ordinary laryngeal forceps (*Archives of Laryngology*, 1880). Charles

E. Sajous, of Philadelphia, has recommended touching small areas of the cord with chromic acid at intervals of several days (Transactions of the American Laryngological Association for 1888). This treatment seems to promise well and should be given a fair trial after the ordinary measures have proven unsuccessful. In applying the chromic acid, a very small portion should be fused on the end of a guarded applicator

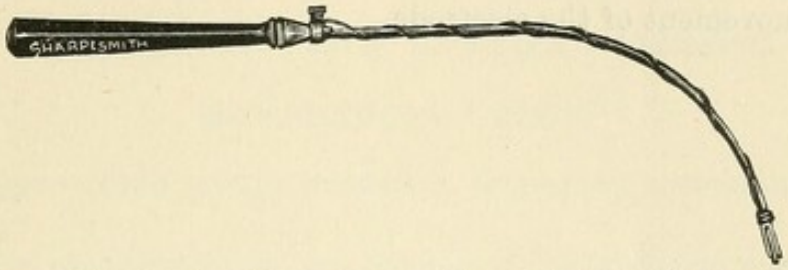

FIG. 110.—INGALS' CHROMIC ACID APPLICATOR AND HANDLE (1-3 size). This is a long aluminium wire, properly curved to correspond with the faucial angle, and guarded at the end by a piece of rubber tubing which protects the parts not to be touched from contact with the agent. The bit of rubber tubing is prevented from slipping off by a silk thread which is tied about it and wound around the stem up to the handle.

(Fig. 110) with which the part should be accurately touched, the larynx having first been anæsthetized by cocaine to prevent injury to other parts.

PHLEBECTASIS LARYNGEA.

Phlebectasis laryngea is a varicose condition of the laryngeal veins, characterized by more or less alteration of the voice and discomfort in the larynx.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—In mild cases fine veins are seen running along the epiglottis and the lower portions of the ventricular bands; in more severe cases the enlarged veins appear tortuous and extend also over the vocal cords and arytenoid cartilages.

FIG. 111.—INGALS' GALVANO-CAUTERY HANDLE (½ size). In this the circuit is closed by moving the finger from the contact button.

ETIOLOGY.—There is no known cause of the disease.

SYMPTOMATOLOGY.—The patients usually complain of uneasy sensations in the larynx, of slight cough, and of more or less hoarseness.

DIAGNOSIS.—The diagnosis is made by careful inspection of the larynx, care being taken not to mistake for enlarged veins the blackened mucus which sometimes collects upon the surface.

TREATMENT.—Topical applications of strong astringents may be made, but the most satisfactory treatment consists of destruction of

the vein by repeated small cauterizations with the galvano-cautery, a period of from ten days to two weeks intervening between the operations. Intra-laryngeal cauterization should be made with an electrode provided with a small fine platinum tip, which will heat or cool quickly. The best handle for this purpose is one in which the circuit is closed on relieving the pressure from a spring (Fig. 111) instead of by the usual method of pressure; this allows the circuit to be completed with the least movement of the electrode.

CHAPTER XXIV.

DISEASES OF THE LARYNX.—*Continued.*

MEMBRANOUS CROUP.

Synonyms.—True croup, exudative laryngitis, membranous laryngitis.

Croup, in the strict sense, is a disease of the laryngeal mucous membrane characterized by the exudation of inflammatory lymph, forming false membrane, and attended by more or less muscular spasm of the larynx. Mackenzie and some other authors, together with a large number of the profession, believe it identical in nature with diphtheria, but I am convinced that these are two distinct diseases. Most of the older writers, and not a few of the more recent, agree with Aitken, who says of this affection: "Any one who has seen much of croup in children can have no difficulty in recognizing it as a disease distinct from diphtheria in its attack, its course, and results." I know of no better definition for the disease than that given by Lennox Browne (Diseases of the Throat, second edition), who defines it as a pseudo-membranous inflammation of the air passages, non-infectious and non-contagious. The disease occurs most frequently in children between two and seven years of age. It seldom occurs in older children, and is extremely rare in young infants and in adults.

Anatomical and Pathological Characteristics.—The inflammation is almost entirely confined to that portion of the larynx above the cords. The false membrane, though deposited partially upon the epiglottis and ventricular bands, is mainly found about the glottis itself and upon the vocal cords. The inflammation may extend to the submucous tissues, resulting either in spasm or paralysis of the laryngeal muscles. The false membrane is comparatively thin, only involving the epithelial layer of the mucous membrane, whereas in diphtheria the whole thickness of the mucous membrane is affected.

Etiology.—Those who believe in the identity of diphtheria and croup attribute this to a specific contagium, the action of which, however, they admit may be favored by the usually recognized causes of the diseases. In some instances there is undoubtedly a strong hereditary predisposition to the disease, and in a large number of cases its onset is certainly favored by acute laryngitis. The disease is also favored by poor general health. There is little doubt that the majority of cases are

directly due to improper clothing or to life in damp, chilly, and ill-ventilated rooms. The disease is peculiarly prevalent in the spring and fall months, when the outdoor temperature is so warm that it is hardly necessary for apartments to be heated, therefore at this time many houses are kept at a temperature of from 60° to 65° F. The adults, who are working about, and who are necessarily in higher strata of air than the children playing upon the floor, do not notice the necessity for more warmth, but the little ones become chilled, a slight catarrhal laryngitis supervenes, and, whether or not this is the direct cause of croup, it certainly favors the development of the false membrane. The disease is not contagious, and it seems to have been satisfactorily demonstrated that it cannot be inoculated from the false membrane, though Mackenzie and others hold contrary views. The theory that this disorder is often the direct result of certain ptomaines generated within the patient's own body seems to me reasonable.

Symptomatology.—For the sake of convenience in description, the disease may be divided clinically into three stages—a catarrhal, an exudative, and a suffocative.

The catarrhal stage is usually preceded for about forty-eight hours by a feeling of malaise attended by slight fever and anorexia; later there is considerable fever, cough, hoarseness, and some dyspnœa. In the latter part of this stage the false membrane begins to form.

In the exudative stage the false membrane is being gradually or rapidly deposited in the larynx, spasmodic action of the muscles becomes more frequent, and dyspnœa more and more severe. There is either hoarseness or complete aphonia, and cough may or may not be troublesome. Finally, the membrane becomes so thick as to seriously obstruct the glottis, giving rise to the last stage.

In the suffocative stage, dyspnœa is constant, but still more or less aggravated at times by spasm of the laryngeal muscles. As the stage advances, all of the symptoms of gradual suffocation supervene, and finally, in the majority of cases, the patient dies from the effect of imperfect aëration of the blood.

In the first stage the temperature is raised from one to three degrees, and the pulse is quickened from twenty to thirty beats per minute; yet frequently the friends may not notice these symptoms until the child is suddenly wakened at night struggling for breath. This paroxysm, which is due to spasm of the laryngeal muscles, continues for a few minutes, and then may pass off till the following night, or other attacks may occur from time to time during the same night. In the interval between the attacks the child breathes with comparative ease and soon falls into a troubled sleep. It usually plays about the house on the following day, but more or less hoarseness is noticed, and at night all of the symptoms become more aggravated. Again, there may be an intermission in the symptoms during the day following, and it is not un-

usual to find the child running about the house after a second night of suffering and unrest from the paroxysms of true croup; but on the succeeding night the suffocative stage generally begins, in which there is constant dyspnœa, with occasional paroxysms which add greatly to the distress. The spasms are less pronounced than in the catarrhal stage, because carbonic acid poisoning renders the muscular action sluggish. There are some unfortunate cases, however, in which the disease runs rapidly through the three stages and many terminate fatally within a few hours. In the exudative stage, hoarseness is persistent, there is a peculiar shrill, harsh cough, which needs to be heard but once to be remembered, and occasionally particles of false membrane are cast off. Fever and anorexia are usually present, there is constant dyspnœa, and inspiration and expiration are both prolonged, especially the former. The suffocative paroxysms now become more frequent and severe. At the onset of one of these, the child suddenly springs up in great alarm, the eyes stand out like those of one in strangulation, the nostrils are dilated, and the respiratory muscles tense with the violent effort at inspiration; in a few seconds the countenance becomes livid and the child almost ceases its efforts to breathe; but finally the spasm relaxes, air again enters the lungs, lividity disappears, and respiration becomes once more normal, so that excepting for the hoarseness it would hardly be known that the child was ill. One such attack usually lasts two or three minutes, and may be renewed after a short interval of rest. Recurrence in this manner may take place several times; but usually after the first three or four paroxysms the child falls into a restless sleep that may last for several hours. If the larynx can be examined, we find it congested, with here and there patches of thin, yellowish white membrane upon the surface. In this stage the child is extremely restless, throwing itself about the bed, or every few moments asking to be taken up or laid down in its fruitless search for comfort and the oxygen it needs. The face and general surface are ashy pale, with lividity of the lips and finger nails; the skin, which has been hot in the first and second stages, remains so in the earlier part of this the third stage, but later becomes cold and is bathed in a clammy perspiration. The pulse is quick and small, the voice weak or lost, and the cough feeble or suppressed. The tongue usually is coated, and there is much thirst, but no desire for food.

In the first stage of the disease the respiration may be accelerated, as in other catarrhal affections of the mucous membrane, but in the later stages the breathing becomes slow and labored, and with each inspiration there is sinking in of the soft parts of the chest. This is most marked at the lower end of the sternum and over the false ribs, but it is also noted in the interclavicular notch and just above the clavicles.

DIAGNOSIS.—True croup may be mistaken for catarrhal laryngitis, laryngismus stridulus, or for diphtheria. The essential points

in the diagnosis are: gradually increasing hoarseness, slight constitutional symptoms, dyspnœa and the formation of false membrane which is confined to the larynx.

In *catarrhal laryngitis* there is commonly considerable pain in coughing, speaking, or swallowing; there is but little dyspnœa, the cough is short and sharp, there is no expectoration of false membrane, and the respiration seldom becomes slow and labored; all of which symptoms distinguish it from croup. In typical cases there is no difficulty in making the diagnosis, but it is difficult or quite impossible, in complicated or obscure instances, and therefore doubtful cases should be treated as croup.

From acute laryngitis the disease is to be distinguished by the characteristics presented below:

Membranous croup.	Acute laryngitis.
Generally occurs in children.	Generally occurs in adults.
Slight congestion and swelling.	Marked congestion of parts, and swelling.
Slight pain in coughing, speaking, or swallowing.	Marked pain in coughing, speaking, and swallowing.
Cough harsh and stridulous.	Cough sharp and short.
Marked dyspnœa.	Slight dyspnœa.
Slow, labored respiration.	Respiration nearly normal, or may be increased in frequency.
False membrane in larynx.	Tenacious, scanty sputum, but no false membrane.

Laryngismus stridulus differs from croup in that it comes on suddenly when the child is apparently well. It is not attended by inflammation, or quickening of the pulse, or fever, and the dyspnœa passes off in a few minutes, leaving the child breathing with perfect ease until another paroxysm occurs. Sometimes the paroxysms are not repeated. As soon as the attack is over, the voice becomes normal.

From laryngismus stridulus croup is to be distinguished as follows:

Membranous croup.	Laryngismus stridulus.
Slight congestion and swelling.	No congestion or swelling.
Fever, rapid pulse.	No fever, pulse normal except during paroxysm.
Slow in development.	Sudden in its onset.
Labored and slow respiration, but with paroxysms of more pronounced dyspnœa.	Attack may not be repeated; respiration and voice normal except during paroxysm.
Aphonia and dysphonia constant.	Voice normal except during brief paroxysms of dyspnœa.
Presence of false membrane.	No false membrane.
Comparatively long duration, usually two or three days.	Short duration.

Croup cannot always be distinguished from *diphtheria*, as there are some cases which at first appear to be simply true croup, but in which diphtheritic membrane is subsequently deposited in the pharynx and eventually paralysis occurs; or other members of the family are attacked by diphtheria. But in typical cases the distinguishing symptoms are well marked. Diphtheria comes on more suddenly, and the constitutional symptoms are more pronounced. There is usually abundant false membrane in the fauces, and paralysis is a frequent sequel. The fever in diphtheria is more variable than in croup. The dyspnœa in diphtheria is slowly developed, and there is little, if any, spasm of the glottis. Diphtheria is often very contagious, croup is not at all so. An attack of croup is usually preceded by two or three days of malaise or catarrhal symptoms, and is finally suddenly ushered in during the night by a severe paroxysm of dyspnœa; it is attended by mild but continuous and gradually progressing fever; the constitutional symptoms are slight, there is no false membrane in the fauces, and no paralysis following the disease.

True croup and diphtheria present the following differential points:

MEMBRANOUS CROUP.	DIPHTHERIA.
Malaise or catarrhal symptoms at first, but constitutional symptoms comparatively slight.	Constitutional symptoms developed quickly and very pronounced.
Fever mild, but continuous and gradually increasing.	High fever at first, later variable.
Severe paroxysm of dyspnœa ushered in suddenly at night.	Dyspnœa developed slowly, no decided spasm of glottis.
No false membrane in fauces.	False membrane in fauces.
Not contagious.	Often contagious.
No subsequent paralysis.	Frequently paralysis follows.

PROGNOSIS.—In unusual cases patients may die with membranous croup within three or four hours after the first indications of the disease, but commonly the affection extends over two or three days, and sometimes it continues for a week. It is probable that not more than twenty per cent of the cases would recover without surgical interference; and even under the most improved methods, according to Hilton Fagge, sixty or seventy per cent die. It appears, however, that of those upon whom intubation is done by O'Dwyer's method forty to fifty per cent recover. In cases which progress favorably the false membrane gradually disappears, the spasms subside, breathing becomes less and less difficult, the sputum becomes more abundant, the cough easy, and in two or three days the child is out of danger. In fatal cases the dyspnœa steadily increases, the child becomes extremely restless, the cough, which has been severe, loses its loud, croupy sound, and may become almost inaudible; the pulse grows feeble and rapid, the extremities cold, the

skin is bathed in cold perspiration, and the patient finally dies of exhaustion, or gradually passes into a comatose condition, in which death ensues from carbonic acid poisoning. Occasionally life is cut short by a heart clot or by convulsions, and in some instances pulmonary complications are the immediate cause of death. A few cases die from suffocation caused by spasm of the glottis.

TREATMENT.—In the early stage of the disease great benefit may be derived from the external application of either cold or heat; but whichever is used must be employed continuously, as the alternate use of cold and heat makes matters worse. Cold may be applied by cloths wrung out of ice water and frequently changed, but, better still, by means of an ice bag. For this purpose a long, narrow rubber bag not more than three inches in width should be obtained and filled about half full of ice cracked into small pieces not larger than a filbert. It should then be wrapped in a handkerchief and tied closely about the neck. The ice will melt in about an hour, and should then be renewed. An excellent method of applying cold is by means of the Leiter coil. This consists of a coil of metallic tubing, which may be fitted accurately to the neck; to each end is attached a rubber hose, one leading from a receptacle of ice water, and the other carrying off the waste. Continuous cold for the first twenty-four or forty-eight hours will frequently cut short the attack. Sometimes because of the depression of the patient, and in other instances to meet the wishes of the friends, it is better to use heat. This may be applied by means of cloths wrung out of hot water, by hot water bags, or by the Leiter coil, already referred to. In the early stage of the disease, heat may be made to answer exactly the same purpose as cold, but it is usually more beneficial in the later stages of the attack.

The atmosphere of the room should be kept moist and at a temperature of 75° or 80° F. Moisture may be obtained by means of a basin of water on the register or stove, by the steam atomizer, or by constant slaking of lime in the room. Owing to the fact that diphtheritic membrane, when immersed in lime water gradually dissolves, the vapors from lime have been considered especially beneficial in this disease; but it is doubtful whether they are ever inhaled in sufficient quantity or of sufficient saturation materially to affect the false membrane. Many physicians recommend that a basin of slaking lime be kept constantly in the room or upon the stove, others apply lime water by means of a steam atomizer, but probably the most efficient way of using it is by means of the croup tent, as follows: having placed a pan of hot water close by the head of the bed and dropped into it a handful of unslaked lime, a sheet is thrown over the pan and over the child's head, being held up somewhat from the face; the patient is by this means compelled to breathe the vapors arising from the lime. The application should be continued ten or fifteen minutes and repeated every half-hour when

practicable. A steam atomizer may be kept constantly running in the room for the purpose of saturating the air with moisture, and the patient should be induced to inhale from it directly two or three times an hour, for five or ten minutes. For inhalation by means of this instrument, solutions of sodium bicarbonate gr. v. to x. ad ℥ i., the saturated solution of lime water, lactic acid gr. xx. ad ℥ i. to dissolve the membrane, or potassium bromide gr. xx. to xxx. ad ℥ i., or the aqueous extract of opium or belladonna gr. i. to ij. ad ℥ i. may be employed to prevent the paroxysmal dyspnœa. Emetics are employed for the purpose of mechanically dislodging mucus and false membrane from the larynx, and relaxing the muscular system so as to prevent spasms of the glottis. For this purpose tartarized antimony in the form of the compound syrup of squills is probably the agent most frequently employed. It should be given in doses of ♏ xv. to xxx. repeated every fifteen minutes until vomiting occurs, or until its depressing effects are noticed; but the dose should not subsequently be repeated for several hours. Ipecac in some form is used for the same purpose, and it has the advantage over tartarized antimony of causing no subsequent depression. Zinc sulphate, alum, and turpeth mineral are also employed; the latter has been especially recommended by so eminent an authority as Fordyce Barker, who considered it prompt, safe, and efficient in doses of grs. i. to iij. Emesis usually follows its administration, in from five to twenty minutes. Pulverized alum, gr. xx. ad ℥ i., mixed with honey is a prompt, safe, and not unpleasant emetic in these cases. Mercurial preparations have been recommended for the purpose of limiting the formation of false membrane, and within the last few years mercury bichloride has been much employed in comparatively large and frequent doses. I prefer the mild chloride, which is more easily managed and quite as efficient. Turpeth mineral is also used by some physicians in small and repeated doses for the same purpose. In children one or two years of age, I frequently order one grain of calomel to be given every hour until it acts upon the bowels, and subsequently every two hours for ten or fifteen doses. A healthy child of this age will usually be speedily purged by one grain of calomel, but in croup about twenty grains will generally be taken before the effects of the remedy are noticed upon the bowels, and then it does not act vigorously. Thus, these patients often take from thirty to forty grains of calomel within thirty-six or forty-eight hours, and I have never seen any deleterious effects from its use, but have frequently witnessed the most gratifying results in the relief of the laryngeal symptoms. Unfortunately, however, in the majority of cases, no matter what external applications we employ, or what internal remedies are administered, the disease goes steadily on from bad to worse; the glottis becomes narrower until finally suffocation is imminent, and then we must resort to surgical measures or the child is lost.

Mackenzie recommended a croup brush in which the hairs run toward

the handle, designed to be introduced through the glottis and withdrawn so as to dislodge the false membrane. I do not know how efficient this has proved, but it has not become popular with the profession. In a few instances an ordinary catheter has been passed through the glottis, by which the patient has been enabled to obtain sufficient air to support life. In this extremity we should not temporize, but should resort at once to O'Dwyer's intubation, or to tracheotomy, either of which, if performed early, will save many lives. In children under five years of age intubation seems to offer better chances for recovery than tracheotomy; therefore it should be advised, and because of the ease of its performance, the readiness with which the consent of parents is obtained, the speedy relief afforded, and the avoidance of an anæsthetic, it may be recommended in all cases, for it is no bar to the subsequent performance of tracheotomy if that operation should seem necessary. The best cases for either of these operations are those in which the membrane is confined to a small portion of the larynx and where the carbonic acid poisoning

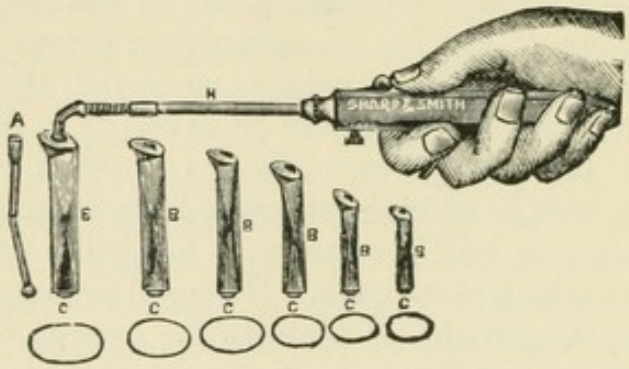

FIG. 112.—O'DWYER'S INTUBATION INSTRUMENTS (¼ size). *H*, Applicator; *A*, obturator; *B*, *B*, tubes of various sizes; *C*, *C*, actual calibre of tubes.

is not very pronounced; when the difficulty of respiration has continued for several hours, giving rise to pulmonary atalectasis, or œdema, or to heart failure, little can be hoped from either. When the glottis becomes so obstructed that there is falling in of the soft parts of the chest with each inspiration, and respiration is long and labored, the lips blue and the skin pale, there should be no delay in adopting surgical measures, for every hour then will materially lessen the chances of recovery.

Intubation is performed by means of the instrument (Fig. 112) devised by Joseph O'Dwyer, of New York. His set of instruments consists of six tubes graduated for children less than ten years of age. It contains a gauge for measuring the tubes to determine the proper size for any given age, an applicator for introducing the tube, an extractor for withdrawing it, and a mouth gag; the latter, however, is not as satisfactory as some others, because the child is sometimes able to displace it from between the jaws and may bite the operator. But the other instruments, which were the outcome of long and patient experimentation, are so nearly perfect that it has been difficult in any way to im-

prove upon them. Henrotin's, Waxham's, or Allingham's gags (Figs. 113, 114, 115) are preferable. In preparing for the operation, the child's age having been ascertained, the proper tube is selected and a strong thread about three feet in length is passed through the eyelet in its head and the ends are tied together; the applicator is then screwed into the obturator, and this passed through the tube ready for the operation. The head of the tube is bevelled so that one side is much shorter than the other, and this short side should be placed toward the handle of the

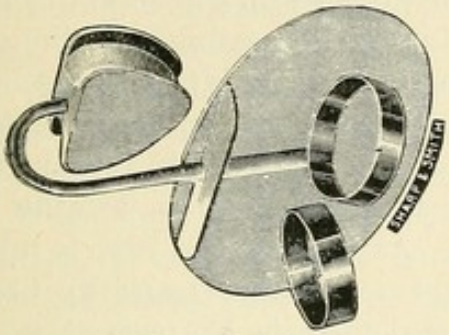

Fig. 113.—Henrotin's Gag (½ size).

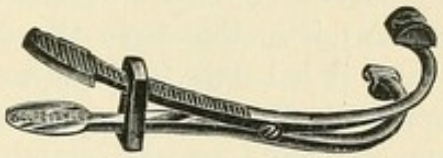

Fig. 114.—Waxham's Gag (½ size).

instrument, so that when introduced into the larynx it will conform to the position of the epiglottis. The child, wrapped in a blanket or sheet, which is pinned closely about the neck so that its arms are pinioned, should be held in the arms of the nurse, with its head against her left shoulder. The gag is then inserted between the teeth upon the left side, and intrusted to the assistant who is to hold the head. The operator's forefinger of the left hand should be oiled or smeared with vaseline to prevent inoculation through any abrasions upon the surface in case

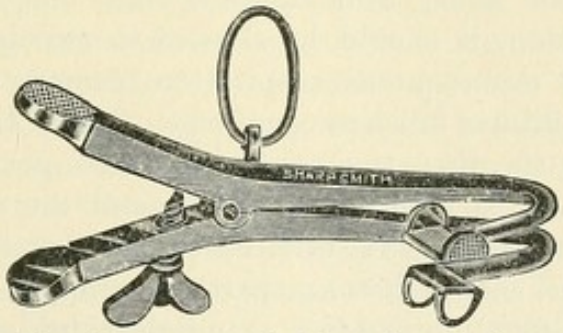

Fig. 115.—Allingham's Mouth Gag (½ size).

the disease should prove to be diphtheria, and a broad metallic ring or a rubber finger cot the end of which has been cut off, should be slipped over the finger to prevent the patient from biting it in case the gag should become displaced; or in the absence of these, the finger may be wound with a strip of cloth, which will answer the purpose fairly well.

The tube with the applicator, having been dipped into warm water to bring it to blood heat, is ready for introduction. The child's head being thrown slightly backward and held firmly by the assistant, the operator introduces the forefinger of the left hand over the base of the tongue,

down behind the epiglottis, until he feels the arytenoid cartilage, upon the upper edge of which the finger is rested. The tube is now guided down along the palmar surface of the finger until it reaches the larynx when, the handle of the applicator being elevated so as to turn the end of the tube farther forward, it is passed into the glottis and crowded downward about half an inch. At the same time the end of the finger which is resting on the arytenoid is brought upward and placed upon the upper end of the tube, which is forced downward as far as possible. The slide upon the applicator is then shoved forward, the obturator disengaged, and the applicator removed, while with the finger of the left hand the operator crowds the head of the tube fairly into the vestibule of the larynx. Not more than ten seconds should be consumed in this operation; if in this time the operator does not succeed in introducing the tube, it is better to withdraw it and allow the child to breathe for a moment before making another effort. As soon as the tube is introduced, the child usually coughs, and the respiration generally has a peculiar tubular sound, which indicates that the tube has been placed in the air passage; if this sound is not heard, the operator should feel again for the tube to ascertain whether or not it has been passed into

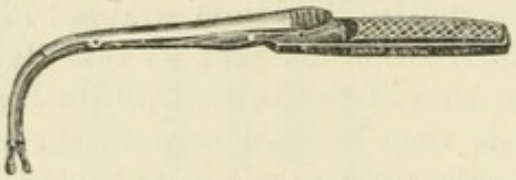

Fig. 116.—O'Dwyer's Extractor (¼ size).

the œsophagus instead of the larynx. If not in proper position it must be withdrawn by the string and another effort made to introduce it. If in proper position, it should be allowed to remain with the string attached for a few minutes until respiration becomes thoroughly established and the child has finished coughing. One of the threads should then be cut near the lips, the operator's forefinger carried down to the head of the tube to hold it in position and the string withdrawn. The tube is left in the larynx, where it should remain for from two to six days, unless it should become partially stopped by dried mucus, as indicated by difficult breathing, or unless subsidence of the symptoms leads us to believe that the swelling has gone down and the false membrane disappeared. In many cases the tube will be coughed out as soon as the necessity for its further use ceases. When it becomes necessary to remove it, the child is placed in the same position as for its introduction, and with the index finger of the left hand the operator guides the extractor down to the larynx, where it may be felt to strike against the end of the tube. It is then moved about gently, no force being used, until it drops into the opening of the tube; the blades should then be separated and firmly held while the instrument and the tube are being withdrawn, especial care being observed not to

relax the pressure just as the tube is being turned out of the pharynx, for, if this is done, the instrument will slip, and the tube may either fall back into the larynx or be swallowed. It is well to have at hand a pair of forceps for the purpose of seizing the tube in case the instrument should slip at this stage of its withdrawal. Special care should be taken that no pressure is made upon the head of the tube in attempting to introduce the extractor, for the tube might possibly be pushed below the vocal cords, an accident which has happened in a few cases. After intubation, mercurials should be given freely for twenty-four or forty-eight hours, as already advised, and care should be taken that when the patient takes fluid none of it passes into the trachea, an accident liable to set up pneumonia, and one which is probably responsible for many of the deaths which occurred in the early days of intubation.

When fluid of any kind is taken while the child is in a sitting position, a cough almost immediately follows, indicating that some of it has passed into the air passages. To avoid this, the most effective plan is that recommended by Frank Cary, of Chicago, and introduced by Wm. E. Casselberry, which consists of placing the patient supine with the head much lower than the body, and feeding it from a nursing-bottle or through a tube. In this position fluid cannot run into the trachea, but will be forced up the œsophagus into the stomach. Soft solids may be given with the child in any position, and some children will speedily learn to swallow even fluids in the erect position; but the friends must be cautioned not to try this experiment. The child may suck small pieces of ice if it wishes, to quench thirst, or it may be given ten or fifteen drops of water without danger, even in the erect posture, but the safer way is the better. Occasionally on introducing the tube some portion of the false membrane is forced below it in the trachea, and suffocation becomes imminent. If this occurs, the tube should be at once withdrawn, when it usually either brings the membrane with it or the latter will be speedily coughed out. If this should not occur, tracheotomy should be done at once. Because of the liability to this accident, the operator should always have his tracheotomy instruments at hand when performing intubation. I consider the operation of intubation preferable to tracheotomy in croup occurring in children under five years of age, and in those older than this it will usually be satisfactory; but there are, all told, many cases among these older patients, especially in diphtheritic laryngitis, where tracheotomy would be advisable.

Tracheotomy is so thoroughly described in all works on general surgery that I need only mention the essential points as they have impressed themselves upon me. The instruments which are liable to be needed are: a sharp pointed bistoury, a scalpel the handle of which should be flat and thin so that it may be used in tearing through the connective tissue, a blunt pointed scalpel which may be used in enlarg-

ing the opening in the trachea, three tenacula, a strong grooved director, an aneurism needle, several artery forceps and sponge holders, several large curved needles, and a suitable double tracheotomy canula, which should have no fenestra in it, for such an opening favors the formation of granulation tissue at the upper end of the incision in the trachea, and is not needed. Two retractors are also needed for holding apart the edges of the wound, and I like very much a pair of rat-toothed artery forceps for taking up and tearing through the connective tissue.

The patient should be placed upon a table before a good light, and, when anæsthetized, a rolling pin wrapped about with a towel (or some other firm roll) should be placed under the shoulders and neck, in order to throw the head backward and raise into prominence the anterior superior tracheal region, and give a good field for the operation. Ether or chloroform may be used as a general anæsthetic for this operation; but the latter is generally preferred especially for children. In adults the parts may be sufficiently anæsthetized by the hypodermic injection of a few drops of a four per cent solution of cocaine along the line of incision three or four minutes before the operation (Form. 140). The operator stands at the patient's right, with his right hand toward the patient as he faces the head, the patient being between him and the light. The first cut is made by pinching up a transverse fold of the skin over the trachea, transfixing it with the sharp pointed bistoury and cutting out so as to make an incision about two inches in length, extending from a little above the inter-clavicular notch to the cricoid cartilage. By this the superficial fascia and adipose tissue are exposed, which should be worked through with the back of the scalpel or with the aid of the rat-toothed forceps and grooved director, accompanied by as little cutting as possible. We then come down upon the dense fascia covering the muscles and important blood vessels. At this stage of the operation I have derived great benefit from the rat-toothed forceps, with which I grasp the fascia and twist out a small piece, thus making a hole into which the director can be inserted. With the director, and handle of the scalpel, the fascia can mostly be torn through, but sometimes portions of it will have to be cut upon the grooved director, in doing which great care should be taken not to incise a blood vessel which it may be difficult to detect when stretched over the director. Thus working through the fascia we come upon the muscles and engorged blood vessels, which must be separated, by the handle of the scalpel, the director, and the finger, and pushed aside, where the assistant should hold them by means of the retractors. A thin layer of fascia covering the trachea is thus exposed; this should be carefully divided with the back of the scalpel before the windpipe is opened.

During the operation blood should be carefully mopped away, and if veins or arteries are accidentally cut they should be caught by the artery forceps and turned aside. In working our way through the soft

tissues down to the trachea, we come upon the isthmus of the thyroid, sometimes found considerably enlarged. This may be crowded out of the way upward or downward, in either direction that is most convenient, though upward is usually best. Sometimes it is so much in the way that it is necessary to pass a double ligature, tie upon each side, and cut between. The ligature may be easily passed with the aneurism needle. If we succeed in reaching the trachea without much bleeding, it will be seen as a round, yellowish tube at the bottom of the wound, and may also be readily felt by the finger. About this time the patient is liable to cease breathing, apparently from the effect of the atmosphere on the pneumogastric nerves, and it frequently becomes necessary to complete the operation at once. However, if time is allowed, the wound should be sponged out and all bleeding checked before the trachea is opened. From the efforts at respiration, the trachea often moves up and down convulsively, and it must be seized and held firmly before an incision can be made. The best way to accomplish this is to pass a tenaculum just below the cricoid cartilage, or first ring of the trachea, and draw it upward and forward. The point of a scalpel should then be passed between the rings of the trachea at the lower portion of the wound, and a cut made upward, dividing three or four rings. I prefer to divide the third, fourth, and fifth rings of the trachea rather than to make either the high or the very low operation, as the high incision comes too near the larynx, and the very low is more difficult because of the deep situation of the trachea. Care should be taken that the point of the scalpel does not pass far enough through to injure the posterior wall of the trachea. As soon as the cut has been made, air will be heard hissing in and out of the trachea, and the knife should be turned sideways to separate the edges, and held a few seconds until the patient obtains a little air; but as soon as possible the cut edges of the trachea should be caught with tenacula and the wound drawn open. The patient then usually has a paroxysm of coughing that throws out blood, mucus, and false membrane, which should be quickly wiped off so as not to be drawn back into the opening. As soon as the patient becomes quiet, the large bent needles, which have been previously threaded with strong ligatures, are passed, one through each side of the edges of the trachea, the needle is removed, and the threads are tied together so as to form two loops by which the trachea may be held open. These are often found exceedingly useful during the next two or three days, providing the tube happens to be displaced, for they relieve us from the necessity of holding the trachea open, with tenacula or with special instruments devised for the purpose, during the reintroduction of the tube; furthermore, if at any time the tube should be accidentally displaced, the nurse, by drawing upon these strings, may open the wound so that breathing can be readily carried on. The tracheal tube, which should always be as large as can be conveniently worn by the patient, never less than

a quarter of an inch in diameter, may now be introduced, it having first been dipped into warm water to bring it to the temperature of the body. This is a part of the operation frequently found difficult, apparently either from the surgeon's having imperfect means of holding the tracheal wound open, or from having only cut two rings where an opening through three is necessary. I have never experienced any difficulty in introducing the tube, a good fortune which I attribute to the use of the ligatures for holding the cut edges of the trachea apart and to making a sufficiently large opening. Before the operation is begun, tapes about eighteen inches in length should be sewed to the tracheal tube; when it has been placed in the trachea, these are passed about the neck and tied upon one side so as to hold it firmly in place. In case the wound is too small, it will not do to try to crowd the tube into the trachea, a procedure very apt to force it into the cellular tissue in front; but the soft tissues should be drawn away from the lower end of the wound and another ring cut, if necessary, to introduce the tube easily.

A probe-pointed scalpel is generally used for enlarging the wound and may be employed for making the main cut after a slight puncture with an ordinary scalpel; in this way all danger of cutting the posterior wall and opening through into the œsophagus may be avoided. If the false membrane has extended below the opening, before the tube is inserted an effort should be made to remove all of it that is possible with Trousseau's tracheal forceps, or by passing down into the trachea a feather, or with the forceps a strip of linen one end of which is held by the hand, thus causing the patient to cough and remove the blood and false membrane. The tube having been inserted, the wound above and below it may be drawn together by one or two stitches and covered with a strip of antiseptic gauze drawn under the rim of the collar of the tube to prevent it from irritating the neck. A strip of cloth may then be tied loosely about the neck and a large piece of gauze folded over it and allowed to fall down over the opening of the tube, thus preventing the patient from coughing out blood or mucus upon the bedding and attendants. After the operation is completed, the inner of the two tracheal tubes should be removed and carefully cleaned every half-hour, for the first twenty-four hours, in order to prevent it from filling with inspissated mucus. Subsequently it may be cleaned less frequently, but it should always be borne in mind that it must be kept free. After the operation, the temperature of the room should be kept at about 80° F. and the air moist. If the secretions show a tendency to dry, the patient may inhale from time to time steam impregnated with lime, soda, or the various other remedies already mentioned. Internal administration of medicine calculated to prevent extension of the false membrane should be continued as before. The patients, even when the operation has been done for diphtheria, usually do exceedingly

well for twenty-four or thirty-six hours, and breathe so easily and rest so comfortably that the friends think a cure has been effected; but at the end of this time the development of bronchitis or pneumonia or the extension of false membrane will often evince itself to the physician by increased fever, quickened respiration, and renewed signs of imperfect aëration of the blood. When these symptoms occur, the disease usually goes on from bad to worse until death comes at the end of fifty to seventy hours after the operation. If the case progresses favorably, it will usually be found in from five to eight days that the patient breathes easily with the tube stopped by the finger, or a cork which should be worn some hours before an attempt is made to remove the canula. When this is removed, the sides of the wound, as a rule, readily fall together, and within a few hours no air will pass through the opening. If the wound does not speedily close, all that is usually necessary is to touch it a few times with the solid silver nitrate. Sometimes, after the tracheal canula has been worn for months, it is found on attempting its removal that the patient cannot breathe, by reason of spasm of the glottis or an obstruction from new growths at the upper part of the wound. If granulation tissue is found in the trachea, it must be removed before a cure can be effected, but to overcome the tendency to spasm, no method has yet been found so satisfactory as the introduction of an O'Dwyer tube, which will generally be coughed out, or may be removed within forty-eight hours, and may not be needed afterward. When a tracheal canula has been worn long, it often becomes necessary, especially in a thin subject, to make a plastic operation in order to cover the tracheal wound. This may be best done by paring the edges of the tracheal wound, loosening up the soft coverings freely on each side, then drawing them forward and stitching the edges together. In performing tracheotomy, chloroform is preferable to ether as an anæsthetic, because of the profuse secretion excited by the latter, and it is probable that in these cases it is quite as safe. When carbonic acid poisoning is pronounced, no anæsthetic is needed, but at other times anæsthesia is important, not alone for prevention of pain, but to keep the patient quiet. In adults who are not timid, and in some children, local anæsthesia, quite sufficient, may be obtained by injecting under the skin along the line of incision a few drops of a weak solution of cocaine (Form. 140).

Rapid Tracheotomy.—In extreme cases it sometimes becomes imperative to open the trachea at once; for this purpose various instruments have been devised. Some surgeons recommend that the child be placed upon its face at the side of the table, the trachea steadied with the thumb and finger of the left hand, and the skin, fascia, muscles, blood vessels, and tracheal walls divided with a single cut. This procedure has also been recommended for ordinary cases in place of the careful dissection generally practised, but the danger of hemorrhage renders it extremely objectionable except in those very rare cases where not a

second can be lost, and an intubation set is not at hand. Hook-like tracheotomes consisting of blades that may be opened after the trachea has been perforated, and which will thus cut a sufficiently large opening to introduce the tracheal tube, have also been recommended, but they do not meet with favor among surgeons. An ingenious trocar which enables the operator to leave the canula in the trachea has been devised, but the canula is too small, and I consider it a dangerous instrument, which is likely to cause the loss of valuable time, if not of the patient's life. By most experienced surgeons, tracheotomy is considered a very dangerous operation, because, with the greatest care, serious hemorrhage will sometimes be encountered, and unavoidable accidents may so delay the operation that breathing ceases before it is completed, and it may become necessary to open the trachea hastily before the superficial tissues have been cleared away. For the avoidance of hemorrhage, great care should be exercised in tearing instead of cutting through the superficial tissues, and if by accident a blood vessel is opened it should be caught immediately with artery forceps, and if large it should subsequently be tied and the ligature cut short; if small, it may be twisted sufficiently to prevent hemorrhage. If during the operation the patient stops breathing, at least five or ten seconds may be safely consumed in opening the trachea, providing artificial respiration is then established; therefore the surgeon should not be precipitate in his incision. In these cases the surgeon will sometimes be able, by keeping up artificial respiration, to restore a child apparently dead for fifteen or twenty minutes. There is danger from gradual oozing of blood into the trachéal wound after the tube has been introduced, but usually this is stopped by the introduction of a tracheal canula. Secondary hemorrhage sometimes occurs; if it takes place, the canula must be removed and the bleeding vessels tied or twisted. The danger from the extension of the disease to the lower air passages, and the development of bronchitis or pneumonitis, cannot always be anticipated, but it is best guarded against by care to prevent the entrance of blood or other foreign substance into the air passages, by keeping the atmosphere of the room warm and moist and by the judicious administration of internal remedies. The tracheal canula is not infrequently coughed out; this is best prevented by having a long tube which will pass into the trachea three-quarters of an inch beyond the cut. Many patients have been lost because of secretions collecting and drying in the tube; this can only be obviated by carefully and frequently cleansing the inner tube. A tracheotomized patient must be left in the care of the best possible nurse, and every detail should be carefully watched by the physician until all danger is passed. The prognosis should always be guarded until convalescence is fully established.

CHAPTER XXV.

DISEASES OF THE LARYNX.—*Continued.*

PHLEGMONOUS LARYNGITIS.

Synonyms.—Submucous laryngitis, diffuse abscess of the larynx, laryngitis phlegmonosa, laryngitis submucosa purulenta, laryngitis sero-purulenta.

Phlegmonous laryngitis is a rare affection, in which inflammation attacks the submucous tissues, causing suppuration and necrosis, with the formation of diffused or circumscribed abscesses which are generally located in the upper portion of the larynx at the base of the epiglottis, or in the aryteno-epiglottidean folds. The affection sometimes involves the ventricular bands, and rarely the vocal cords.

Etiology.—The disease may either originate in the larynx or extend to it from the surrounding parts, especially from the pharynx, in which case it is nearly always due to blood poisoning. In many instances the inflammation begins in the cartilages or perichondrium, usually resulting in such cases from typhoid fever or syphilis, or occasionally from other diseases.

Symptomatology.—At first the patient often complains of a sensation as of some foreign substance in the part, soon followed by actual pain, especially upon deglutition. The voice becomes weak or hoarse and may finally be lost, and, as the swelling advances, dyspnœa occurs, which in severe cases gradually increases, causing stridulous respiration, or orthopnœa, cyanosis, and all the symptoms of strangulation. There are frequent violent efforts to clear the throat, but usually no cough. Dysphagia is more or less prominent in proportion to the swelling of the epiglottis which may often be detected by palpation, but this should be practised carefully as there is danger of exciting suffocative spasm of the cords. Upon inspection, the parts are found deeply congested and much swollen, and often the tracheal mucous membrane is involved. In some cases swelling and fluctuation are present.

Diagnosis.—In adults this may be easy from the history of antecedent disease, with gradually increasing dyspnœa, and from the appearance of the parts on laryngoscopic examination. But in children when the larynx cannot be inspected there is some danger of confounding it with laryngismus stridulus, laryngeal polypus, retro-pharyngeal abscess, foreign bodies in the larynx, or diphtheritic laryngitis. We may exclude

retro-pharyngeal abscess by inspecting the fauces and by lifting the larynx, which will relieve the dyspnœa in most cases of abscess of the pharynx, but not in phlegmonous laryngitis.

A history of their entrance and absence of antecedent disease may readily distinguish *foreign bodies*. Compared with phlegmonous laryngitis, *polypus* develops much more slowly, and *laryngismus stridulus* much more quickly, and neither of them is attended by the symptoms of inflammation.

PROGNOSIS.—The disease usually runs a rapid course and terminates fatally in about seventy-five per cent of the cases, from either suffocation or exhaustion.

TREATMENT.—Early in the disease the best remedies are leeches and warm applications to the neck, with steam inhalations, or, instead of these, constant sucking of bits of ice. As soon as there is œdema or a collection of pus, scarification should be employed. Quinine and strychnine in medium doses and potassium chlorate in full doses are indicated, together with nourishing diet and the free use of stimulants. Remedies and food should be given by enema if the patient cannot swallow. Urgent dyspnœa demands intubation or tracheotomy, the latter generally being most efficient in this disease.

ERYSIPELATOUS LARYNGITIS.

Erysipelatous laryngitis is an inflammation of the larynx, usually associated with erysipelas of the tongue and palate. Most cases are either endemic or epidemic. It sometimes results from metastasis of cutaneous erysipelas, or from its extension along the mucous membrane of the nose, mouth, or ear. The inflammation soon terminates in extensive suppuration and sloughing of the intra-laryngeal or peri-laryngeal tissues.

ETIOLOGY.—The pharynx is usually first involved, the disease subsequently extending into the larynx.

SYMPTOMATOLOGY.—The symptoms are fever, local pain and swelling, with difficulty in speaking, dyspnœa, and great prostration. In severe cases these symptoms are usually succeeded by vomiting and finally by delirium. Early in the disease the laryngoscopic appearances are simply those of laryngitis; subsequently sloughs or extensive ulcers will be observed.

DIAGNOSIS.—The diagnosis must be based upon the symptoms and the evidence of inflammation of the same type affecting the skin or the mucous membrane of the mouth.

PROGNOSIS.—The disease usually runs a rapid course, terminating fatally in the majority of cases. According to Cornil (*Archives générales de Médicine*, Paris, 1862) about one-fourth of those cases die in which the inflammation first begins in the larynx, whereas of those in which

the inflammation extends from the pharynx to the larynx about three-fourths die. This result is apparently due to an increase in the constitutional disease marked by extension of the inflammation from the pharynx downward.

TREATMENT.—The general treatment should be the same as for erysipelas of other localities. Quinine and tincture of iron are most useful medicines. Nourishing diet is essential, and stimulants are indicated early. In view of the more recent bacteriological knowledge concerning the materies morbi of erysipelas, agents opposing the development of micro-organisms are indicated; therefore a saturated boric acid spray, and salol and naphthalin internally, are recommended. Shoemaker, in his late work, praises pilocarpine highly, regarding it as almost a specific in the cutaneous erysipelas. In hopes of aborting the attack, ice may be sucked constantly for the first few hours. Gibb reports a case in which applications of a strong solution of silver nitrate, gr. lxxx. ad ℥ i., every six hours cut short the disease. Steam inhalations and anodynes will be useful in relieving pain. Tracheotomy will naturally suggest itself, but it is of doubtful value. Intubation may be tried.

ABSCESS OF THE LARYNX.

Abscess of the larynx consists of a circumscribed collection of pus in the soft tissues. It is very rarely a primary affection, but occurs not infrequently as the result of inflammation of the cartilages or peri-

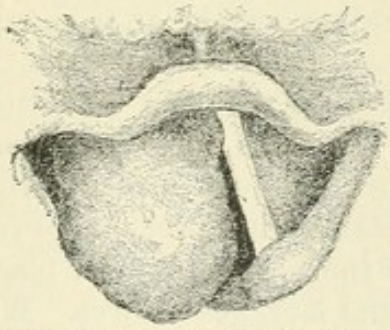

FIG. 117.—PERICHONDRITIS AND ABSCESS OF LARYNX.

chondrium following typhoid fever or pyæmia, or dependent upon tuberculosis, syphilis, or local injuries. Abscesses occurring as the result of typhoid fever are generally found during the second or third week of the fever. The smaller of these appear just beneath the mucous membrane, and the larger ones beneath the perichondrium.

SYMPTOMATOLOGY.—The symptoms of abscess of the larynx are: pain which is aggravated by pressure, cough, dysphonia or aphonia, difficulty in swallowing, and dyspnœa. Upon laryngoscopic examination, the abscess appears as a glistening swelling, red at its base, and either red or yellowish at its apex. It is usually located on the inner

surface of the larynx, either at the base of the epiglottis, upon the arytenoid or supra-arytenoid cartilages, or in the aryteno-epiglottidean folds.

Diagnosis.—In children the disease may be mistaken for croup or retro-pharyngeal abscess, and the diagnosis is sometimes attended with great difficulty. In adults the laryngoscopic appearances are characteristic if the abscess points; otherwise it is not always possible to distinguish it from simple inflammatory swelling.

It is distinguished from *croup* by the history, pain, and difficulty in deglutition; from *retro-pharyngeal abscess* by inspection and palpation of the pharynx; from *acute catarrhal inflammation* by the history, localized inflammation and swelling; from *œdema* by the history, symptoms, and signs; œdema follows renal or cardiac disease instead of inflammation of the cartilages and perichondrium, and it is characterized by a pale, translucent color, and the absence of pain and dysphagia.

Prognosis.—The affection usually terminates in from three days to two weeks and if seen in time and properly treated, most cases recover.

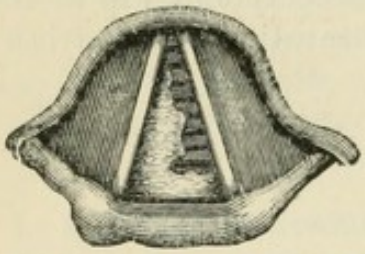

Fig. 118.—Infra-glottic Abscess of Larynx, due to Syphilis. Great dyspnœa.

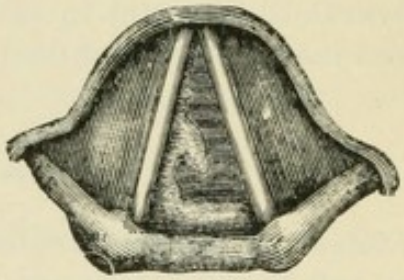

Fig. 119.—The Same as Fig. 118, Twelve Hours after Opening of Abscess.

Sometimes fistulous openings remain after opening of the abscess into the œsophagus or externally; and in the former case liquids or soft food are apt to pass into the larynx during deglutition, causing dangerous spasms or pneumonia. In some cases subcutaneous emphysema has resulted. When the affection proves fatal, death may occur from suffocation or the exhaustion attending prolonged suppuration.

Treatment.—When the abscess can be reached, the pus should be evacuated by means of the laryngeal lancet. When this cannot be accomplished, the patient must be carefully watched, and if dyspnœa threatens, tracheotomy must be performed. Subsequently, with the trachea completely stopped by a large canula, renewed efforts should be made to open the abscess.

ŒDEMA OF THE LARYNX.

Synonyms.—Œdematous laryngitis, sub-mucous laryngitis, supra-glottic or infra-glottic dropsy, œdema glottidis.

Œdema of the larynx consists of a serous or sero-sanguinolent infiltration into the areolar tissue beneath the mucous membrane, which, owing to the formation of the parts, at once diminishes the size of the

air tube, causing dyspnœa, and unless the process is checked or promptly relieved, speedily inducing suffocation.

When the infiltration is of a sero-purulent character, the affection would more properly come under the head of phlegmonous laryngitis.

A spasmodic element frequently coexists with the mechanical interference to respiration, and thus adds greatly to the gravity of the case.

ETIOLOGY.—The trouble may result from simple acute catarrhal inflammation, but most frequently from tuberculosis, syphilis, or Bright's disease. It is sometimes induced by exposure to impure atmosphere, sewer gas, and the like, or by inhalation of extremely cold air; it may follow injuries from foreign bodies and operative procedures or scalds and burns. It occasionally follows small-pox, typhoid fever, and scarlatina, or results from submucous hemorrhage, from erysipelas, or from chronic inflammation of the cervical tissues, and sometimes from the pressure of aneurisms of the larger arteries.

SYMPTOMATOLOGY.—There is usually a history of extreme fatigue, exposure to excessive heat or cold, an injury to the larynx, or of some of the diseases already mentioned. The acute attack not infrequently comes on suddenly during the night, the patient awaking with a sense of discomfort in the throat, or choking. The symptoms increase in severity with great rapidity, giving rise to frequent suffocative attacks, with intervals of less impeded respiration. These intervals grow shorter and shorter until relief is obtained or death occurs. When œdema follows chronic diseases, the progress of the case is more gradual. At first, symptoms due to slight obstruction present themselves. These gradually increase in severity, until finally a suffocative paroxysm occurs, which usually subsides after a short time, to recur after a few hours and again and again at shorter intervals, until it proves fatal. The symptoms referable to the larynx are slight local tenderness, with a sense of dryness, heat, and constriction in the throat, hoarseness, aphonia, dyspnœa with labored and sometimes stridulous respiration, and more or less difficulty in swallowing. The inspiratory act is chiefly obstructed, expiration being comparatively free; this is an important point in the diagnosis. Upon inspection, the fauces are sometimes found to be œdematous; and by the aid of the laryngoscope the epiglottis, or aryteno-epiglottidean folds, or both, are seen to be greatly swollen, and occasionally the ventricular bands or vocal cords are also affected. The affected parts are translucent, of a pinkish or yellowish color, and closely resemble, in their general appearance, an œdematous eyelid or prepuce. The epiglottis has the appearance of a roll or ridge, and the aryteno-epiglottidean folds are globular or irregular in form, and usually project upon both sides; though occasionally only one side is involved, and at other times the swelling is greater on one side than on the other. When œdema results from catarrhal inflammation, the vocal cords are always

of a bright red color, and the other parts even more congested, sometimes showing distended veins upon the surface. When resulting from renal, hepatic, or cardiac disease, the membrane is pale and translucent. In hemorrhagic effusion there is localized swelling of a deep red color. When occurring during scarlet fever, the mucous membrane is apt to be congested in patches of varying shades. In typhus fever the œdematous larynx is usually of a dusky red hue. When inflammation has been excited by irritant poisons, excoriations of the epiglottis can frequently be detected; when caused by scalds, patches of thin false membrane are observed; and when by other traumatic causes intense congestion beginning at the seat of injury is generally present.

Prognosis.—Most cases terminate within five or ten days, but some are more prolonged. About fifty per cent of all these cases prove fatal.

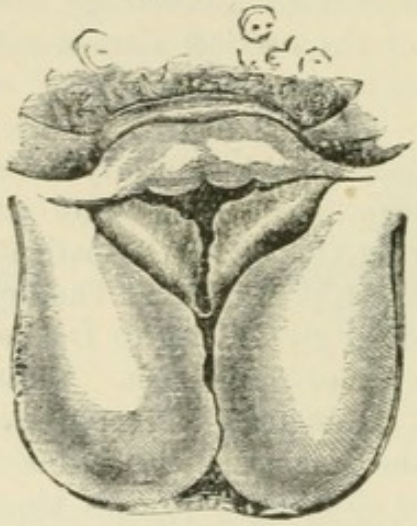

Fig. 120.—Œdema of Larynx (Cohen).

Œdema caused by pharyngeal inflammation usually terminates favorably, but when resulting from inflammation of the cervical tissues it is generally fatal. In œdema of the larynx resulting from syphilis, the prognosis is fairly favorable if proper treatment is adopted. Tubercular cases ultimately end in death, and those due to blood poisoning are nearly always fatal.

Treatment.—Prompt and complete relief is sometimes given by the administration of pilocarpine hydrochlorate which may be used hypodermically in doses of gr. $\frac{1}{8}$. It will cause profuse salivation or diaphoresis, or both, in about twenty minutes. Larger doses cause a profuse and prostrating diaphoresis. Its depressant effect upon the cardiac muscle should always be borne in mind; and when œdema of the larynx attends heart disease, or when the heart is weakened from other causes, this remedy should be exhibited with much care. It often causes vomiting after two or three hours, but this action is also favorable in œdema of the larynx. If we fail with the remedy, scarification of the larynx is the best treatment; when this does not afford relief, tracheotomy or intubation must be performed.

Chronic œdema of the larynx should be treated by scarification, followed by the stronger stimulating or astringent pigments, as zinc chloride or silver nitrate. When the œdema is located below the vocal cords, very little can be accomplished by topical applications. Schrötter's method of dilating the larynx by means of hard rubber tubes of gradually increasing size, which are introduced every day or second day, and kept in position several seconds or as much longer as the patient can tolerate them, has been successfully employed in cases of this kind; but from the limited experience of the past few years, dilatation by O'Dwyer's laryngeal tubes seems the most satisfactory for the majority of cases. If dyspnœa cannot be relieved by these methods tracheotomy must be performed.

CHONDRITIS AND PERICHONDRITIS OF THE LARYNGEAL CARTILAGES.

An inflammation of the laryngeal cartilages or perichondrium seldom occurs as a primary affection. The acute disease is seldom found except in persons of advanced life. The inflammation soon results in more or less caries of the cartilages and thickening of the remaining portions. In severe cases the whole cartilage may be destroyed and thrown off.

ETIOLOGY.—The disease, sometimes primary, is usually the result of tuberculosis, syphilis, typhoid fever, or of trauma. It has been produced by injury done in laryngeal operations, by external wounds, and in rare instances when the cricoid cartilage is ossified, by introduction of the œsophageal sound.

SYMPTOMATOLOGY.—Excepting in traumatic cases, the patient usually first complains of tenderness and pain in the larynx, soon followed by hoarseness and more or less dyspnœa and difficulty in swallowing. The crico-arytenoid articulations are early affected, and as a result there is partial or complete immobility of the vocal cords. Finally, especially after typhoid fever, the consolidation and contraction of the inflammatory lymph may cause permanent anchylosis of this joint. Occasionally a grating or crepitating sensation may be detected on palpation. Until an abscess forms, laryngoscopic examination will often reveal nothing except slight hyperæmia, with very trifling swelling of the parts.

Inflammation of the thyroid cartilage causes some tumefaction of the ventricular bands and of the arytenoid or crico-arytenoid articulations, impairment of the movement of the vocal cords and occasionally subglottic swelling. Inflammation of the cricoid cartilage causes swelling below the vocal cords, which may not be detected at first, but as the disease goes on to suppuration the tumefaction becomes more prominent and sometimes a yellowish spot may be seen as the abscess is about to open. Abscesses of the arytenoids present above and those of the cricoid just below the glottis. Abscesses of the thyroid cartilage usually point below

the glottis, but sometimes externally. When the affection is secondary, ulceration of the mucous membrane may sometimes be first detected, extension of which finally causes inflammation of the cartilage or perichondrium.

Diagnosis.—Primary perichondritis may be suspected when the patient complains of dull aching or boring pain, and laryngoscopic examination reveals enlargement of some of the cartilages without much congestion of the parts. Secondary perichondritis may escape notice owing to swelling of the parts. Late in the affection abscesses are formed, the movements of the vocal cords become impaired, distortion of the larynx may occur without the presence of cicatricial tissue, and often a fetid discharge takes place. From a consideration of these conditions and the history, the affection can generally be easily distinguished from other laryngeal diseases.

Prognosis.—The majority of cases prove fatal. Cases have occurred, however, in which the whole arytenoid or even cricoid cartilages have been thrown off, and recovery has taken place. Usually gradual extension of the disease produces progressive dyspnœa, or the rapid formation of an abscess may cause sudden suffocation unless tracheotomy is performed. When an abscess ruptures, pus may escape externally or into the œsophagus or larynx, and the continued discharge may finally exhaust the patient's strength. Tracheotomy may be performed to avert suffocation; but if recovery takes place, it is probable that the patient will have to wear the tracheal canula during the remainder of life. Even after tracheotomy there are but few who live longer than twelve or eighteen months, but those in whom the disease is not of specific or tubercular origin may live many years.

Treatment.—When the disease is slowly progressing, the patient's general condition demands our first attention. In specific cases the iodides in large doses are of the most importance, and in all cases tonics and nutritious diet are usually necessary. Tracheotomy must be performed when dyspnœa becomes marked, and the lower operation will be most likely to prolong life. If the patient recovers, subsequent attempts at dilatation of the larynx, either by Schrötter's dilators or by O'Dwyer's tubes, should be made, and will sometimes be successful. A fistulous communication between the larynx and the œsophagus demands feeding by the œsophageal tube. Occasionally nutritive enemata must be employed.

TUBERCULAR LARYNGITIS.

Synonyms.—Laryngeal phthisis, throat consumption, helcosis laryngis, laryngeal tuberculosis.

Tubercular laryngitis is a chronic affection of the throat attended by dyspnœa, dysphagia, emaciation, and hectic fever. It is characterized by moderate congestion and swelling of various portions of the larynx

followed by ulceration and severe pain on attempts at swallowing, and usually by a peculiar pyriform swelling of one or both arytenoids or ary-epiglottic folds; which is often pathognomonic.

Anatomical and Pathological Characteristics.—The characteristics vary considerably in different cases and at different times in the same case. Early in the attack there is sometimes simple congestion, but more frequently anæmia. Ere long in most cases swelling of

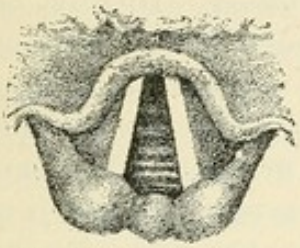

Fig. 121.—Tubercular Laryngitis.

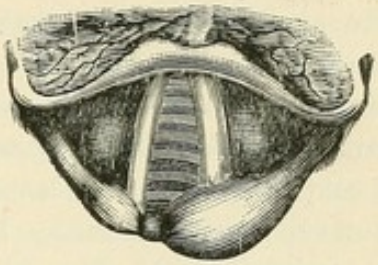

Fig. 122.—Tubercular Laryngitis, showing Pyriform Swelling of Left Ary-Epiglottic Fold and Paresis of Left Vocal Cord.

the soft tissues over the arytenoids from tubercular infiltration gives rise to the pyriform appearance. This swelling may occur on one or both sides, and the epiglottis may also be much swollen or, in rare instances, it may be thickened while the arytenoids remain normal. Shortly afterward, at about the time this swelling takes place, ulcers usually occur on the cords or the ventricular bands, and they may subsequently be found in the upper portions of the larynx. Ulceration in this disease nearly always begins in the lower part of the

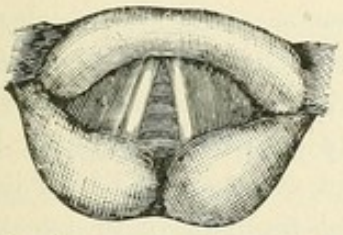

Fig. 123.—Tubercular Laryngitis, showing Pyriform Swelling of Both Ary-Epiglottic Folds and Thickening of Epiglottis.

Fig. 124.—Tubercular Laryngitis.

larynx, subsequently extending upward to involve the arytenoids, the posterior commissure and the epiglottis. The ulcers are superficial and at first small; later these may coalesce, forming large, irregular patches, and they may attain considerable depth when the cartilages are involved. Occasionally the tubercular deposit may be detected before ulceration has taken place; these macroscopic deposits consist of small, yellowish or grayish granules not larger than a millet seed or a pin's head. Not more than two or three of these are likely to be detected, but they are sometimes found in groups. It is probable that in most cases these immediately precede the ulceration. Warty growths are sometimes found about the edges of the ulcer or upon its surface; these are soft,

easily broken down, and have somewhat the appearance of papillomata (Figs. 125, 126). Bosworth describes as one of the phases of the the disease an acute follicular inflammation of the epiglottis which may extend to other portions of the larynx. This is characterized by congestion and swelling of the mucous membrane, with numerous pearly white or gray granulations upon its surface, which at first appear like the follicles in follicular tonsillitis, except that they are smaller. After a short time they rupture, coalesce, and form superficial ulcers. In this way the entire epiglottis may become implicated. In such cases the patient is almost unable to swallow on account of the severe pain, and as a result he declines rapidly, and may die within two or three weeks. Tubercular deposit and ulceration frequently affect the perichondrium or the cartilages. If the latter are affected, necrosis and extensive suppuration are liable to ensue. Paresis of the laryngeal muscles is common, due to atrophy of the fibres or pressure upon the nerve trunks. This

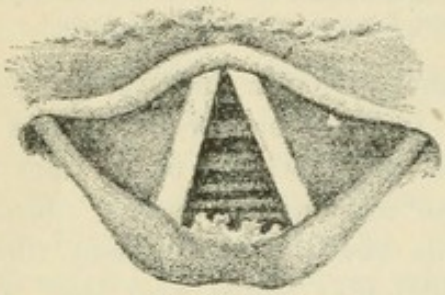

FIG. 125.—INCIPIENT TUBERCULAR LARYNGITIS.

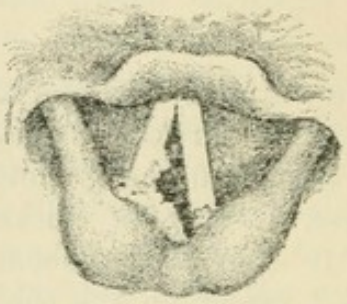

FIG. 126.—TUBERCULAR LARYNGITIS. Granulating tissue resembling papillary tumor.

may occur early in the disease when it is indicated only by weakness of the voice and loss of tonicity of the vocal cords.

ETIOLOGY.—The causes of this disease are the same as those of pulmonary tuberculosis, which generally precedes the throat affection.

SYMPTOMATOLOGY.—The patient usually complains of first having taken a cold, which lasted for some time and was followed by a hacking cough, that may have continued for several months, or in exceptional cases for two or three years. As soon as the disease has made much progress, nutrition is disturbed, and there is gradual emaciation with fever and night sweats. The patient gradually loses strength, the voice is weak, and later when ulceration takes place, and sometimes even before this, deglutition becomes difficult, and even phonation may be painful. The pain on swallowing is liable to grow steadily worse, and finally to become exceedingly distressing.

Indeed, I know of no disease in which the patient suffers more than in the later stage of laryngeal tuberculosis, though in the beginning he may notice only pricking or tickling sensations in the larynx. When the disease is fairly established, the patient has the appearance of one with pulmonary tuberculosis. The skin is sallow, hot, and dry or bathed with profuse sweat, fever of three or four degrees occurs at some part of

the day, and the pulse, which is soft and small, ranges from 100° to 120° F., or higher. Hoarseness is present in about nine-tenths of the cases, and in some there is complete aphonia. Most cases soon exhibit more or less dyspnœa, especially upon exertion, due partly to weakness and partly to obstructed respiration. It is said that laryngeal obstruction occurs in about two and two-tenths per cent of all cases of tuberculosis and becomes so grave as to demand tracheotomy in nearly a third of these. Cough may not annoy the patient much, but usually it is very troublesome. The amount of expectoration is not very great unless the bronchial tubes or pulmonary parenchyma are also involved, but in the latter part of the disease the thick secretions which cover the mucous membrane of the larynx are very difficult to remove and cause the patient much distress. The tongue is coated and often, as in pulmonary tuberculosis, shows smooth, red, oval patches from which the epithelium has been entirely removed. The difficulty in swallowing, varying much in different patients, depends upon the extent and location of

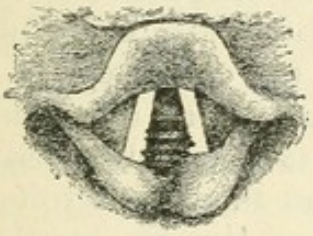

FIG. 127.—TUBERCULAR LARYNGITIS.

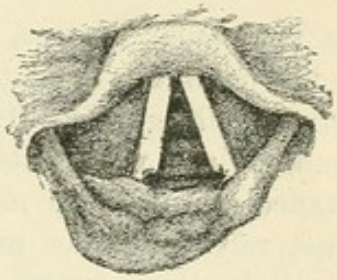

FIG. 128.—TUBERCULAR LARYNGITIS.

the ulceration; in some cases there may be considerable ulceration without difficulty in swallowing; in others a small ulcer will give great pain and prevent taking of food.

When the epiglottis or ary-epiglottic folds are so swollen that the orifice of the larynx cannot be properly closed, fluids find their way into the trachea and excite spasms of cough attended by such distress that the patient prefers to suffer from thirst and hunger rather than to swallow. Anorexia is generally but not always present. Upon examination of the parts very early, there is sometimes simple congestion, but in the majority of cases the mucous membrane is anæmic. Where congestion is observed first, the progress of the case is likely to be slow, but cases where anæmia is pronounced generally advance rapidly. The peculiar pyriform swelling (Figs. 121, 122, 123) of the ary-epiglottic folds is present in a large number of cases; it may be confined to one side or may be found on both, and the epiglottis may or may not be involved. Ulceration of the cords (Figs. 127, 128) or ventricular bands (Figs. 129, 130) is common early in the disease. The vocal cords act sluggishly (Fig. 131) in many cases even before swelling or ulceration, and their movements afterward are often very much restricted.

DIAGNOSIS.—The affection is to be distinguished from anæmia,

œdema of the larynx, catarrhal laryngitis, and from syphilis. The essential points in the diagnosis are the pain, the peculiar swelling, the character of the ulceration, and the physical signs which may be found by examining the lungs.

Tubercular laryngitis is distinguished from *chronic catarrhal laryngitis* by the history and by the physical appearance. In simple chronic laryngitis there is usually diffused congestion with but little swelling.

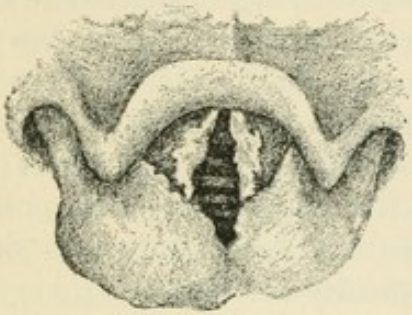

FIG. 129.—TUBERCULAR LARYNGITIS. Ulceration of ventricular bands.

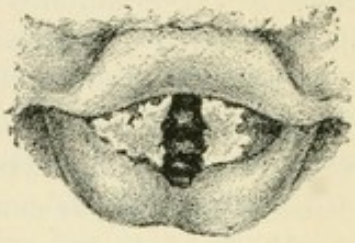

FIG. 130.—TUBERCULAR LARYNGITIS. Ulceration of ventricular bands and vocal cords.

In the tubercular disease, while there may be congestion, more commonly the parts are anæmic, and sooner or later there is the peculiar pyriform swelling (Figs. 121, 122, 123). In the early stage of laryngeal tuberculosis when attended by congestion instead of anæmia, the appearance of the parts may not enable us to make a diagnosis; then we must rely upon the pulmonary signs and the discovery of tubercle bacilli in the sputum. Ulceration is uncommon in catarrhal, but is the rule in tubercular, laryngitis; yet there are rare cases of laryngitis with ulceration, in which it is difficult to determine whether the patient has tuberculosis or not; and in such instances, should we find but

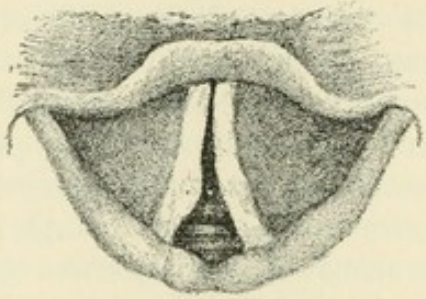

FIG. 131.—TUBERCULAR LARYNGITIS. Paresis of muscles preceding œdema and ulceration.

little change in the physical signs over the apex of one lung, it will be especially difficult to determine whether we have an instance of laryngeal tuberculosis or one of catarrhal laryngitis. I recall two or three obstinate laryngeal cases in which the condition of the apex of one lung aroused my suspicions, though I could not be certain of a deposit, and in whom the ulceration finally completely healed, and the patients remained well for a number of years; apparently indicating that there was no pulmonary tuberculosis. If ulceration occur upon the vocal

cords in front of the vocal process, or upon the ventricular bands, we may generally safely conclude that it is not a case of catarrhal laryngitis; and if the ulceration extends to the upper part of the larynx (Fig. 132), and there is a peculiar pallid or light pink appearance of the tissues, with more or less swelling, we are then certain of our diagnosis.

The disease can be differentiated from chronic catarrhal laryngitis by the following characteristics:

Laryngeal tuberculosis.	Chronic catarrhal laryngitis.
Usually very slight congestion. Parts generally pale, change of contour by pyriform swelling or ulceration. Pain, hectic, rapid pulse, sallow skin.	Congestion of membrane. Usually normal contour of parts. Rarely ulceration. No pain, no fever.
Emaciation.	No emaciation.
Aphonia and dysphagia.	Hoarseness, but no dysphagia.
Sometimes anorexia	No anorexia.
Short duration.	Long duration.
Usually tubercles elsewhere.	No pulmonary complication.

The essential points in *œdema of the larynx* are: semi-transparency of the swollen tissues, and the absence of ulceration and pain.

The distinguishing features are indicated in the following table:

Laryngeal tuberculosis.	Œdema of the larynx.
May be slight congestion of parts.	Usually no congestion of parts.
Early change of contour slight.	Great change of contour by marked swelling, with parts pale and semi-transparent.
Pain, fever.	Absence of pain and fever.
Emaciation.	No emaciation.
Respiration commonly normal.	Labored respiration.
Long duration.	Short duration.

We may be able to distinguish laryngeal tuberculosis from *syphilis* of the larynx, in the first place, by the history, though it is frequently difficult to obtain this satisfactorily. The majority of people who have had syphilis flatly deny it, no matter how much it affects the condition under which they are laboring. In syphilis the larynx is occasionally involved early but usually not until the tertiary stage; although ulceration may occur at the upper part of the larynx in the secondary stage. The margin of a syphilitic ulcer is sharply defined and has an areola of reddened and slightly thickened tissue about it. On the other hand, the tubercular ulcer has a grayish, worm eaten appearance, the border is not regular and well defined, but here and there runs into the sound tissue, and commonly numerous small ulcers are visible about the larger one. In syphilis, ulceration is apt to occur first upon the epiglottis; in tuberculosis, on the vocal cords or ventricular bands. This is not an absolute rule, but holds in a large number of cases. The ulcer in tertiary syph-

ilis is deep, and its sharply cut edge is frequently undermined; in tuberculosis the ulcer is shallow except in rare cases where the process has existed for a long time, but these have not the sharp cut, undermined edges of the syphilitic ulcer. Very often in the latter affection cicatrices may be seen in the upper part of the pharynx or about the fauces and on the soft palate, significant of former ulceration. In the syphilitic affection the pain is not nearly as marked as in the tubercular; many cases of pronounced syphilitic ulceration of the throat occur in which there is no pain, and in others it is slight; while the tubercular ulcer is attended by severe pain, especially on attempts at deglutition. There are, unfortunately, not a few cases in which the tubercular infection has occurred in syphilitic subjects (Fig. 133); giving rise to an atypic ulceration. General evidence of tuberculosis and marked laryngeal pain may be associated with an ulcer of the syphilitic type, and in such cases particularly, the results of treatment must often clear up the

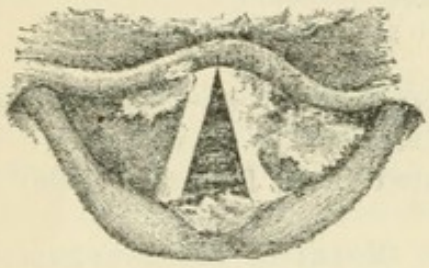

FIG. 132.—TUBERCULAR LARYNGITIS. Superficial ulcers and fungus granulations.

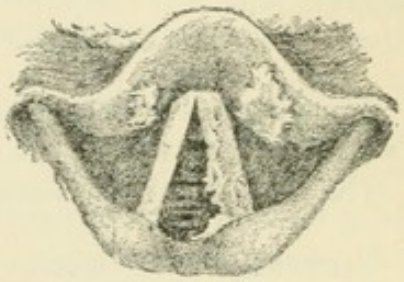

FIG. 133.—TUBERCULAR LARYNGITIS OCCURRING IN PATIENT WITH SPECIFIC HISTORY. Ulceration continued for eighteen months.

doubtful points of a diagnosis. If upon the free administration of antisyphilitic remedies such as potassium or sodium iodide the ulceration begins to heal and the patient to improve, we may be at once satisfied of the character of the disease. There are some cases, however, in which there is undoubted evidence of syphilis, where the patient will not improve quickly, but only recovers after prolonged use of antisyphilitic remedies; therefore, exceptionally, a diagnosis cannot be made until the course of the disease has been watched for some weeks.

Between laryngeal tuberculosis and syphilitic laryngitis the following are the chief points of difference:

LARYNGEAL TUBERCULOSIS.	SYPHILITIC LARYNGITIS.
Generally in adults.	Syphilitic history. Sometimes seen in children, if hereditary.
Ulceration usually superficial, with grayish worm-eaten appearance; usually steadily progresses for three or four months to a fatal issue.	Ulcer sharp cut with indurated and congested border, sometimes undermined. May attain a large size within two or three weeks, but is apt to progress but slowly afterward or may be checked or completely healed.
Comparatively short duration.	Long duration.

PROGNOSIS.—Tubercular laryngitis usually runs a rapid course, many cases terminating within six months. It is claimed that sixty-six per cent die within from six to twenty-four months. In most instances the earlier stages run on gradually, and it is some time before ulceration takes place; when this occurs and is accompanied by difficulty in swallowing, we may expect the disease to run a rapid course, mainly because of deficient nutriment. When extensive ulceration of the larynx is found, we may safely predict that the patient will not live more than eight or twelve weeks. A few cases die within six weeks of the beginning of the disease. It is not now the belief, as formerly, that all of these cases are fatal, for there is ample proof that a few recover. We nearly always find accompanying pulmonary tuberculosis; and it is probably safe to say that where laryngeal tuberculosis is so complicated, nine-tenths of the patients die. Finally, while the local reparative process depends largely upon the ability to better the general nutrition, the hope of cure, as well suggested by Jarvis, should be also based upon the extent of ulceration (Transactions American Laryngological Association, 1883).

TREATMENT.—Constitutional treatment is of the first importance, and should be similar to that for pulmonary tuberculosis. Local soothing applications, in the form of inhalations, sprays, and powders are of more or less benefit. The principal inhalations which are recommended are: the compound tincture of benzoin, camphorated tincture of opium, or solutions of opium or belladonna with or without carbolic acid, or eucalyptol (Forms. 56 to 59). These give some relief, but are not of great importance, for they do not appear to check the disease. Soothing sprays which may be applied cold by the atomizer are preferable when the patient is able to be out of doors, as the warm inhalations predispose to acute colds. Early, before much swelling has taken place, mild astringents such as carbolic acid gr. ij., and zinc sulphate gr. ij., ad ℥ i., or similar preparations are often helpful. These should be applied by the physician every second day when convenient, in sufficient strength to cause smarting for about half an hour, or by the patient twice daily of a strength that will cause some discomfort for only five or ten minutes. Menthol has also been highly recommended as a spray or inhalation in the strength of a drachm to the ounce of liquid albolene.

Wm. T. Belfield has recently communicated to the *New York Medical Record* a preliminary paper on the use of iodine trichloride in surgery; from which I am led to hope for good effects in the local treatment of tubercular laryngitis; and also in the general treatment of pulmonary phthisis.

The demonstrations by W. S. Haines, revealed to him that when brought in contact with saliva, blood, pus, and other animal matter, iodine trichloride is quickly decomposed; setting free iodine and chlorine in the nascent state, most potent for destruction of disease germs. I have used this remedy in many cases of laryngeal tuberculosis applied

by spray in a solution in distilled water gr. ss. to gr. iiss. ad ℥ i., and have used it hypodermically in the manner recommended when speaking of pulmonary tuberculosis for Shurly's solution of iodine. Hypodermically it may be used in solution in distilled water; gr. i. to gr. iiss. ad ℥ i. and ♏ x. to ♏ xx. may be administered.

The results have been favorable, and justify its extended trial in all forms of tuberculosis of the air passages and pleura.

Powders are often better than sprays, because patients generally apply them to the throat more easily. The most serviceable powders are: iodoform, morphine, bismuth, tannin, iodol, and gum benzoin, in various combinations with each other and sugar of milk, starch or acacia (Form. 163–165, 172, 177); an excellent soothing powder is composed of equal parts of gum benzoin and bismuth, with two parts of iodoform. The latter, however, is so exceedingly unpleasant to many patients that it is better to substitute iodol, which has nearly, if not quite, as good effect and has but slight odor. When there is much pain, unless contra-indicated by idiosyncrasy, morphine may be advantageously combined with any of these powders in the proportion of about five per cent, so that the patient will receive one-tenth of a grain with each insufflation. For the same purpose cocaine has been highly recommended, but I have found that it affords the patient very little relief and often proves to be exceedingly uncomfortable. Morphine, iodol, and bismuth, in proper proportions (Form. 165), give more relief than other combinations, in my experience; though a small amount of tannin or gum benzoin may be advantageously added, if not too irritating. If the epiglottis becomes destroyed by ulceration, the patient may need to be fed with an œsophageal tube, which if of small size may be passed without much discomfort. The patients sometimes swallow more easily with the head low in the manner recommended for patients who are wearing the laryngeal tube. They often suffer greatly from thirst and hunger, rather than endure the agony caused by swallowing. For mitigating the torture under these circumstances, I have had great satisfaction from the use, by swab or atomizer, of a pigment of morphine, carbolic acid, and tannic acid with glycerin and water (Form. 139). This applied to the larynx in full strength usually causes intense smarting for a few moments and subsequently so benumbs the parts that the patient may swallow readily, the anæsthesia continuing for some hours. In one case where I frequently used it, anæsthesia would often continue for thirty-six hours. I often give this preparation diluted with an equal quantity of water, for the patient to use by the atomizer two or three times daily. There is now and then a case, in which it only causes suffering. F. D. Owsley, of Chicago, informs me that he has been able to give great relief in these cases by having the patient spray into the larynx, before eating, a saturated solution of oil of cloves (¾ of one per cent) in water. Tracheotomy has been recommended in these cases, not only to prevent dyspnœa, but also to give the larynx rest. With the

latter end in view, it has been advised comparatively early in tubercular laryngitis, but there is no proof that it improves the patient's chances for recovery, and I think it unjustifiable, excepting, of course, when there is marked obstruction of the glottis, in which case it may be the means of prolonging life for several months.

The question of artificial feeding in these cases is ably discussed in a paper by Beverley Robinson, to be found in the Transactions of the American Laryngological Association, 1883.

SYPHILITIC LARYNGITIS.

The local laryngeal phenomena of syphilis vary at different stages of the disease. Syphilitic laryngitis, although frequent, is present in only a comparatively small portion of cases of all varieties of throat disease. Primary syphilitic laryngitis is extremely rare. The symptoms of secondary syphilitic laryngitis make their appearance within from six to twenty-four months after infection, and are characterized by hyperæmia with alteration of the voice and frequently condylomatous formations. The tertiary manifestations do not usually appear until three or four years or much longer after the primary affection, and it is not uncommon to observe cases in which they are delayed fifteen or twenty years. This stage is indicated by gummatous tumors, deep ulcerations, and vicious cicatrices, with consequent dyspnœa and alteration of the voice. Syphilitic patients are more subject than others to acute inflammations of the larynx, which are usually slow to recover. The disease is more frequent in men than in women, and the tertiary symptoms are about twice as frequent as the secondary. In secondary syphilis of the larynx, chronic hyperæmia and superficial ulcers are found, but Mackenzie thinks that smooth, yellow, round or oval condylomata are most characteristic (Diseases of the Throat and Nose, Vol. I, p. 355). These are from five to ten millimetres in diameter, but may be twice as large, and are most frequently found upon the epiglottis or posterior commissure.

Lennox Browne states that he has seen several cases in which these formations were essentially like warty growths (Diseases of the Throat, second edition). There is usually nothing characteristic about the persistent hyperæmia, but, as Browne observes, in many cases there is a well defined, mottled discoloration, apparently less superficial, and not so vivid in color as in simple chronic inflammation. This is most distinct on the vocal cords. Small superficial ulcers or mucous patches are occasionally seen on the ventricular bands, edge of the epiglottis or posterior part of the larynx. These are described by Gottstein as round or elongated, grayish white spots of thickened epithelium, slightly raised above the congested tissue which surrounds them, and either gradually shading off into it or sharply defined. In tertiary syphilis of the larynx, gummata, deep ulceration, cicatrices, or chronic thickening (Fig. 137) are characteristic. The gummata may occur singly or in

groups, and are most frequent upon the posterior commissure or arytenoid cartilages. They are usually observed as round, smooth elevations of the same color as the surrounding tissue, or of a slightly yellowish tint; but as breaking down occurs they usually become yellowish at the centre. The ulceration may be superficial at first, but ere long it becomes deep and destructive. It may occur in any portion of the larynx, but the epiglottis is the most vulnerable point, and frequently

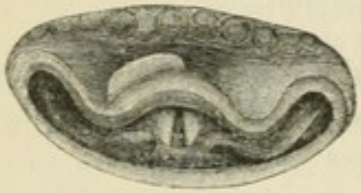

FIG. 134.—CONDYLOMA ON THE UPPER SURFACE OF THE EPIGLOTTIS (MACKENZIE).

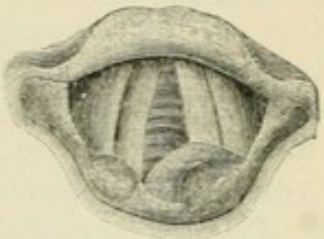

FIG. 135.—GUMMA (MACKENZIE).

it is destroyed by the progress of the disease. When the ulcers heal, resulting cicatrices may seriously interfere with swallowing or respiration. These ulcers are often, though not always, the result of softening of the gummatous tumors. Chronic thickening of the walls of the larynx or of the vocal cords, with anchylosis of the cartilaginous articulations, are among the common results of the disease.

ETIOLOGY.—The affection is due to constitutional syphilis, either inherited or acquired. It sometimes gradually extends from the pharynx, but more frequently occurs after it has disappeared from that locality.

SYMPTOMATOLOGY.—By careful inquiry, a history of some of the manifestations of hereditary or acquired syphilis may generally be ob-

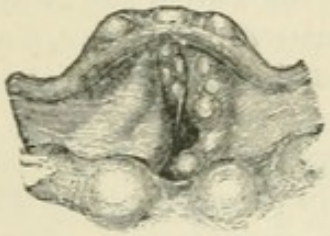

FIG. 136.—MULTIPLE GUMMATA (MANDL).

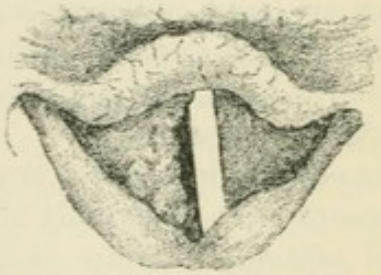

FIG. 137.—SYPHILITIC LARYNGITIS.

tained, though the great majority of patients, if the question is asked them directly, will positively deny ever having been affected. The symptoms will necessarily vary greatly in proportion to the amount of tissue involved and the parts immediately affected. There may be only the symptoms of a slight laryngitis, or, in the advanced disease, difficulty in swallowing, aphonia, or dangerous dyspnœa. Superficial ulcers usually occur in from six to twelve months after primary infection. The condylomata are seldom troublesome excepting as regards the voice, and they often spontaneously disappear. The symptoms of the sec-

ondary disease, as in other parts, rapidly decline under appropriate treatment, but show a peculiar tendency to recurrence. The tertiary symptoms may not occur until many years after inoculation; Mackenzie states that in hereditary cases he has never seen the disease before the seventh year of age. In these unfortunate cases I have seldom seen the disease develop before the person was fifteen years of age; though several instances have been reported of its occurrence in young infants. Even when there is extensive ulceration, patients are peculiarly exempt from pain except on deglutition and occasionally on using the voice, and even then it may be absent if the perichondrium is not involved.

Fever is often present in severe cases, and colliquative sweating may occur in those who are much debilitated. Specific eruptions upon the skin are said to be infrequent in these patients. The voice is easily affected by exposure or vocal exertion, and the singing voice is commonly destroyed. Hoarseness is usual early in the disease, and in many cases there is a peculiar huskiness of the tone said to be quite characteristic. Impairment of the voice may gradually progress until there is complete aphonia; if, however, the disease is limited to the epiglottis, the voice may be but little influenced, and even after complete destruction of this portion of the larynx the voice is sometimes quite restored. Respiration is seldom affected in the secondary disease; but in the tertiary, marked and even dangerous dyspnœa may result from thickening of the parts; or from new growths, anchylosis of the cartilages, or contraction of cicatricial tissues. The dyspnœa may only be noticed on exertion or on the occurrence of acute inflammation, but usually it gradually increases, with frequent exacerbations until eventually life is threatened by exhaustion, by spasm of the glottis, or by suffocative attacks due to collection of tenacious secretions upon the parts. Cough is often present, but it is not usually a prominent symptom in either secondary or tertiary forms of the disease. Early it is occasioned simply by efforts to remove the secretions, and is not peculiar; but when the larynx becomes constricted the cough often acquires the characteristic stridor and spasm of true croup, and when the trachea is obstructed it may closely resemble the cough of pertussis. Constitutional symptoms are usually slight unless the disease in the larynx seriously interferes with deglutition or respiration. The appetite remains good and digestion normal in the majority of cases, but obstinate dyspepsia may be caused by accompanying syphilitic disease of the stomach. In the early stages there is seldom difficulty in swallowing, but in the tertiary form dysphagia is often present, especially where the pharyngeal border of the posterior wall of the larynx is ulcerated. Thickening of the epiglottis does not seem to interfere greatly with the act of swallowing, and sometimes ulceration or even extensive destruction of this valve (Fig. 138) has little effect upon deglutition. Upon laryngoscopic examination, congestion or other changes already mentioned are discovered. The superficial

ulceration of the secondary stage most frequently occurs upon the ventricular bands, the epiglottis, or posterior walls of the larynx. Condylomata, if found, are usually at the posterior commissure, or on the epiglottis. In the tertiary affection the general surface of the larynx is usually of a deep pink or light red color. Gummata have the appearance already described. The superficial ulcer of this stage has sharply defined borders, which distinguish it from tubercular ulceration. The deep ulcer has been well described by Türck, as more or less circular in

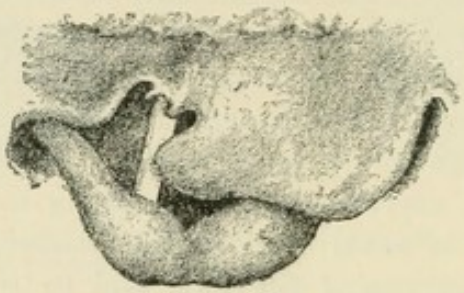

FIG. 138.—SYPHILITIC LARYNGITIS. Partial destruction of epiglottis.

form, with sharp margins sometimes elevated and surrounded by an inflammatory areola. The floor is covered by a dirty yellowish white coating. When the ulcers heal, the resulting cicatrices are dense, fibrous, and unyielding, and exceedingly prone to return if divided. There is usually no external swelling of the larynx, excepting when there is extensive perichondritis, but enlargement of the cervical glands is common.

DIAGNOSIS.—The disease is to be distinguished from simple chronic catarrhal inflammation from tubercular laryngitis, and from benign and malignant tumors. The essential points in the diagnosis are: the history

FIG. 139.—SYPHILITIC ULCERATION OF EPIGLOTTIS. Hypertrophy of left ventricular band and ary-epiglottic fold (Mackenzie).

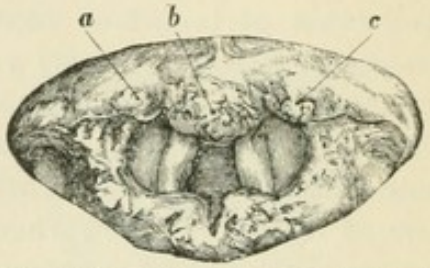

FIG. 140.—SYPHILITIC ULCERATION (TURCK). *a*, *b*, *c*, Remnants of epiglottis.

and absence of grave constitutional symptoms, the presence of scars in the pharynx or upon other parts of the body and of one or more deep ulcers of the larynx. After the surgeon has satisfied himself of the nature of the disease by the appearance of the parts and a cautious inquiry about former symptoms, such as prolonged sore throat, loss of hair, and eruptions upon the body, he should ask the patient, How long since you had syphilis? Put in this way the question is nearly always answered honestly. While there is simply hyperæmia without ulceration it is impossible to arrive at an accurate diagnosis from the examination

of the parts alone, but the discovery of mucous patches or tertiary ulcers, together with the appearance of the pharynx and of the fauces, and the patient's history, with the absence in most cases of constitutional symptoms, will nearly always enable us to make an accurate diagnosis. Sometimes, however, we are obliged to give antisyphilitic treatment for some time before we can be certain of the case.

Between typical cases of *tubercular laryngitis* and syphilitic laryngitis there is little difficulty in making a diagnosis; but when the two diseases are combined, or when the patient is greatly debilitated, it is sometimes impossible to arrive at an accurate conclusion. Usually there is no fever, no excitation of the pulse, and no emaciation in the syphilitic affection, while all of these are present in the tubercular disease. In the early stages of both there may be simple hyperæmia of the parts, but very soon there is a peculiar, pale red swelling in tuberculosis, having a semi-solid appearance much like œdema, instead of the darker red color and dense appearance of syphilitic swelling. The ulcers in tuberculosis are usually comparatively numerous; they are superficial with irregular, poorly defined borders; and are attended by much pain. This is not the case in syphilis. The ulceration is usually rapid in syphilitic laryngitis, slow in tubercular. It is more apt to begin at the upper part of the larynx in the former, and at the lower in the latter. In syphilitic laryngitis, administration of the iodides usually causes speedy improvement, whereas in tuberculosis it is likely to work an injury to the patient, and the symptoms grow worse. Tubercular laryngitis is nearly always attended by distinct signs of pulmonary phthisis.

The rapid growth of condylomata, their location, and, under appropriate treatment, their speedy disappearance, together with other evidences of specific disease, will usually enable us to easily distinguish them from papillomata or other *laryngeal tumors*. The gummata are not likely to be mistaken for any other growths in the larynx. The fungous growths which sometimes occur about the edges of syphilitic ulcers are not likely to be mistaken for any of the benign tumors of the larynx, but are not unlike those which may be observed in some cases of tuberculosis, and can only be distinguished from the latter by a careful consideration of other symptoms and signs.

In the early stages, while there is simple congestion of the larynx, it may be impossible to distinguish *cancer* from syphilitic laryngitis, but congestion in the malignant disease is usually confined to one side or to a limited portion of the larynx, whereas that of the specific affection is more apt to be uniformly distributed. In cancer the growth precedes the ulceration, whereas in syphilis the ulceration is often first. In syphilis the ulceration is more rapid, though there is less inflammation about it, and the ulcers are usually smaller and more apt to be multiple. In the later stages of cancer, when a large, irregular tumor has been formed there can be but little difficulty in making the diagnosis. In

rare cases where there has been much thickening of the larynx, with ulceration and cicatrization so that portions of the organ are much distorted, it is sometimes impossible at first to tell with which disease we are dealing. In these cases, as suggested by Lennox Browne, much reliance may be placed upon the evidence obtained by frequently weighing the patient while he is taking the iodides. Although under antisyphilitic treatment, persons suffering from cancer of the larynx sometimes do well for a short time; improvement soon ceases, and they lose weight; whereas in the syphilitic disease there is generally steady increase in weight for a considerable time while this treatment is pursued.

Prognosis.—In the secondary stage of the disease appropriate treatment usually effects a speedy cure, though the singing voice may be permanently lost. However, there is a peculiar predisposition to relapses under exposure to the causes of catarrhal inflammation. In the tertiary variety a favorable prognosis may be given where the case comes under observation sufficiently early; but if the perichondrium or the cartilages are extensively involved, there is great danger to life. In either case restoration to the larynx of its perfect functions is impossible, though improvement may be expected under appropriate treatment. The ulcerations will usually heal within two or three weeks, but the thickening or cicatrices remaining may interfere with deglutition, respiration, or phonation. Death may result from acute œdema, and has occurred from hemorrhage though this is not a likely termination. Chronic thickening or distortion of the larynx is liable to remain permanent in all cases where there has been extensive ulceration; and gradual exhaustion due to stenosis of the larynx may finally wear the patient out if tracheotomy is not performed. Destruction of the epiglottis may for a short time interfere with deglutition, but the patient soon learns to swallow without this valve.

Treatment.—In the secondary disease, local stimulating applications, similar to those recommended for simple chronic laryngitis, are indicated and are peculiarly beneficial. For this purpose solutions of zinc chloride or copper sulphate have been found most useful. A mild mercurial course is also indicated; and whenever condylomata or ulcerations appear, potassium or sodium iodide should be given. Bitter and ferruginous tonics are indicated if the appetite is fitful. The use of tobacco in any form should be interdicted, and alcoholic stimulants are generally hurtful. In the tertiary form of the disease the greatest reliance is placed upon the internal administration of potassium or sodium iodide. If for any reason these cannot be borne, the patient may be given a mercurial course; gold and sodium chloride sometimes acts equally well. It is sometimes found necessary to use the iodides in very large doses; for example, I have seen a patient in whom twenty grains of potassium iodide taken four times daily had no effect; whereas, when he was given much larger doses the condition of the larynx im-

mediately improved. The remedy should always be given freely diluted with water, and it is best to begin with small doses, which can be steadily increased. I usually begin with seven and one-half grains after each meal and at bedtime, and the dose is increased each day two and a half grains until fifteen or twenty grains are taken at a dose. If with this treatment the patient does not improve, and the symptoms of iodidism do not occur, the dose is increased each day five grains until thirty, forty, or sixty grains, and in extreme cases even one hundred and twenty grains are taken at a dose four times daily. The maximum dose having been reached, it is continued for two or three days, and then the patient again begins with the minimum dose and increases the quantity daily as in the first instance. This plan has seemed to me much more satisfactory than the continued administration of large doses. Usually it is well to direct the patient to drink nearly half a pint of water with each dose of the medicine. Locally, Lennox Browne (Diseases of the Throat, third edition), especially recommends the solid silver nitrate, or, when the epiglottis is ulcerated, the galvano-cautery. I prefer at first the tincture of iodine full strength, thoroughly and accurately applied to the ulcers daily for five or six days, and subsequently less often until healing has occurred. In case the tincture of iodine fails, I resort to copper sulphate in solution of from gr. x. to xx. ad ℥ i., or to zinc chloride in solutions of from gr. xv. to xxx. ad ℥ i. Under this course, even large ulcers will usually heal within two or three weeks. After cicatrization of the ulcers has taken place, if stenosis of the larynx occurs, it must be dilated by means of Schrötter's bougies or O'Dwyer's laryngeal tubes, as described in the treating of stenosis of the larynx. At times the specific medication should be discontinued and tonics substituted. Where the patient is much run down, it is best to administer nux vomica and quinine while the specific course is continued.

SYPHILITIC LARYNGITIS IN INFANTS.

The attention of the profession was first directed to congenital syphilis of the larynx by John N. Mackenzie, of Baltimore, according to whom it is not very infrequent, and occurs mostly within the first year of life (*American Journal of Medical Sciences*, 1880). It is characterized by cough, dysphonia, dysphagia, dyspnœa, and deep, destructive ulceration. The voice of the child may pass through all stages from slight huskiness to aphonia. Paroxysmal cough is frequent, and respiration is more or less embarrassed according to the condition of the part. Laryngismus stridulus is also spoken of by John N. Mackenzie as a not infrequent symptom in these cases. Deglutition is often difficult, and cutaneous eruptions may be present.

Diagnosis.—The diagnosis must be made from the symptoms, and personal and hereditary history; from the signs as manifested upon

the skin or the fauces; and from the appearance of the larynx, when laryngoscopic inspection is possible.

PROGNOSIS.—The prognosis is always unfavorable. The younger the child, the more rapid will be the course and the greater the certainty of a fatal termination. Some cases recover under proper treatment, but there is a strong predisposition to recurrence.

TREATMENT.—The treatment is essentially the same as for the acquired disease; but when difficulty in respiration occurs, prompt intubation or tracheotomy should be performed. The former is to be especially recommended, as it will generally insure sufficient breathing space and give time for the administration of medicine adapted to promote healing of the parts. If stenosis of the larynx occurs, so that it is necessary to wear an instrument permanently, tracheotomy is preferable; but the good results obtained from intubation in chronic stenosis of the larynx would lead me to recommend first a persistent trial of O'Dwyer's method.

CHAPTER XXVI.

DISEASES OF THE LARYNX.—*Continued.*

LUPUS OF THE LARYNX.

Lupus of the larynx is a rare affection said to occur with about eight per cent of all cases of lupus in other parts of the body. It is usually secondary to lupus of the face, is more frequent in women than in men, and is most common in the lower classes of society.

For a history of this disease we are indebted largely to G. M. Lefferts, of New York (*American Journal of the Medical Sciences*, April, 1878). The literature has been much enriched by Chiari and Riehl (Lupus vulgaris Laryngis, *Vierteljahresschrift für Derm. und Syph.*, 1882); Morris Asch, of New York; F. I. Knight, of Boston (*Archives of*

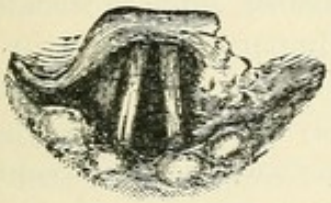

Fig. 141.—Lupus of Larynx (Ziemssen).

Fig. 142.—Lupus of Larynx (Türck). *a*, *b*, Epiglottis.

Laryngology, 1881), and by numerous other writers. Although the various investigators have observed numerous cases, it is not yet possible to point out any diagnostic characteristics of the disease.

Anatomical and Pathological Characteristics.—According to Lefferts, the essential pathological characteristic is hypertrophy of tissue. This is followed by slow but very destructive ulceration, and when healing occurs the cicatricial tissue is very hard and of low vitality. About these scars congested nodules are usually seen.

Etiology.—The causes of the disease are not known. It has generally been considered as an evidence of a scrofulous taint. By some it is believed to be tubercular. The experiments of Koch, in discovering tubercle bacilli in the lupus nodules, and from them obtaining pure cultures, while not furnishing conclusive evidence of the tubercular charac-

ter of the disease, make this the most plausible hypothesis, though the difference in the clinical aspect of the two affections has not as yet been satisfactorily explained. Whatever the ultimate cause of the disease, it is evidently the same as that which causes lupus on other portions of the body. According to Harries and Campbell, the disease requires for its development a suitable soil ("Lupus," etc., London, 1886) —possibly allied to tuberculosis and scrofula; a predisposing cause, particularly traumatism; and an exciting cause, probably a microorganism.

SYMPTOMATOLOGY.—At first the patient may complain of mild sore throat, but the symptoms are not marked and are entirely out of proportion to the physical signs. There is often neither pain nor discomfort, and the patient is usually ignorant of laryngeal disturbance; but as the disease progresses, the voice is often affected and in many cases dyspnœa is developed. In some there is distressing cough and a sense of obstruction in the throat, and occasionally there is complaint of dysphagia. No characteristic physical appearances are observed upon laryngoscopic examination, but in many cases congested nodules will be seen on the epiglottis or anterior surface of the arytenoids. These nodules are irregular or may be almost spherical. Ulcers or cicatrices may also be seen, similar to those observed when the disease affects the face. Ramon de la Sota speaks of marked absence of bleeding from the ulcers (Transactions of the American Laryngological Association, 1886).

DIAGNOSIS.—The disease is to be distinguished from tuberculosis, syphilis, or cancer of the larynx. The most important points in the differentiation are the history and the presence of lupus externally. When the latter exists the diagnosis is not usually difficult, and in young subjects lupus can scarcely be confounded with any disease excepting hereditary syphilis. In cases where the disease is confined to the larynx a diagnosis can only be reached by a careful exclusion of other diseases.

Lupus is to be distinguished from tubercular laryngitis by the characteristics presented in the following table:

LUPUS OF THE LARYNX.	TUBERCULAR LARYNGITIS.
Generally in young adults.	Commonly in middle-aged persons.
Usually associated with disease of the face, and no signs of pulmonary disease.	Nearly always signs of pulmonary disease.
Absence of constitutional disturbance.	Marked constitutional disturbance.
Little, if any, pain.	Severe local pain.
Progress slow and may be arrested.	Progress rapid and seldom arrested.
Ulcers deeply destructive.	Ulcer generally superficial.

Lupus of the larynx is to be distinguished from syphilis as follows:

LUPUS OF THE LARYNX.	SYPHILITIC LARYNGITIS.
Most apt to occur in young adults.	If of hereditary origin, it may occur in children; otherwise it is most apt to occur in middle life, five or ten years later than the advent of lupus.
No syphilitic history.	Syphilitic history.
No constitutional symptoms; absence of pain.	May be marked constitutional symptoms. Frequently no pain, but this symptom may be severe.
Progress slow; aggravated by anti-syphilitic treatment. (Brown, in the third edition of his work, p. 429, remarks that mercurial treatment does not aggravate true lupus, but he appears to contradict this statement on p. 437 of the same.)	Progress may be rapid, but benefit or cure follows anti-syphilitic treatment.

Between lupus and cancer of the larynx the following are the chief points of difference:

LUPUS OF THE LARYNX.	CANCER OF THE LARYNX.
Presence of the disease or the scars which follow it upon the face.	No lesions upon the face.
Usually occurs in early life.	Appears usually after the age of forty.
Slow progress, and may be arrested. Apt to extend over several years.	Comparatively rapid progress, seldom or never arrested, and usually terminates fatally within from twelve to eighteen months, but sometimes extends over four or five years.
But slight pain.	Frequently severe pain.
Slight constitutional disturbance.	Marked cachexia, rapid emaciation and exhaustion.

PROGNOSIS.—The disease progresses very slowly and may last indefinitely, without materially shortening the patient's existence. It is certainly not dangerous to life, but sometimes new formations so obstruct respiration as to demand tracheotomy. Any interference with cicatrices by incision is liable to result in renewed ulceration. The disease may sometimes be arrested.

TREATMENT.—Ferruginous and bitter tonics and cod-liver oil are recommended internally, though their effects are not very apparent. Chemical caustics, of which the solid silver nitrate is preferable, have been used, but not very satisfactorily. The galvano-cautery is recommended by Lennox Browne as the best means of destroying the diseased tissue and promoting a healthy condition of the parts. Thorough scraping and the application of lactic acid, as specially recommended by Ramon de la Sota (*loc. cit.*) are worthy of fair trial. This author also lays stress upon strict hygienic and tonic treatment, arsenious acid giving especially good results. Koch's tuberculine has not been found

more valuable than other remedies, and its use is not infrequently followed by disastrous consequences.

LEPRA OF THE LARYNX.

Lepra of the larynx is an affection which attends some cases of general leprosy or elephantiasis, and is characterized by inflammation and the formation of nodular masses similar to those seen upon the skin. These usually ulcerate and are a cause often of dyspnœa or hoarseness.

Anatomical and Pathological Characteristics.—The disease is attended by congestion of the mucous membrane, with uniform or nodular swelling, and considerable deformity. In advanced cases extensive ulceration may have occurred. In some cases the vocal cords have been found thickened and of a yellowish red color, while the mucous membrane of the ary-epiglottic folds and ventricular bands has been much congested, and has the appearance in some cases of having been loosened from the tissue beneath. In the only case which has come under my observation, the mucous membrane was of a reddish yellow color, the vocal cords had a grayish appearance, and the epiglottis and supra-arytenoid cartilages were thickened, and several nodules appeared on the ventricular bands, epiglottis, and vocal cords.

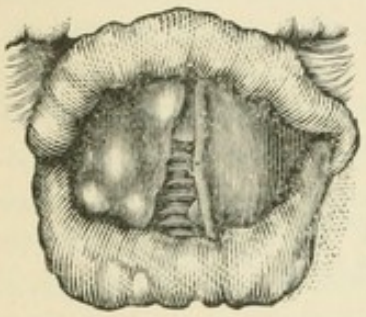

Fig. 143.—Lepra of Larynx. Besides the irregular thickening of the epiglottis and ary-epiglottic folds, five distinct tubercles can be seen on the vocal cords and ventricular band, and one is indistinctly seen on the anterior surface of the infra-glottic portion of the larynx.

There is a tendency of these nodules to ulceration, but, owing to the slow progress of the disease, this stage in many cases is not reached. In some instances great thickening occurs, and very considerable stenosis results.

Etiology.—The causes are the same as those of external lepra, which in nearly, if not quite, all cases precedes the disease of the larynx.

Symptomatology.—There are no characteristic symptoms, but the patient may become hoarse or suffer from dyspnœa, according to the thickening of the laryngeal walls or vocal cords. Pain in swallowing was only observed in one out of twenty-five cases reported by Morell Mackenzie (*Journal of Laryngology*, London, 1887 88). As noted by Lennox Browne, dyspnœa is commonly an unimportant symptom, even in cases of marked stenosis ("Diseases of the Throat," third edition).

Diagnosis.—The diagnosis is based upon the presence of external lepra and the abnormal appearance of the larynx, as already described; also upon the rarity of pain in speaking or swallowing, even though the disease may be far advanced; and on the infrequency of ulceration.

Prognosis.—The prognosis is unfavorable.

Treatment.—Tracheotomy is rarely indicated, but it may be necessary if œdema of the glottis develops. No treatment has yet been

discovered which will surely relieve lepra, but the internal administration of chaulmoogra oil, five to sixty drops daily in an emulsion, has apparently benefited some cases. At the same time an inunction of an ointment prepared from the same oil with five or six parts of lard should be used. In the single case which I have observed, J. Nevins Hyde, of Chicago, employed this remedy with apparently much benefit to the patient.

HYPERTROPHY OF THE LARYNX.

In his work on "Diseases of the Throat and Nose," J. Solis Cohen cites one instance in which all of the tissues were thickened and hypertrophied, but without congestion of the parts; the obstruction of the glottis became so great that tracheotomy was necessary. No cause was known for the disease.

LARYNGITIS DUE TO SMALL-POX.

Laryngitis due to small-pox is always secondary to the eruption upon the skin, and may be either mild, or severe. In the latter case, the exudate interferes with respiration in the same way as diphtheritic membrane in the same locality, and should be treated in a similar manner, intubation or tracheotomy being performed if dyspnœa becomes urgent.

LARYNGITIS OF MEASLES.

Most cases of measles are attended by inflammation of the larynx, either mild or severe. Usually there is simple catarrhal inflammation in the earlier part of the attack, which gradually passes away as the disease progresses; but in some cases, just as the eruption on the skin is disappearing the larynx becomes involved. This form of inflammation is generally very obstinate and may permanently impair the voice. In some epidemics of measles there is a peculiar proneness to a deposit of false membrane in the larynx, occurring, as a rule, from the third to the sixth day. It causes the same symptoms as diphtheritic laryngitis and calls for the same treatment, but unfortunately the majority of these patients die; so great, indeed, is the mortality that some authors have stated that none of them recover even after intubation or tracheotomy. Intubation has seemed to be followed by more favorable results in this particular disease than tracheotomy.

LARYNGITIS OF SCARLET FEVER.

Laryngitis of scarlet fever is a comparatively rare affection which may be simple in character, but is sometimes complicated with œdema of the glottis or with a diphtheritic exudate. In the latter case it should receive the same treatment as diphtheritic laryngitis.

CHRONIC STENOSIS OF THE LARYNX.

Chronic stenosis of the larynx usually occurs in syphilitic subjects, or in persons who have suffered from chondritis or perichondritis resulting from typhoid fever or tuberculosis. It is characterized by more or less alteration of the voice, and dyspnœa in proportion to the narrowing of the glottis.

Anatomical and Pathological Characteristics.—The obstruction usually occurs from vicious adhesions or from the contraction of large cicatrices. The chink of the glottis may have various forms, and in size may vary from the normal to a minute opening scarcely large enough to permit the passage of sufficient air to support life; the parts are usually thickened, hard, and distorted in various ways. The vocal cords, ventricular bands, or the arytenoid cartilages may be more or less adherent to each other.

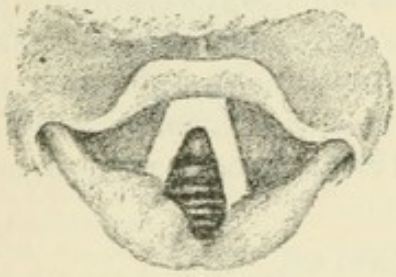

Fig. 144.—Syphilitic Laryngitis. Adhesion of anterior portion of vocal cords, and swelling of arytenoids.

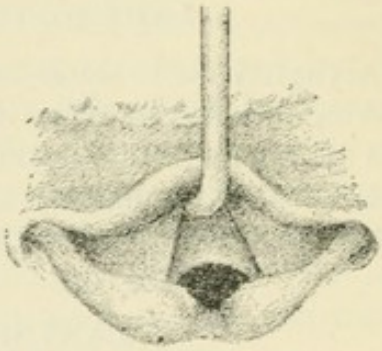

Fig. 145.—Syphilitic Stenosis of Larynx. Adhesion of greater portion of vocal cords.

Etiology.—The disease usually results from syphilis, but it may follow inflammations of the cartilage or perichondrium caused by wounds, typhoid fever, or tuberculosis; in exceptional instances it has been caused by chronic catarrhal laryngitis. The obstruction may be caused by submucous infiltrations or hyperchondrosis, or two or more of these conditions may be combined.

Symptomatology.—In connection with the history of one of the causes already mentioned we may find that the larynx has become involved and that the disease has gradually or rapidly progressed until there is great difficulty in respiration. Sometimes there has been a sudden amelioration of the inflammatory symptoms and apparent improvement of the condition, but the difficulty in respiration has gradually increased owing to the contraction of the cicatricial tissue which has been formed. The voice will be impaired, and respiration obstructed, according to the part of the larynx involved or to the narrowing of the glottis present. Distortion or thickening of the larynx and narrowing of the glottis may be seen upon a laryngoscopic examination.

Diagnosis.—Chronic stenosis of the larynx is to be distinguished

from asthma, compression of the trachea or larynx by tumors or other causes, foreign bodies in the air passages, and paralysis of the abductors of the vocal cords. The diagnosis must usually be based upon the history and the laryngoscopic appearances.

In *asthma*, there is a history of sudden and repeated paroxysms of dyspnœa with more or less complete intermissions or remissions of the attack, instead of the gradually increasing obstruction found in laryngeal stenosis; there are many instead of few bronchial râles and slight, if any, alteration of the larynx.

A history and a laryngoscopic appearance entirely different belong to *foreign bodies* in the larynx.

We are to diagnosticate *tumors pressing on the larynx or trachea* by a careful physical examination of the neck and chest. When this does not succeed, an inspection of the larynx enables us to distinguish between this condition and stenosis.

Dyspnœa, often as pronounced as that of stenosis, is caused by *paralysis of the abductors*. Here again the history must be carefully considered, and upon inspection the position of the cords near the median line, their slight movements with respiration, and the absence of thickening or cicatrices, will indicate the true nature of the morbid process.

PROGNOSIS.—The voice is usually permanently lost, and the disease progresses gradually to a fatal termination unless appropriate treatment is adopted. By proper surgical procedures, however, life may be indefinitely prolonged, though the patient often has to wear a tracheal canula during the rest of his days.

TREATMENT.—Whatever the cause of chronic stenosis, medicinal treatment alone is of little, if any, avail in most cases, for even when of syphilitic origin the disease usually progresses so rapidly that surgical interference becomes imperative. If dyspnœa is great, it is essential that it should be promptly relieved by intubation or tracheotomy, and it is highly advisable that these operations should be recommended early. The anæsthesia for tracheotomy in these cases is best obtained by the hypodermic injection of a few drops of a four per cent solution of cocaine (Form. 140) along the line of incision. If the dyspnœa is not pronounced, Schrötter's laryngeal bougies may be employed for gradual dilatation, but otherwise tracheotomy should be performed unless one of O'Dwyer's laryngeal tubes of sufficient size to give the patient relief can be introduced. After tracheotomy, or when there is no immediate danger to life, dilatation of the parts should be practised by some of the various methods recommended in standard works. The repeated and persistent use of Schrötter's bougies, gradually increasing sizes of which should be introduced two or three times a week, will sometimes prove successful, but the treatment is necessarily tedious, and there is much liability to recurrence of the stricture. Schrötter's, Mackenzie's, or Navratil's dilators may be employed with satisfaction in some cases

(Morell Mackenzie's Diseases of the Throat and Nose), but when adhesions of the ventricular bands or vocal cords have occurred, Whistler's cutting dilator will often be found more satisfactory. O'Dwyer's method

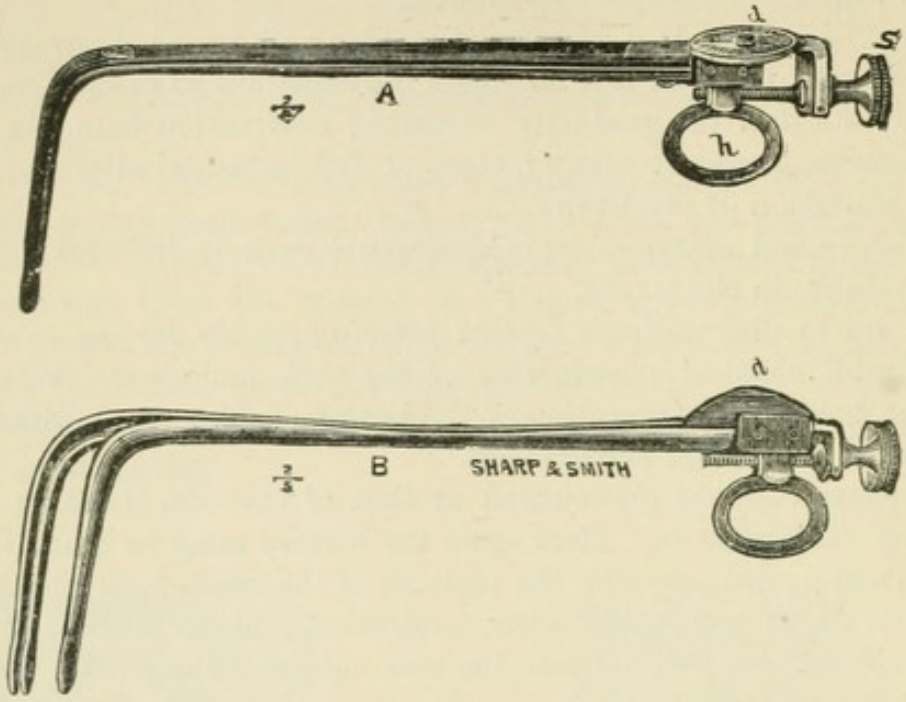

FIG. 146.—MACKENZIE'S LARYNGEAL DILATOR. A, Closed; B, open. The blades may be separated by turning the screw *s*, and the extent of the separation will be registered on the dial *d*.

of intubation furnishes an admirable means of treating chronic stenosis of the larynx. The laryngeal tubes for this purpose are similar to those used for croup. They are ten in number, varying in size just below the head from six to ten millimetres in lateral diameter by nine to nineteen

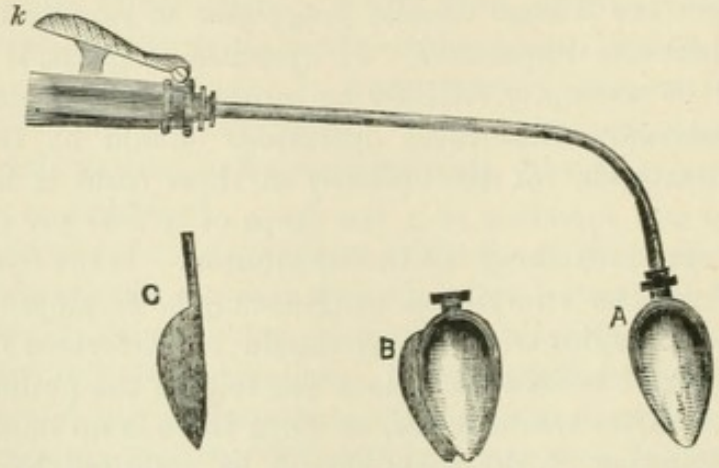

FIG. 147.—WHISTLER'S CUTTING DILATOR. A, Dilator ready for use; B, knife protruding; C, knife; *k*, handle for protruding knife.

millimetres antero-posteriorly. Several cases have been reported where these have given much satisfaction, and I have treated two with excellent results. If the opening of the glottis is very small, it should be enlarged with Whistler's cutting dilator, followed by the laryngeal tube. A tube which can be easily introduced should be worn for a few days at first,

being succeeded by larger sizes from time to time as rapidly as may be without giving the patient discomfort. When the full size has been reached, it should be worn for several weeks, by which time in most cases the tendency to recurrence of the trouble has disappeared; but if contraction occurs, the tube should be worn occasionally to keep the glottis

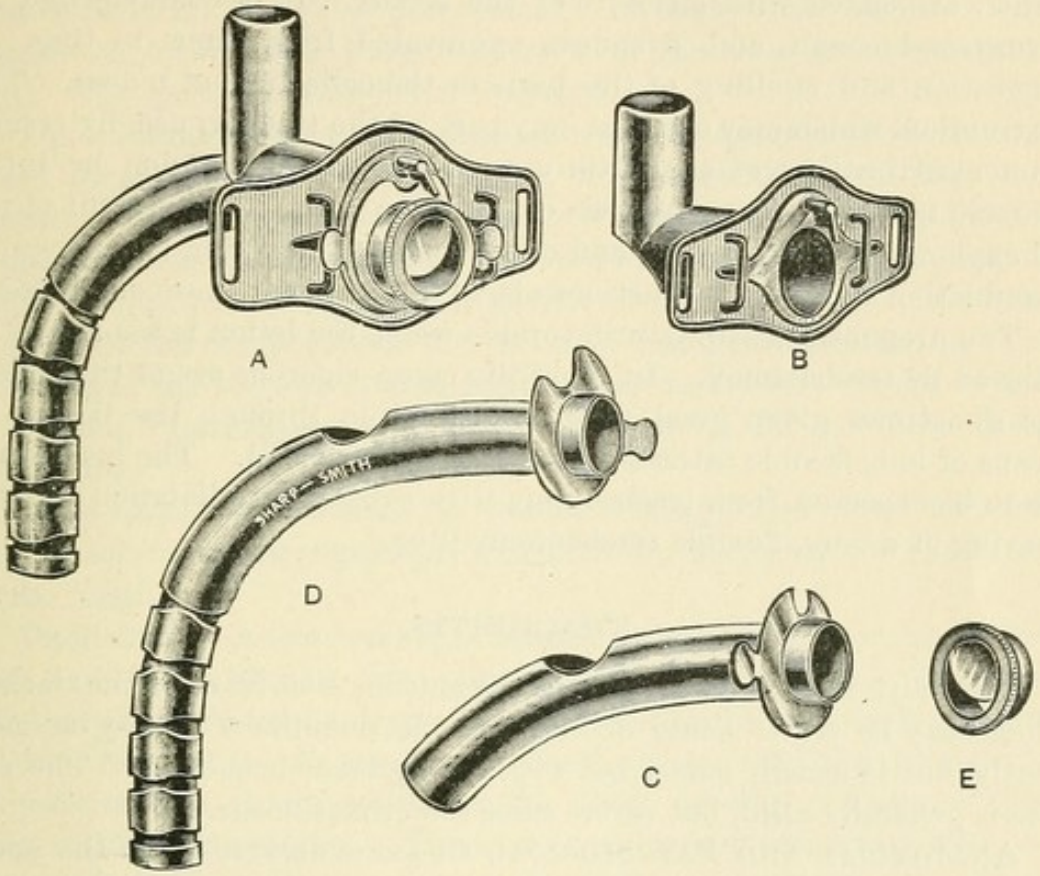

FIG. 148.—TUBE FOR LARYNGO-TRACHEAL STENOSIS. A, Tubes in position; B, outer tube which passes up to the larynx; C, middle tube which passes through the fenestra, in the outer tube, into the trachea; D, inner tube of sufficient length to relieve stenosis low down the trachea; E, valve which opens on inspiration and closes on phonation or expiration.

open. Whatever treatment is adopted, the voice is apt to be permanently impaired. It has seemed to me that continual wearing of an O'Dwyer's tube is more liable to injure the voice than intermittent dilatation. Possibly these tubes might be used for much shorter periods with equally good results in keeping the glottis open, and without so much injury to the voice, but this is a matter to be determined by future experience.

After tracheotomy when the lower portion of the larynx or upper part of the trachea become obstructed by vegetations or cicatricial contractions above the canula, these must be removed. The operation will be facilitated by the punch forceps spoken of when treating of post-tracheotomic vegetations (Fig. 178). The air passage may then be kept open by the combination tube shown in Fig. 148. This tube allows the patient to talk, and may be worn as long as necessary.

Sometimes the constant tendency to contraction will necessitate its retention during the remainder of the patient's life.

STENOSIS OF THE TRACHEA.

The close relation of the larynx and the trachea in some sense compels the discussion of tracheal diseases with those of the larynx.

Stricture of the trachea is a condition frequently, though not constantly associated with stricture of the larynx. . It is characterized by paroxysmal cough and dyspnœa, aggravated from time to time by congestion and swelling of the parts or the collection of mucus. The obstruction, which may occur at any part of the trachea, usually results from cicatrizations of syphilitic ulcers or from compression by intrathoracic tumors. The diagnosis can only be made after careful physical exploration of the throat and chest, and a painstaking laryngoscopic examination whereby obstructions above the vocal cords are eliminated.

The prognosis is always unfavorable when the lesion is too low to be relieved by tracheotomy. In syphilitic cases, vigorous use of the iodides has sometimes given great relief. Dilatation through the larynx by means of long flexible catheters has been recommended. The best results are to be expected from tracheotomy with subsequent dilatation and the wearing of a long, flexible tracheotomy tube.

TRACHEITIS.

Tracheitis is an inflammation of the mucous membrane of the trachea, which may be either acute or chronic. It sometimes occurs independently, but is usually associated with laryngitis or bronchitis. The disease is generally mild, but severe cases sometimes occur.

Anatomical and Pathological Characteristics.—In the acute cases the mucous membrane may be red and swollen, so that the interspaces between the cartilages cannot be seen. In chronic cases the membrane is usually slightly swollen and of a deep pink color, and the intercartilaginous spaces are not very distinct or may be invisible; there are some cases, however, in which post-mortem examination reveals no congestion. In chronic cases masses of mucus may often be seen adhering to the surface, and rarely, ulcers are present. A peculiar form of this disease is sometimes met with in which the mucous membrane is covered by desiccated and decayed secretions similar to those found in the nasal cavity in *ozæna*.

Etiology.—The causes of tracheitis are the same as those of laryngitis and bronchitis. Chronic cases are frequently due to rheumatism.

Symptomatology.—In *acute* cases the patient generally complains of a sense of soreness or rawness in the superior sternal region or at the upper portion of the trachea, with tickling or itching of the part and frequent cough. During the first few days the expectoration is scanty, thick, and tenacious; but as the disease progresses toward recovery, it becomes muco-purulent as in ordinary cases of subacute bronchitis.

In the *chronic* disease there is sometimes localized pain over a small portion of the trachea, but usually simply a sense of discomfort due to swelling of the mucous membrane, dryness, or a collection of mucus upon its surface. Sometimes the tickling sensation is very annoying. These symptoms are associated with a hacking or hemming cough and expectoration of small quantities of mucus usually discolored by dust. Occasionally the cough is paroxysmal. In many cases there is slight hoarseness, or simply a loss of control over the voice on attempting to sing. The general health is not impaired.

Upon examination of the chest, mucous or sonorous râles are sometimes found over the trachea alone, or transmitted over the entire thorax. When the mucous membrane is dry and the secretions are decomposing, the patient is greatly annoyed by constant efforts to clear the trachea, and by an offensive odor similar to that of ozæna. In some of these cases the crusts collect just beneath the glottis and may give rise to spasm of the larynx; in others the symptoms are very similar to those of asthma. Laryngoscopic inspection will reveal the condition already mentioned.

DIAGNOSIS.—The disease is readily distinguished from *laryngitis* and *bronchitis* by laryngoscopic examination, and physical exploration of the chest.

PROGNOSIS.—Acute tracheitis usually subsides in from five to fourteen days. The chronic form may last for several months or even years. The variety attended by drying of the secretions is peculiarly obstinate. Neither form of the disease is considered serious; and the common fear of patients that it may extend to the lungs, causing phthisis, is apparently without foundation. There are some cases, associated with consumption, but this appears to be accidental.

TREATMENT.—The *acute* cases may be given the same local treatment as acute laryngitis, and the internal remedies suited to acute bronchitis. At the same time, cold compresses over the chest in the earlier part of the attack, and hot compresses later, will often be found beneficial. The patient should be kept in as equable temperature as possible, and should avoid exposure. In the ordinary *chronic* cases treatment similar to that employed in chronic bronchitis is applicable, but the greatest benefit will be derived from local applications. Sinapisms or blisters over the sternum are sometimes efficient.

Whenever syphilis exists, or the rheumatic, gouty, or dartrous diathesis is present, these should receive first attention. The local applications which have been found most beneficial consist of inhalations of ammonium chloride with oil of tar or eucalyptol, and the application of various astringent sprays, and stimulating powders. It is difficult to apply a spray to the trachea because the glottis will close as soon as the application touches the larynx, but it may sometimes be accomplished by directing the patient to cough while the spray is being thrown in

quite forcibly. The sprays which I usually employ consist of solutions of zinc sulphate or chloride gr. ii. to x. ad ℥ i., the stronger of these being contra-indicated unless the larynx is also involved. In any case the patient should not experience unpleasant sensations for more than half an hour or at most an hour after the application.

Some physicians favor injecting stimulating solutions with a syringe. Powders have given me the most satisfaction in the treatment of tracheitis, as they can be applied accurately and will remain in contact with the parts longer than solutions. These are used two or three times a week, beginning with mild applications, and gradually increasing the strength as found necessary to produce sufficient stimulation. They are applied while the glottis is wide open by means of a bent glass tube and an ordinary insufflator. Iodol usually has a salutary influence upon the inflamed mucous membrane, and many patients experience speedy relief; from half a grain to two grains may be used at each sitting. A slightly more stimulating powder, and one that answers a good purpose in some cases, consists of equal parts of iodol and boric acid. Where still more stimulation of the parts is desired, I usually combine with the iodol or the boric acid from five to fifteen per cent of alum thoroughly triturated with sugar of milk. Bismuth, gum benzoin, and other powders are occasionally used, but the three already mentioned generally work satisfactorily. Menthol may be used in the same manner, but it has no specially beneficial effect.

Treatment of the fetid form is eminently unsatisfactory. Where the crusts collect close beneath the glottis so as to cause spasm of the larynx, inhalations of ammonium chloride or carbonate, or sodium carbonate, with glycerin and water by means of the steam atomizer, have proved beneficial, the strength being regulated by the sensations of the patient. I have employed a great variety of substances and have had the patient use many different remedies at home, but most drugs seem to have no influence in separating the incrustations or in limiting their formation. The most satisfactory results have been obtained from the frequent inhalation of oil of mustard in combination with alcohol in proportion of about ♏ v. ad ℥ i.; a small quantity of this two or three times daily is poured upon the handkerchief and inhaled by the patient, with the result of enabling him more readily to clear the trachea and finally of greatly decreasing the collection of secretions and the offensive odor.

CHAPTER XXVII.

DISEASES OF THE LARYNX.—*Continued.*

MORBID GROWTHS IN THE LARYNX.

Laryngeal tumors include several varieties of morbid growths similar to those found in many other portions of the body. They are commonly benign, and of these the papillary form constitutes about seventy-five per cent. Next in order of frequency, respectively, come fibrous tumors and fibro-cellular growths, the latter constituting only about five per cent of the whole number of intra-laryngeal tumors. Following these we find cystic, lipomatous, and malignant epithelial and sarcomatous growths; cartilaginous tumors are among the most infrequent. Intra-laryngeal tumors are usually characterized by dysphonia or complete loss of voice, often by dyspnœa and occasionally by dysphagia. They occur most frequently in middle aged men, but they occasionally appear in advanced age, and are seen in children, sometimes being of congenital origin. Previous to the development of laryngoscopy in 1857, only seventy laryngeal tumors had been recorded. Subsequently, up to the year 1871, about three hundred were observed, according to Morell Mackenzie; but since then the number has run rapidly into the thousands, and many of these have been cured by intra-laryngeal operations.

Anatomical and Pathological Characteristics.—The larynx is usually more or less congested, and the tumor may spring from any portion of the organ, though certain parts are especially liable to certain varieties of morbid growth. The appearance of the tumor and its pathological peculiarities depend upon its character, size, and location. Their microscopical appearance is not unlike that of similar neoplasms in other parts of the body, but it frequently happens that it is impossible by such examination to determine the true character of the growth.

Etiology.—Benign tumors nearly always have their origin in continued local hyperæmia; their causation is therefore often the same as that of chronic laryngitis. Cohen believes that they are not infrequently caused by catarrhal inflammation, due to the exanthemata, or to that resulting from croup, diphtheria, pertussis, or the inhalation of irritating substances; he also shows that they sometimes occur in persons suffering from syphilis or tuberculosis (Diseases of the Throat and Nasal Passages). Morell Mackenzie, on the other hand, states that

neither syphilis nor phthisis is a predisposing cause, though he admits that both may give rise to false excrescences or outgrowths (Diseases of the Throat and Nose, Vol. I). He attributes laryngeal neoplasms in many cases to the professional use of the voice.

Symptomatology.—The symptoms of a tumor in the larynx depend upon its size and position, and are essentially the same whether it is benign or malignant. The usual symptoms, which vary, of course, with the size of the growth and the part of the larynx involved, are: cough, dyspnœa, dysphonia or aphonia, dysphagia, and occasionally pain.

Cough is not apt to be troublesome unless the growth is large or involves the glottis, or unless it is attended by bleeding; that which does occur is often paroxysmal and may be of a croupy character.

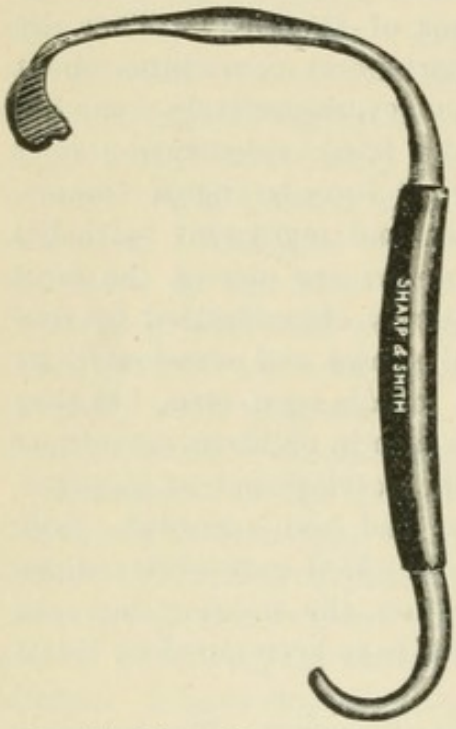

Fig. 149.—Mount Bleyer's Tongue Depressor (¼ size).

Dysphonia or aphonia, hoarseness, or even complete loss of the voice occur when the growth is located on the vocal cords, or when its position or the concurrent inflammation interferes with their vibration. It is surprising how small a growth located on the edge of the cord will cause hoarseness while large tumors differently situated sometimes but slightly interfere with phonation. Sometimes the aphonia is intermittent and it may disappear or change with alteration of the patient's position.

Dyspnœa occurs whenever the neoplasm is sufficiently large to materially obstruct the respiratory passages.

Dysphagia is not a common symptom, but it may occur when the tumor involves the epiglottis or posterior laryngeal wall, or when by its size it encroaches on the pharynx. This symptom is more likely to be present in malignant growths.

Pain is not a common symptom in benign growths, although patients frequently complain of a sense of aching or discomfort, or the sensation as of a foreign body in the throat. Occasionally, even with small tumors on the vocal cords, patients experience slight pain, especially upon deglutition. Severe paroxysms of pain are not uncommon in malignant growths, though even with these it is frequently absent. In adults a laryngoscopic examination will usually at once reveal the presence of a morbid growth, but laryngoscopy is frequently difficult, and sometimes impossible, in young children, especially in those less than six years of age. By forcibly pressing the tongue downward and forward with a tongue depressor similar to that shown in Fig. 149, a good view

may commonly be obtained even in rebellious children. In young subjects the larynx can be readily reached by the finger, and it is often easy to feel the growth, provided it is located above the cords. It is impossible to be certain of the true character of a tumor until it has been subjected to a microscopic examination, and even then the diagnosis may remain doubtful, for sometimes laryngeal growths of malignant histological appearance possess a non-malignant history from beginning to end. Nevertheless, in most cases, inspection of the larynx will enable the physician to practically determine the true nature of the growth.

BENIGN TUMORS OF THE LARYNX.

SYMPTOMATOLOGY.—The most common symptom of these growths consists of alteration of the voice, though this is not invariably present. A growth upon the vocal cord usually causes hoarseness or aphonia, sometimes more marked from small tumors than from large ones. Growths below the cords usually affect the voice by being forced upward

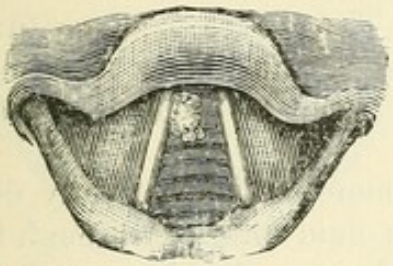

FIG. 150.—PAPILLOMA OF RIGHT VOCAL CORD.

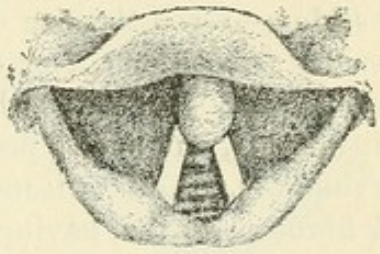

FIG. 151.—PAPILLOMA OF LARYNX. Supra-glottic.

during expiration. Those upon the ventricular bands usually cause no alteration in the intonation. Tumors upon the epiglottis and ary-epiglottic folds do not usually alter the voice unless they become very large.

Cough is not a common symptom, but it sometimes becomes very annoying. Dyspnœa is present in only a small proportion of cases, usually being inspiratory and sometimes paroxysmal. According to Morell Mackenzie, these paroxysmal attacks are due to sudden swelling of the mucous membrane in most cases, but occasionally to an unusual position of the growth. According to Lewin, if the inspiration is noisy and stridulous the growth is probably above the cords (*Deutsche Klinik*, 1862). If interference with expiration occurs, the tumor is usually below the cords. Dysphagia is much less frequent than dyspnœa.

PAPILLOMATA are usually located on the upper surface or on the free margin of the vocal cord, but they may occur in other portions of the larynx. They are generally of a light pink color but may be white or even red. They usually have an irregular, cauliflower or raspberry like surface, and vary in size from a few millimetres in diameter to a mass large enough to completely occlude the larynx They are sometimes pedunculated, but most commonly they spring from a broad base; they

are generally single but not infrequently multiple (Figs. 153, 154). These tumors are usually soft and may be readily crushed or torn off with forceps, but sometimes they are quite firm.

FIBROMATA are usually observed as small, round or oval pedunculated growths (Fig. 155) of a grayish or reddish color, and are most frequently attached near the anterior extremity of the vocal cords. They vary in size from a pin's head to ten or fifteen millimetres in diameter, though

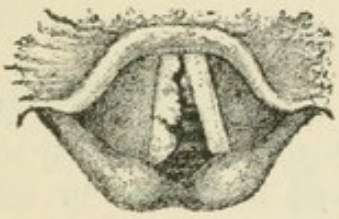

FIG. 152.—PAPILLOMA OF VOCAL CORDS.

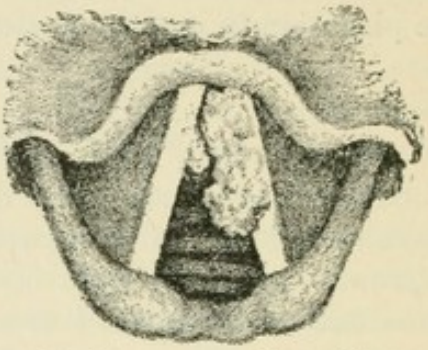

FIG. 153.—PAPILLOMA OF VOCAL CORDS.

they seldom exceed the size of large pea. The surface of these tumors is usually smooth, but it may be rough and irregular; they are firm and resisting when touched with the probe. They are generally, though not invariably, single and pedunculated.

FIBRO-CELLULAR TUMORS consist of more or less perfectly developed fibrous growths, having a serous like fluid diffused through their substance (Fig. 156). They are small, pyriform or globular growths hav-

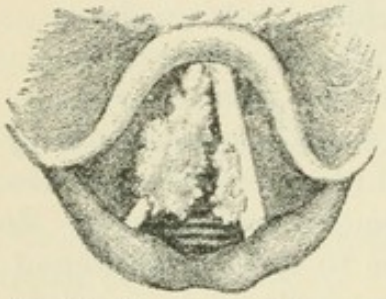

FIG. 154.—PAPILLOMA OF LARYNX.

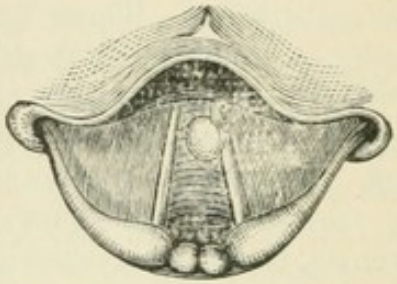

FIG. 155.—FIBROMA OF LEFT VOCAL CORD.

ing a smooth or slightly irregular surface of a pale pink or reddish hue. They are usually pedunculated, but may be sessile, and are generally attached to the vocal cords or laryngeal surface of the epiglottis.

MYXOMATA, or true mucous polypi, are seldom found in the larynx. They are generally of a light gray or pinkish color, commonly translucent; the surface may appear smooth or irregular, and they are soft to the touch.

CYSTIC GROWTHS, when found in the larynx, vary in color from a light yellow to a red, and are usually surrounded by a zone of congested mucous membrane. They are round or oval in form, and generally arise

from the epiglottis or ventricle of Morgagni. They vary in size from three to fifteen millimetres in diameter. They are ordinarily filled with a semi-fluid, sebaceous like material.

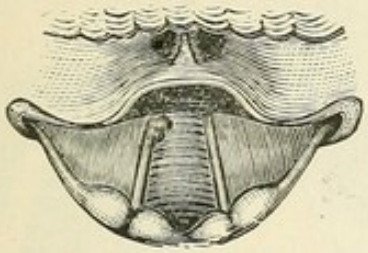

FIG. 156.—FIBRO-CELLULAR TUMOR ON RIGHT VOCAL CORD.

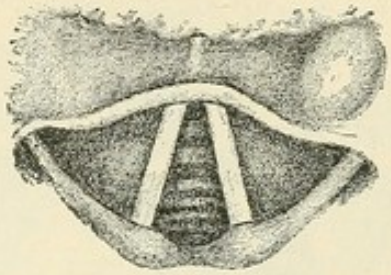

FIG. 157.—CYSTIC TUMOR AFFECTING BASE OF LEFT SIDE OF EPIGLOTTIS.

FASCICULATED SARCOMATA, ADENOMATA and LIPOMATA possess no characteristic appearances, and are extremely rare. They may spring from the epiglottis or mucous membrane over the arytenoid cartilages or other parts outside the larynx, but not usually from within it.

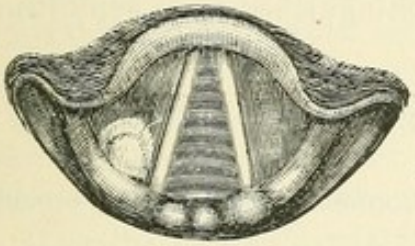

FIG. 158.—CYSTIC GROWTH IN RIGHT VENTRICULAR BAND.

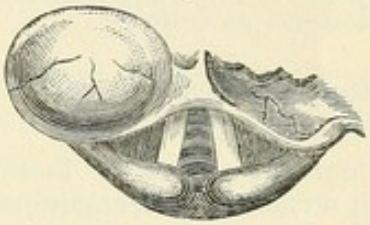

FIG. 159.—CYST OF EPIGLOTTIS (MACKENZIE).

CARTILAGINOUS TUMORS are extremely rare. Fig. 162 illustrates one of this variety growing from the lower part of the thyroid cartilage. It had a smooth mucous covering, was of a yellowish color and cartilaginous consistence.

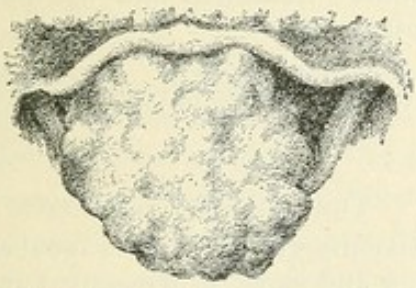

FIG. 160.—ADENOID TUMOR OF THE LARYNX.

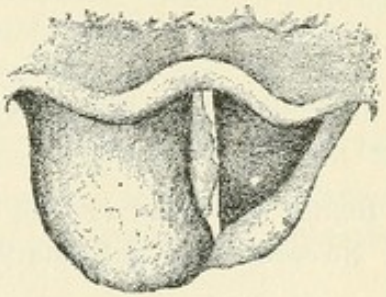

FIG. 161.—ADENOID TUMOR OF LARYNX, INVOLVING VENTRICLE OF MORGAGNI.

ANGIOMATA or vascular tumors are also very rare. They are dark, blackberry-like in color and appearance. They are soft, and bleed easily when touched, and may give rise to severe hemorrhage if removed.

DIAGNOSIS.—Granulation tissue such as is frequently found in tubercular laryngitis might closely resemble papillary growths, but it is

usually lighter in color and softer in consistence, and more or less covered by the same secretions which are seen upon the neighboring ulcerated surfaces. The affections most likely to be mistaken for benign growths of the larynx are syphilitic or tubercular laryngitis, lepra, lupus,

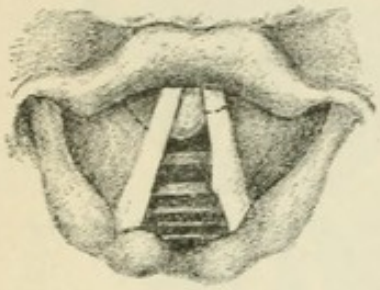

FIG. 162.—CARTILAGINOUS TUMOR OF LARYNX. Situated just below the vocal cord.

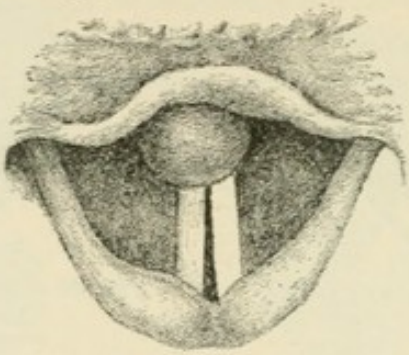

FIG. 163.—VASCULAR TUMOR OF LARYNX, INVOLVING SURFACE OF RIGHT VOCAL CORD.

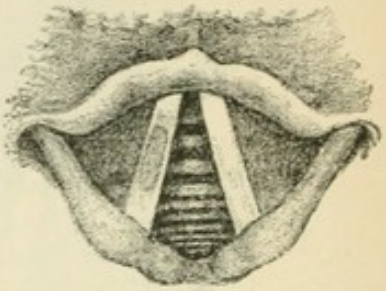

FIG. 164.—VASCULAR TUMOR OF LARYNX. Of a deep livid color and raspberry like surface.

fibrous, cartilaginous, or lymphoid outgrowths, eversion of the ventricles of the larynx, and malignant tumors.

Benign growths of the larynx are distinguished from syphilitic condylomata as follows:

BENIGN GROWTHS OF THE LARYNX.	SYPHILITIC CONDYLOMATA OF THE LARYNX.
Commonly in middle and advanced life; occasionally in children.	Commonly in early and middle life.
History of continued local hyperæmia.	History of infection; appearance five or six weeks after inoculation.
Usually found upon the vocal cords or ventricular bands.	Usually situated at back part of the larynx.
Distinct line of demarcation between growth and surroundings.	No distinct line of demarcation.
Usually no ulceration present.	Ulceration frequently present.
Operative measures usually necessary.	Rapid disappearance under antisyphilitic treatment and use of local astringents.

Benign growths of the larynx are distinguished from tubercular laryngitis as follows:

BENIGN GROWTHS OF THE LARYNX.	TUPERCULAR LARYNGITIS.
No cachexia or pulmonary disease.	Usually grave constitutional symptoms and signs of associated pulmonary affection.
Absence of pain.	Usually painful.
Hyperæmia or normal color of mucous membrane; no ulceration or peculiar swelling.	Pallor of the mucous membrane, with peculiar swelling of the arytenoids and ulceration.
Benign papillary tumors less sessile than tubercular granulations; no purulent secretion.	Tubercular fungous granulations are of light color; appear as thickenings rather than outgrowths; and are associated with ulceration and purulent secretion.

Lepra of the larynx is associated with similar manifestations upon the skin. The epiglottis and lower parts of the larynx are likely to be swollen and nodular, but no distinct tumors are present.

Thickening and nodular outgrowths, which are generally soon followed by ulceration, are caused by *lupus;* and in nearly, if not quite all cases the disease in the larynx is preceded by ulceration on the face or in the fauces, which will materially aid in the diagnosis.

We can recognize *outgrowths* of various character as merely thickening of the tissues, lacking the distinct demarcation of true tumors.

We might possibly mistake *eversion of the ventricle of the larynx* for a tumor, but the condition is so extremely rare that the error is not likely to occur.

Generally *malignant tumors* may be recognized through being more thoroughly blended with the surrounding tissues, which become irregularly swollen and thickened so that the tumor does not stand out distinctly, an appearance very unlike that of benign growths. In some cases where diagnosis by inspection is extremely difficult, the presence of pain, the constitutional symptoms apparent in the later stages, the ulceration of the growth and the microscopic appearances, must all be considered in drawing a conclusion.

PROGNOSIS.—The growths tend to increase in size slowly or rapidly, according to their character, except in very rare instances of papillomata where spontaneous atrophy or expulsion may take place.

Growths in the larynx which cannot be removed are always dangerous, especially in young children, in whom smallness of the organ and disposition to spasm enhance the danger. In children, these tumors are more dangerous than in adults, because of the difficulty of endo-laryngeal operations, and the less favorable results of tracheotomy; an operation which if successful, removes one of the serious dangers by averting the tendency to suffocation. This operation, however, is often grave in young children, and is far from being devoid of danger in adults; for in either, a fatal bronchitis not infrequently supervenes. As regards the voice, the prognosis is favorable where the growth is single and pedunculated and an endo-laryngeal operation can be performed. In the opposite condition the prognosis is necessarily less favorable. Some forms of papillomata show a strong disposition to reproduction after removal. With the exception of sarcomata or carcinomata, other laryngeal growths seldom recur.

TREATMENT.—Small growths in the larynx situated above the vocal cords commonly cause little or no inconvenience, and often, especially when fibrous, enlarge but slowly. In such instances, active interference is unnecessary, provided the growth can be inspected once or twice a year. Even when the tumor is situated upon the cords, causing more or less complete aphonia, it is frequently wise not to interfere, especially in the aged or in those whose occupation renders the voice

relatively of little importance. Even the most skilfully performed endo-laryngeal operations are not entirely devoid of danger, and occasionally they excite sufficient inflammation of the soft parts, cartilages, or perichondrium, to render tracheotomy necessary; and it is possible, though not probable, that the irritation of frequent attempts at removal may cause a benign growth to take on malignancy.

Palliative treatment consists in the application of various astringent remedies, which sometimes apparently retard the growth; and where respiration is seriously impeded in the performance of tracheotomy or the introduction of an O'Dwyer's laryngeal tube. The latter is to be first recommended in most cases, because the pressure which it exerts may possibly cause atrophy of the growth, and the relief of dyspnœa is usually complete except in cases of large tumors at the upper part of the larynx, which may fall over the opening in the tube.

Radical treatment for the destruction or the removal of the growth should in nearly all cases be carried out through the natural passage by the endo-laryngeal method; but in exceptional instances laryngotomy or a combination of the exo-laryngeal and endo-laryngeal methods may be required. The endo-laryngeal removal of neoplasms may be accomplished by chemical or mechanical means, or by a combination of the two. Local treatment by astringents or mild caustics is sometimes beneficial, especially in removing concomitant inflammation, and so possibly preventing increased growth of the tumor. Mild caustics have little effect upon the growth itself, but accurate applications of escharotics, especially chromic acid, are not infrequently followed by most satisfactory results. The same may be said of the galvano-cautery and, with less confidence, of solid silver nitrate. Usually before any endo-laryngeal operation is commenced for the removal of growths, the parts should be thoroughly anæsthetized by several applications, by spray or swab, of a ten per cent to twenty-five per cent solution of cocaine or the solution recommended for anæsthetizing the nasal mucous membrane (Form. 143). This done, silver nitrate or chromic acid fused upon the end of an aluminium probe, and protected to prevent contact with other portions of the larynx, should be accurately applied to the growth with the aid of the laryngoscope. The skilful laryngologist may sometimes apply the escharotic without injuring other parts, by means of an unguarded probe, but it is safer to employ some of the various instruments designed to prevent accidental contacts. The simplest, and to me the most satisfactory instrument is a comparatively stiff aluminium-wire probe, over which has been slipped a section of small rubber tubing about half an inch in length; about this tubing is tied, with a slip-knot, a piece of silk thread which is then wound about the stem and carried up to the handle, thus preventing the possibility of the tube slipping off into the trachea. The tube is slipped upward upon the stem while the caustic is being fused upon the probe and is pushed back to the end of the in-

strument when it has cooled. When it is desired to cauterize with the end of the probe only, the rubber tube is pushed down far enough to completely protect the caustic, for as the instrument is pressed upon the growth the elasticity of the rubber will allow the end to protrude sufficiently. If, however, it is desired to touch the tumor with the side

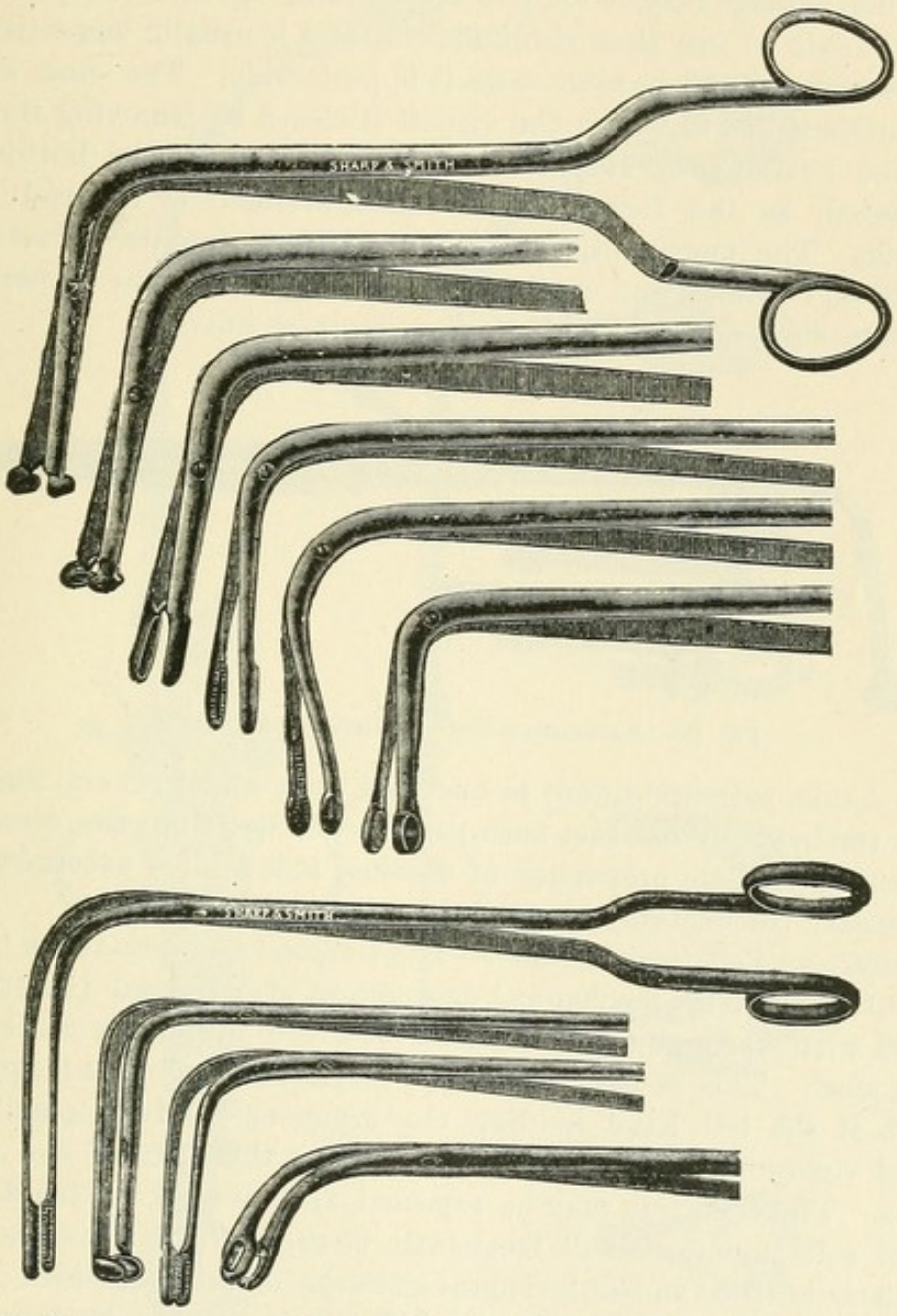

FIG. 165.—COMMON LARYNGEAL FORCEPS (⅓ size). These are grasping and cutting forceps bent at the proper angle, and with beak of the needed length, that the larynx may be reached with ease.

of the probe close to its end, a small piece may be cut out of the rubber tube at this point, which can then be turned so as to expose the proper part. This was shown under trachoma of the vocal cords (Fig. 110).

As soon as the escharotic has been applied, the instrument is quickly withdrawn without injury to other tissues. Various other instruments

have been devised for this purpose, the most satisfactory of which are those recommended by Sajous, of Philadelphia, and Jarvis, of New York.

The galvano-cautery is sometimes an excellent instrument for destroying these growths. It is important that the electrode employed should have a small platinum point which will heat or cool rapidly, otherwise much damage may be done to surrounding tissues. This cautery is more difficult to use than chromic acid, and is usually less satisfactory in its results, though in some cases it is preferable. The most satisfactory handle is one in which the circuit is closed by removing the finger from the button (Fig. 111), instead of one in which the button must be pressed, as the former causes less movement of the end of the electrode. The mechanical treatment of these tumors is carried out by friction, evulsion, and crushing or cutting, which may be performed by various snares, écraseurs, forceps, scissors, or knives.

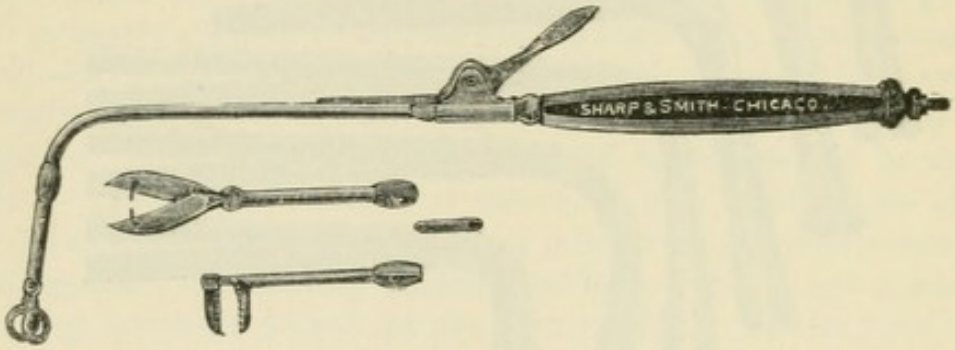

Fig. 166.—Mackenzie's Tube Forceps (⅔ ordinary size).

As a rule, patients cannot be operated upon under general anæsthesia unless tracheotomy has first been performed; but since the discovery of the local anæsthetic properties of cocaine, it is seldom necessary to do a preliminary tracheotomy except in young children.

Friction—Voltolini's Method.—The simplest and sometimes the most efficient measure for mechanical destruction of laryngeal tumors is performed with a sponge firmly fastened to a staff preferably made of malleable steel. This is passed into the larynx, and, with the finger and thumb of the left hand holding the organ as firmly as possible, it is rubbed vigorously up and down for two or three times and then removed. The operation may be repeated after a week or ten days. In case of soft tumors, it will frequently be successful. This operation is peculiarly adapted to the laryngeal growths of infants, which are generally of a papillary character and difficult to remove by forceps. In these patients it is more easily carried out if tracheotomy has first been performed and a general anæsthetic given. The probang may then be carried into the larynx by the aid of the index finger of the left hand, and the treatment accomplished without pain. As a rule, an expert may do this operation without previous tracheotomy, but O'Dwyer's tube or Schrötter's dilator should be at hand for use in case of prolonged spasm of the glottis.

F. H. Hooper, of Boston, recommends operating on these growths by forceps in children, who are thoroughly anæsthetized by ether and held by the nurse in a sitting posture so that the laryngoscope may be readily used. Under such circumstances, previous tracheotomy renders the operation easier, but it is not always necessary (*International Clinics*, October, 1891).

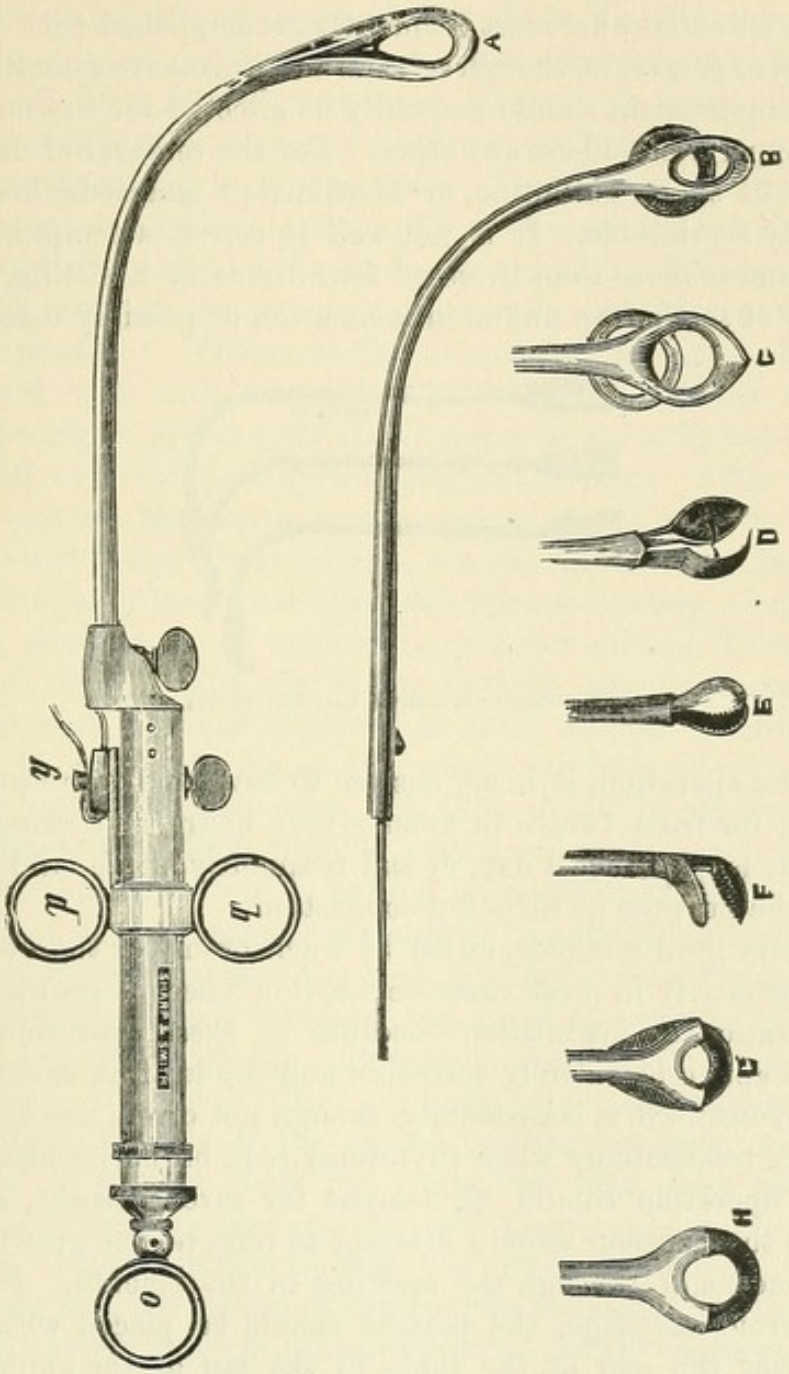

FIG. 167.—STOERK'S INSTRUMENTS: *A*, Écraseur; *B*, *C*, *G*, and *H*, guillotines of various size and form; *D*, *E*, *F*, forceps blades of different kinds.

Evulsion is effected with various forms of snares, forceps, or écraseurs. The snare forceps of Jarvis (Transactions of the American Laryngological Association, 1886) may be useful for removing growths below the cords in some cases. Evulsion is the method most commonly adopted and is most applicable to comparatively soft growths.

Crushing may sometimes be accomplished with stout forceps, and is especially applicable to firm growths where undue force would be necessary for their evulsion. Not infrequently a tumor which has been firmly nipped with forceps will be found to atrophy and completely disappear within two or three weeks.

Cutting operations are most frequently accomplished with cutting forceps, snares, or écraseurs, though scissors and knives are sometimes useful. A guarded instrument should generally be selected for the purpose, and none but experts should use any other. For the removal of firm growths some form of snare, guillotine, or Mackenzie's guarded-wheel écraseur is peculiarly serviceable. It is not well to repeat attempts at removal of these tumors more than three of four times at a sitting, because of the danger of setting up undue inflammation or possibly œdema.

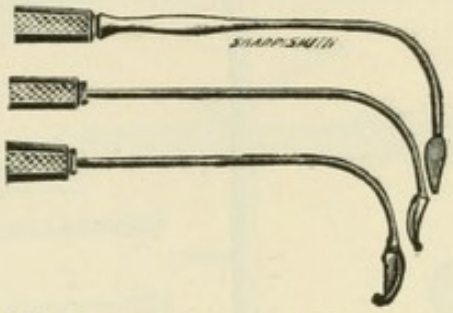

FIG. 168.—TOBOLD'S LARYNGEAL KNIVES (¼ size).

After the operation, it is my custom to have cold applications made to the neck for from twelve to twenty-four hours, and subsequently to apply to the larynx once a day, or less frequently, some mild astringent spray for the purpose of reducing congestion.

Extra-laryngeal methods, either by tracheotomy or thyrotomy, are of doubtful propriety in most cases—excepting where a growth interferes with respiration or deglutition—because by these operations the vocal function is apt to be entirely destroyed and life is often endangered.

THYROTOMY.—It is occasionally, though not often, necessary to do a preliminary tracheotomy when thyrotomy is to be performed, but then the latter operation should be delayed for several weeks, and in the mean time the surgeon should attempt to remove the growth by endo-laryngeal means or through the opening in the trachea. For division of the thyroid cartilage, the patient should be placed with the head hanging over the end of the table, in the lap of the surgeon, who is seated at the end of the table with his back to the window. The primary incision is made in the median line from the cricoid cartilage to the thyroid notch. The thyroid cartilage should then be carefully divided with a strong knife or, if ossification has taken place, with a small circular or convex saw. If possible, a small portion of the upper part of the thyroid cartilage should be left intact, in order that the parts may be accurately approximated afterward, so as to maintain the proper

relation of the vocal cords to each other. In order to avoid paroxysms of coughing, great care should be exercised that the instrument does not penetrate through the mucous membrane into the larynx before the cartilage has been thoroughly divided. When the division is complete, the alæ should be drawn apart by blunt pointed retractors. If this cannot be done, the crico-thyroid membrane should be divided along the lower border of the thyroid cartilage, on one or both sides as may be found necessary. The division of this membrane, however, is quite apt to injure subsequent vocalization, owing to the direct continuity of the vocal cords with it, as pointed out by Joseph Leidy (Transactions of the American Laryngological Association, 1886). If the opening still remains too small the thyro-hyoid membrane should be divided along the upper border of the thyroid cartilage, but this is not generally necessary, and should be avoided if possible. When a sufficient opening has been attained, the alæ are held back with retractors, the cavity is carefully cleansed of blood, and under a bright light the tumor is seized with hook or forceps and torn off or divided with strong curved scissors. After the growth has been removed, Mackenzie recommends that the base be thoroughly cauterized with solid sliver nitrate, which, he states, is less liable to cause a subsequent laryngitis than the galvano-cautery, or other escharotic, and seems quite as efficacious on a raw surface (Diseases of the Throat and Nose). The alæ of the thyroid are then carefully approximated and fastened together in their normal position by two silver sutures, and the edges of the wound carefully closed. If tracheotomy has been previously done, the tube should be allowed to remain until all danger from laryngitis has passed and the surgeon is confident that no other operation will be needed for destruction of the growth.

Sometimes the firmness of the tumor or its extensive attachments prevent perfect removal, so that the operation must be abandoned without being completed; in such instances, as much as possible of the tumor should be removed, and the cut surface thoroughly cauterized with silver nitrate. Krishaber (Tait's Cliniques de Laryngotomie, Paris, 1869) says that division of the cricoid cartilage is never necessary for the removal of tumors above the cords, and that those below can be easily removed through the crico-thyroid membrane or the opening in the trachea. The operation, though not extremely difficult, is attended by some degree of immediate or consecutive danger to life from primary or secondary hemorrhage or inflammation of the air passages; therefore it should not be undertaken without due consideration of the possible consequences. Mackenzie has shown that in the majority of cases the voice is lost, and that the tendency to recurrence is quite as great as when the growth has been removed through the natural passages.

SUPRA-THYROID LARYNGOTOMY is accomplished by a transverse incision through the superficial tissues and thyro-hyoid membrane, either along the lower border of the hyoid bone or the upper border of the

thyroid cartilage. It is less dangerous than division of the thyroid cartilage, but it is of very little service, because the growths which could be removed by this method can usually be equally well removed through the mouth.

INFRA-THYROID LARYNGOTOMY, that is, through the crico-thyroid membrane, according to Mackenzie, has been strongly recommended by Paul Bruns for the extirpation of growths originating from the free borders or under surface of the vocal cords, or below the glottis, provided previous endo-laryngeal operations have been unsuccessful. Sometimes division of the membrane alone is sufficient, but large or sessile tumors may require division of the cricoid cartilage or of some rings of the trachea also. The operation is done in the manner recommended for crico-thyroid laryngotomy, but all soft tissues are carefully dissected out from the crico-thyroid opening, so that only its cartilaginous borders remain. A canula is then inserted and allowed to remain for several days until acute inflammation has subsided; it is then removed, the head is thrown back so as to make the opening as large as possible, the growth located by an infra-glottic mirror, which is then removed, and the tumor is torn off by short forceps. When the crico-thyroid opening is too small, tracheotomy should be performed in the first instance. After the inflammation has subsided, the edges of the wound should be drawn back and the attempt made to remove the tumor. The patient should wear the canula for a few months afterward, until the surgeon is convinced that recurrence will not take place.

MALIGNANT TUMORS OF THE LARYNX.

The term cancer of the larynx embraces a variety of tumors of which epithelioma is by far the most frequent, and sarcoma next. Fauvel, Cohen, Bosworth and Gottstein also recognize medullary or encephaloid, and scirrhous, as possible varieties of cancer in this locality. Such growths give rise to hoarseness, dyspnœa, pain, sometimes dysphagia, and finally, in most cases, to that peculiar cachexia which generally attends malignant tumors.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—The growth of these tumors is first manifested by localized hyperæmia, with thickening of the parts which gradually increases, progressively involving all the subjacent tissues in the cancerous process. By a process of cell proliferation a large irregular tumor is formed intimately blended with the surrounding structures and early undergoing ulceration, which ultimately causes deep and widespread destruction of the parts. The microscopical appearances of these growths, and their causes, are similar to those of like growths in other localities.

SYMPTOMATOLOGY.—The symptoms vary with the size, location, and condition of the growth. Pain, usually lancinating in character, is com-

monly present. This, at first, is generally confined to the larynx, and is not particularly severe, but after ulceration occurs, it becomes intense and frequently radiates to the ears and occasionally to the submaxillary and cervical glands. Mackenzie states that early external evidences of laryngeal cancer are seldom present (Diseases of the Throat and Nose).

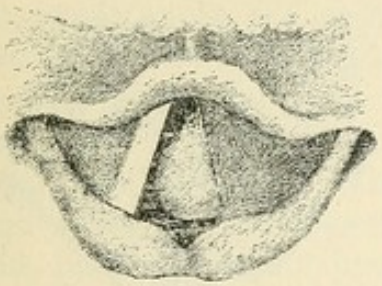

FIG. 169.—CANCER OF LARYNX. Subglottic.

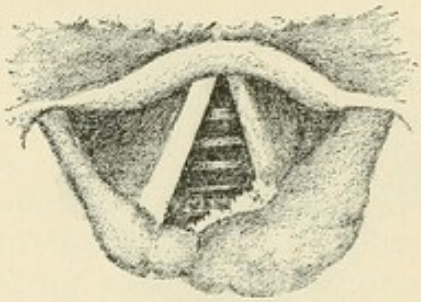

FIG. 170.—CANCER OF LARYNX. Ary-epiglottic fold.

In most cases after the disease has progressed for a few months the submaxillary or cervical glands, especially those near the cornua of the hyoid bone, will be found affected, and undue prominence of the thyroid cartilage may be seen or felt. In rare cases ulceration extends to the surface. Hoarseness is an early symptom, but the voice is seldom entirely lost. Dyspnœa on exertion is frequently an early occurrence; and later may be constant or subject to severe paroxysms. When ulceration has taken place, usually the breath has a peculiar fetor which is

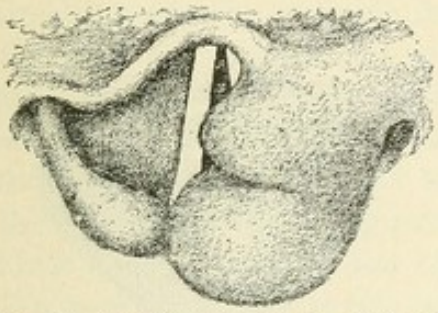

FIG. 171.—CANCER OF LARYNX. Epiglottis and Arytenoid.

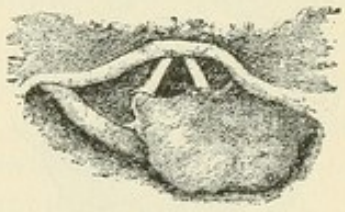

FIG. 172.—CANCER OF LARYNX. Arytenoid.

almost diagnostic. Sensations as of a foreign body in the throat cause frequent efforts for its expulsion, but cough is not a prominent symptom. The amount of secretion from the ulcers themselves is not very large, but there is profuse salivation which causes the patient great inconvenience or distress. The sputum consists of muco-pus, frequently tinged with blood; sometimes there is profuse hemorrhage. Dysphagia often attended by some pain is an early symptom with pharyngo-laryngeal epithelioma. When the disease is confined to the interior of the larynx, this symptom is not experienced so early, but later it is always present.

Upon inspection the neoplasm appears at first as a circumscribed

area of congestion and submucous thickening, the borders of which are not well defined. Usually it is located upon one of the ventricular bands; but occasionally the vocal cords, epiglottis, or ary-epiglottic folds are first affected. In color the growths vary from light red to scarlet. Epitheliomata usually have the deeper hue. The most characteristic

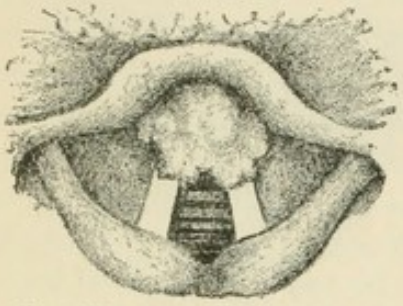

FIG. 173.—CANCER OF LARYNX. Supra-glottic.

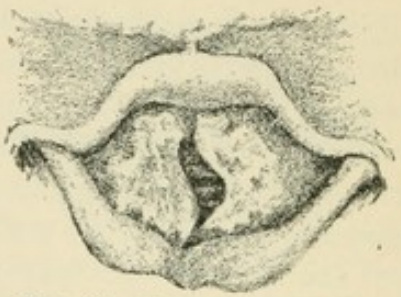

FIG. 174.—CANCER OF LARYNX. Ventricular Bands.

feature of malignant tumors in the larynx is the great deformity which attends their progress. As the process of proliferation and infiltration of the surrounding tissues advances, the growth which at first appeared as a limited area of submucous thickening without well defined borders, presents a raised and irregularly nodular surface. These tumors may be single or multiple, and usually attain a large size—two or more centimetres in diameter. Laryngeal sarcomata are soft, light in color, bleed easily, and ulcerate early. In epithelioma this process may be

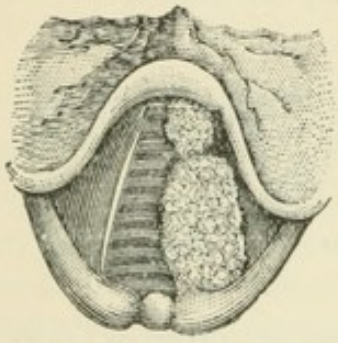

FIG. 175.

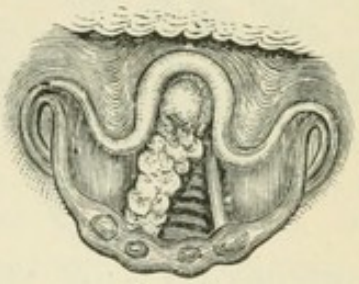

FIG. 176.

FIG. 175.—MIXED SARCOMA. This tumor was found in a man about fifty years of age, who had been troubled with dysphonia for about two years, and with some dyspnœa for a few months. The growth was so firm as to resist attempts at evulsion or crushing. I. N. Danforth made a microscopic examination of some portions which I removed, and pronounced it a mixed sarcoma.

FIG. 176.—CANCER OF THE LARYNX. Vocal Cord. This growth was supposed to be a simple papilloma, but a microscopic examination showed it to be of a semi-malignant character. About four weeks after its removal, the disease appeared in the ventricular band and ary-epiglottic fold, and ran a rapid course.

long delayed. In either case, whether occurring early or late, the ulceration steadily progresses without any attempt at repair. Where both the pharynx and the larynx are involved, ulceration usually first occurs at the free edge of the epiglottis or on the glosso-epiglottic or ary-epiglottic folds, and quickly extends to the deeper portions of the larynx. The epiglottis is frequently so much swollen that the lower portions of

the larynx cannot be seen, but occasionally it is slowly destroyed without much tumefaction. Ulceration usually commences at a single point, though sometimes two or more ulcerated spots may be seen in the beginning. When the disease is advanced, a large surface or the whole mass of the tumor appears in a state of fungous ulceration, bathed in an offensive, purulent secretion.

DIAGNOSIS.—In the early stages an accurate diagnosis of cancer of the larynx is often difficult and may be impossible, but as the disease progresses it can generally be readily recognized by the experienced laryngologist. Cancer of the larynx is to be distinguished from syphilis, chronic catarrhal inflammation, lupus, tubercular laryngitis, and benign growths. The essential points in the diagnosis are: the age of the patient, the pain, irregular thickening with marked deformity, extensive ulceration, glandular enlargement, and the microscopic appearance.

Cancer of the larynx is distinguished from *syphilis* by the history, the absence of cicatricial tissue, the more or less distinct tumor instead of simple thickening, the progressive ulceration in spite of treatment, and, in some cases, by the cancerous cachexia and by the effect of the iodides on the body weight. In tertiary syphilis free administration of the iodides, as a rule, is speedily followed by increase of weight, with other evidences of general improvement; whereas in malignant disease, although at first slight improvement may apparently follow the administration of these remedies, it is soon observed that the weight is steadily diminishing and the strength failing.

Great thickening seldom, and large ulcerating tumors never, arise from *chronic catarrhal inflammation* of the larynx, although occasionally considerable thickening and deformity of the parts is present; but in these instances the history of long continued inflammation and absence of the peculiar lancinating pain, of deep ulceration, or of a malignant cachexia and of the glandular enlargement establish the diagnosis.

We have in *lupus* a slowly progressive disease occurring most often in young subjects; its development in the larynx is preceded by its appearance upon the face or fauces. It is attended by little or no pain and comparatively slight swelling. The ulceration progresses but slowly, and repair may follow at some points. There is not the cachexia which is frequently witnessed in the patients of more advanced age suffering from cancer.

Cancer of the larynx is distinguished from *tubercular laryngitis* by the history, the absence of pulmonary disease and severe cough, the presence of an irregular tumor instead of the more or less uniform thickening, and the deep destructive ulceration, with the peculiar fetid breath. In tuberculosis when the epiglottis is involved, swelling is comparatively uniform over the whole valve, and when the arytenoids or ary-epiglottic folds are affected there is a peculiar pyriform appearance

commonly on both sides, not observed in cancer. The swollen tissues in tuberculosis, so long as ulceration has not taken place, are usually lighter in color and less dense than in the malignant tumor. The sarcomata have an irregular surface and the appearance of an abnormal growth, quite distinct from the more or less uniform swelling of tuberculosis. When ulceration takes place in tuberculosis, it is usually superficial, though sometimes deep and destructive; but by the time the latter occurs, the hectic and cough, the cachexia and pulmonary signs, will at once indicate the nature of the disease.

In the early stage or until ulceration occurs, it is often very difficult to distinguish malignant growths from *benign tumors*. During the course of cancer, before ulceration has occurred, the age (past middle life), the pain, the irregularly defined tumor of a dirty gray or bright red color, with almost constant glandular infiltration in pharyngo-laryngeal cancer, and the occasional occurrence in intra-laryngeal cancer of glandular enlargement farther down the trachea at the root of the neck, renders the diagnosis fairly certain.

Prognosis.—Cancer of the larynx sometimes terminates fatally within from three months to a year; but the average duration is about eighteen months. Epithelioma is sure to terminate fatally, though life in some instances may be considerably prolonged by operative measures. Sarcoma may probably be completely eradicated in some cases. Death is finally caused by inanition, asthenia, asphyxia, or hemorrhage.

Treatment.—All medicinal means have proved inefficient in checking the onward progress of the disease. There are certainly no specifics, and all drugs fail in the end; even those which are held in most esteem, such as arsenious acid, calcium sulphide, iodoform, carbolic acid, ergot, mercury, and turpentine. As a palliative remedy to relieve pain, opium in some form, and belladonna or cocaine are of importance. Morphine, tannic acid, and carbolic acid locally (Form. 139, 148) render the ulcer less painful and offensive. Continuous heat is especially valuable in relieving the severe earache which often attends this disease. Anti-syphilitic remedies should be thoroughly tried in all cases where there is any doubt as to the diagnosis, and sometimes they apparently check the progress of the disease for a short time. Surgical measures should be adopted in all suitable cases. These are: endo-laryngeal attempts at removal; endo-laryngeal cauterizations; tracheotomy; resection of the larynx; extirpation of the larynx.

It frequently happens that the true nature of the laryngeal growth cannot be determined at first, and under such circumstances its removal by endo-laryngeal methods should be attempted when there is any probability of success. In a doubtful case portions of the tumor should be subjected to microscopic examination and if cancer is demonstrated, all endo-laryngeal operations not calculated to effect complete eradication should be discontinued, except in extreme cases where re-

moval of portions of the growth will prevent suffocation. In cancer, partial operations upon the tumor usually accelerate its growth.

Lennox Browne ("Diseases of the Throat," second edition) recommends endo-laryngeal cauterizations in certain cases confined to the epiglottis and not susceptible of removal. However, he justly remarks that he fears the benefit of such measures is but temporary. Though I have never practised cauterization of laryngeal cancers, my experience with it in cancerous growths of the nasal passages leads to the belief that in this affection, as a rule, it would be productive of more harm than good.

Tracheotomy to prevent suffocation is frequently necessary, and may prolong life from three to twelve or even eighteen months. In case of myxo-sarcoma, I have known life thus prolonged for four or five years.

Resection, or partial extirpation of the larynx, in suitable cases, has been attended with very favorable results, where complete extirpation of the disease is possible by removal of the epiglottis or the lateral half of the larynx. This operation is indicated in small endo-laryngeal epitheliomata confined to one side, and in sarcomata not yet markedly infiltrating. It is useless when the larynx is invaded from the pharynx and whenever the adjoining structures and cervical glands are involved. Immediately fatal results have followed this operation in only a small percentage of cases, and usually life has been very considerably prolonged; in a few instances the disease seems to have been completely eradicated. The following description of the operation is taken from the report of a case by Lennox Browne (*op. cit.*):

> The patient being anæsthetized a high tracheotomy was done, and Hahn's tampon canula introduced for twenty minutes, which time was allowed for the compressed sponge about the canula to expand. A median incision over the thyroid was made from just above the tracheal opening to the hyoid bone. The tissues were carefully divided down to the thyroid and cricoid cartilage; the soft parts, with the perichondrium, were carefully lifted with a raspatory, the perichondrium being peeled away from the cartilage, while its relations to the soft parts remained undisturbed. The separation was carried back as far as the median line of the boundary between the larynx and pharynx, solely by the use of the one instrument. Part of the hyoid attachment of the thyro-hyoid muscle was divided, but the horizontal incision over the hyoid bone, as recommended by Hahn, was unnecessary. The thyroid cartilage was then split in the median line by cutting-forceps. The attachments to the pharynx were further separated by the raspatory, knife handle and finger nail, and the thyro-hyoid membrane was divided close to its thyroid attachment, the superior cornu of the thyroid cartilage cut off by sharp pliers, and the cricoid cartilage severed with the same instrument in the median line in front and behind. The divided half of the larynx was then separated from the first ring of the trachea and removed entire. There was but little hemorrhage, and only two small blood vessels required torsion, the comparative freedom from hemorrhage being due to the use of the raspatory in keeping close to the cartilage.

Laryngectomy, or extirpation of the larynx, has been recommended and practised in many instances, yet with but few successes. Since the

operation involves great danger, and the patient's subsequent condition is most wretched, it should not be advised, unless we are confident that the disease is wholly confined to the larynx, and then only after the patient has been fully apprised of the danger and probable results.

The operation is described by Mackenzie as follows:

A vertical incision should be made from the hyoid bone to the second ring of the trachea, and the front and sides of the larynx should be thoroughly freed and exposed by careful dissection, partly with the cutting blade of the scalpel, but as far as possible with its handle. Should there be any decided arterial hemorrhage, the necessary ligatures must be applied. The trachea should be drawn forward with a hook, and cut across, care being taken to avoid penetrating the œsophagus. A siphon tube of vulcanite is then to be inserted into the windpipe. In order that the siphon may fit accurately, it is well to have at hand several tubes of different sizes. The upper and posterior attachments of the larynx should next be cut through, but in dissecting out the cricoid cartilage the risk of button-holing the gullet must be avoided by keeping the knife close to the cartilage ("Diseases of the Throat").

Sometimes the whole larynx must be removed, but not infrequently the superior cornua of the thyroid cartilage may be left. Hemorrhage may be stopped by ligature or torsion, or by some styptic solution. When the surfaces have healed and the gap in the throat has partially contracted, Gussenbauer's artificial larynx may be used. Though from the description the operation seems very simple, the disease will often be found more extensive than anticipated, making the procedure most formidable. J. Solis Cohen has recommended a modified form of laryngectomy (Transactions of the American Laryngological Association, 1887), which appears to offer many advantages over the ordinary operation, when the disease is not extensive. As claimed, the wound is small, the operation may be done rapidly and with comparative safety to the patient, the attachments of many of the ligaments and muscles are preserved, important functional structures retained, and a firm natural support is left for an artificial larynx. His description of the operation is as follows:

1st. Make an incision from the hyoid bone to the lower border of the cricoid cartilage and exactly in the median line. 2d. Carefully separate the sterno-hyoid muscles. 3d. Hold the soft parts aside and insert from above one blade of a strong cutting forceps, with narrow blades, beneath one wing of the thyroid cartilage, one-fourth inch from the angle of junction with its fellow, and sever the cartilage vertically its entire length to the crico-thyroid membrane. 4th. Make a similar cut on the opposite side. 5th. Seize the freed angular portion of the thyroid cartilage comprising its entire respiratory contingent with a vulcellum forceps and draw it to either side, the soft parts being separated meanwhile, from the inner surfaces of the attached wings of the thyroid cartilages, with the handle of the scalpel. 6th. Make a transverse cut to sever the cricoid cartilage from the trachea. At this step in the living subject, a sterilized cotton plug should be inserted into the upper end of the trachea, preliminary tracheotomy

having been performed previously. (If the cricoid cartilage is to be retained, disarticulate the arytenoids and then sever the soft parts above the cricoid instead of below. This modifies the next step in the procedure accordingly.) 7th. Lift the cricoid cartilage forward, and carefully separate it with the edge of the knife from the inferior cornua of the thyroid laterally and superiorly, the nfrom the œsophagus posteriorly. 8th. Insert a finger into the pharynx from below and carry its tip over the epiglottis to draw that structure down. 9th. Divide the thyro-hyoid membrane and the fibrous tissues still holding. 10th. Lift out the exsected respiratory portion of the larynx. The arteries likely to require ligation will comprise small branches of the superior, middle, and inferior laryngeals.

The operation should be strictly aseptic, and where practicable should have been preceded several days by a preliminary tracheotomy. George R. Fowler has adopted this operation once for the removal of an epitheliomatous larynx, with most satisfactory results (*American Journal of Medical Sciences,* October, 1890). Gussenbauer's artificial larynx was placed in position on the forty-first day, and on the seventy-third day after the operation the patient was discharged, and was able to speak in a loud whisper without the aid of the artificial larynx. Several months later there was no evidence of recurrence, and the patient remained in good health.

EVERSION OF THE VENTRICLE OF MORGAGNI.

The eversion of the ventricle of Morgagni is a very rare occurrence. I am not aware that more than three cases are on record. One of these was diagnosticated before death by George M. Lefferts (*New York Medical Record,* June, 1876), but the others were not detected until the autopsy; therefore we are unable to give any distinctive signs. The condition is likely to be mistaken for a morbid growth. In the case reported by Lefferts thyrotomy was performed, and the everted sacculus cut off with scissors.

TRACHEAL TUMORS.

Laryngeal and tracheal diseases, as already noted, are so nearly related that it is most convenient to consider them in close connection. Tumors in the trachea are extremely uncommon. Those at its upper extremity may generally be seen by laryngoscopic examination, but it may be difficult to determine whether they are located below the cricoid cartilage or in the lower part of the larynx. Great care must always be observed in the diagnosis of disease in the trachea, otherwise we are apt to be misled by imperfect reflection of the light. Poor illumination may apparently reveal objects which do not exist. I have seen but three cases of tracheal tumor: one a large growth, as represented in the cut, and two others, papillary growths, upon the anterior wall of the

trachea about two inches below the glottis. Tumors in this situation may be either benign or malignant.

Etiology.—The causes are similar to those of corresponding tumors in the larynx.

Symptomatology.—These neoplasms when small cause no distinctive symptoms, but as they increase in size dyspnœa results and there is usually considerable cough and some expectoration. Upon inspection the growth usually presents a cauliflower or papillary appearance, sometimes congested, occasionally semi-transparent. It is usually sessile, but it may be pedunculated.

Diagnosis.—A diagnosis can only be made by laryngoscopic examination, and the exclusion of tracheal involution and syphilitic strictures.

Prognosis.—The duration varies greatly according to the nature of the tumor, but the affection is ultimately fatal in the majority of cases.

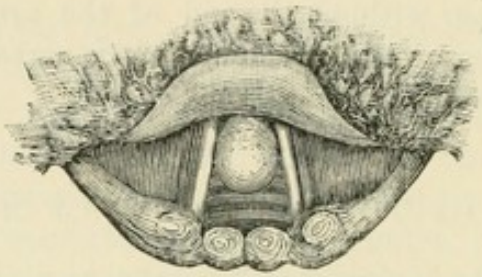

Fig. 177.—Tumor in Upper Part of Trachea. This tumor occurred in a patient about sixty years of age, but owing to the large size of his trachea it gave him very little inconvenience, and therefore he declined to have any attempt made for its removal. The symptoms in the case were hoarseness and moderate dyspnœa.

Sometimes the growth may be removed, but usually it is so deeply seated that it is reached with difficulty and the patient eventually dies of suffocation.

Treatment.—When practicable, the tumor should be removed through the mouth by means of forceps or the snare, or destroyed with chromic acid. In either case the parts should first be thoroughly anæsthetized by cocaine, and the operation performed with great care and precision. It is quite possible that some cases may be relieved by the introduction of an O'Dwyer tube, which by continuous pressure may cause absorption of the growth; but if the tumor cannot be reached by any of these methods, and respiration is seriously obstructed, tracheotomy should be performed, and if possible the growth removed by the cutting-forceps. Otherwise a long, flexible tracheal tube should be introduced and allowed to remain.

Malignant tumors in the trachea are necessarily fatal, and no form of treatment will be found of value, excepting palliative measures sometimes of a general, and sometimes of a local nature.

POST-TRACHEOTOMY VEGETATIONS.

After tracheotomy, especially where the tube has been worn for more than two or three weeks, not infrequently granulations spring up about the point of incision in the trachea, which more or less occlude its calibre, and, when the canula is removed, interfere with respiration. In some instances true papillary growths are developed.

ETIOLOGY.—These vegetations are apparently due to irritation caused by the tracheal canula, especially where one with a fenestra has been used.

SYMPTOMATOLOGY.—While the tracheal tube remains in place, no difficulty is experienced; but on its removal, respiration is impeded, or may be completely obstructed, by the abnormal growth.

DIAGNOSIS.—The symptoms already named will immediately suggest the nature of the affection, but an accurate diagnosis must rest upon the exclusion of stenosis by a careful inspection of the tracheal wound and of the larynx. It will be necessary in some instances to

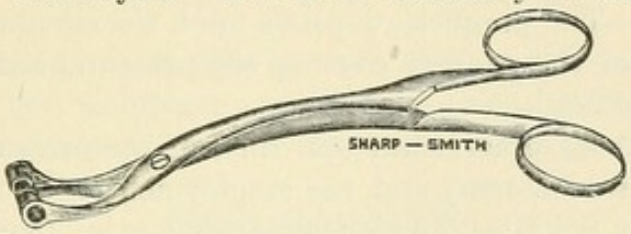

FIG. 178.—INGALS' PUNCH FORCEPS (½ size). They were devised to remove granulations in the trachea, but are also serviceable for certain cutting operations on the nose or throat.

pass a Schrötter dilator through the larynx to crowd the vegetation downward before it can be seen at the opening in the trachea.

PROGNOSIS.—The cases are usually very difficult to remedy, and in a few instances it has been impossible to remove the tracheal canula.

TREATMENT.—Under general anæsthesia, the granulations should be removed by forceps, and their bases cauterized by silver nitrate; or they may be destroyed by chromic acid or the galvano-cautery. It is sometimes very difficult to grasp these with ordinary forceps, and in such instances a pair of punch forceps (Fig. 178) which I have had made specially for these cases will be found very serviceable. Sometimes it will be necessary to crowd the growth down, with Schrötter's dilator or some similar instrument introduced through the larynx, before it can be reached at the tracheal wound. Two or three such cases have been cured by wearing for a short time an O'Dwyer tube; but it is not wise to allow the tracheal wound to heal until we are certain that the vegetations have been completely removed. In some instances the laryngo-tracheal tube shown in the article on stenosis of the larynx (Fig. 148) will be found necessary.

INVOLUTION OF THE TRACHEA.

Involution of the trachea consists of bulging inward of its walls resulting from external pressure. It is characterized by dyspnœa proportionate to the obstruction of the tube.

Etiology.—It may be due to pressure upon the trachea by an enlarged thyroid gland, or aneurismal tumor, or by substernal syphilitic growths, and rarely by disease of the cervical glands.

Symptomatology.—The chief symptom is dyspnœa, increased by exertion, and sometimes occurring in severe paroxysms dependent upon swelling of the mucous membrane or partial closure of the opening by tenacious mucus.

Diagnosis.—The affection is to be distinguished from asthma or any disease causing obstruction of the glottis. It can only be diagnosticated by exclusion after a careful laryngoscopic examination and consideration of the history, physical signs, and symptoms. For this inspection, a bright light must be carefully focused upon the parts to be examined. Unless one is thoroughly familiar with the appearance of the region, it is easy to make an error on account of the peculiar reflection of the light.

Prognosis.—The prognosis depends upon the amount of obstruction and the nature of the growth causing the pressure, but sooner or later most cases prove fatal.

Treatment.—If practicable, the cause of the pressure should be removed; if not, tracheotomy and the employment of König's long, flexible canula (Max Schüller, "Tracheotomie," u. s. w., *Deutsche Chirurgie*, 1880) will afford the most relief.

TRACHEOCELE.

Tracheocele consists of a hernial protrusion of the mucous membrane of the trachea between its cartilaginous rings. Several cases have been reported by Larry under the title of Aërial Goitre.

Anatomical and Pathological Characteristics.—The sac is generally lined with mucous membrane and contains some muco-purulent secretion. The walls of the sac vary according as it remains under the muscles or becomes subcutaneous.

Etiology.—The origin of the disease is usually obscure, though in most instances it apparently results from accidental straining. Mackenzie cites two congenital cases (Diseases of the Throat and Nose).

Symptomatology.—The voice may be weak and there is occasional dyspnœa. During ordinary respiration there may be but slight fulness in the front of the neck; but on forced expiration with the mouth and nose closed, or during cough, a tense, circumscribed swelling appears upon the front of the neck, the position corresponding nearly to that of the thyroid gland—sometimes median, sometimes upon one or the other side, occasionally bilateral. By pressure while the patient stops breathing or during inspiration, the tumor can usually be made to disappear almost entirely, although the thickened sac can ordinarily be felt under the skin.

DIAGNOSIS.—The diagnosis is made by causing the patient to expire forcibly with nose and mouth closed, or to cough, which will make the tumor distinct; then by pressure during inspiration it may be reduced. The varying size of the tumor, its increase on obstructed expiration, the impulse during cough conveyed on palpation, together with the other signs just mentioned, render the diagnosis certain.

PROGNOSIS.—When congenital, the affection will usually last a lifetime; but when due to accident, it may disappear spontaneously, or, if not, it can usually be cured by an appropriate appliance. It is not dangerous to life.

TREATMENT.—Some mechanical appliance to prevent undue distention of the sac is indicated and thus its enlargement may be retarded. Surgical interference has not proved advisable in the majority of cases.

SYPHILIS OF THE TRACHEA.

Various pathological changes are met with in the trachea similar to those found in the secondary and tertiary stages of syphilis affecting mucous membranes elsewhere, but they are comparatively rare.

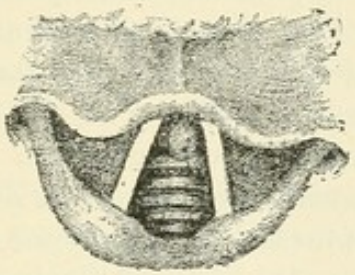

FIG. 179.—TRACHEAL PUSTULE, SPECIFIC.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—Simple congestion or superficial ulceration, projecting ridges, small ulcers, and occasional ulcers of a larger size are observed. In the tertiary stage, gummatous deposits in the submucous tissue seem usually to constitute the first change. These soften, leaving ulcers that on healing result in dense cicatricial tissue, accompanied by contraction and stenosis. Dilatation may occur above and below the stricture so formed. These changes usually extend over a large superficial area, and through the whole thickness of the tracheal wall; even the tissues surrounding it may be involved. Most frequently the lower portion of the trachea is the seat of the disease. The tube itself is sometimes shortened, according to Mackenzie, but stricture is the most common condition.

ETIOLOGY.—The localized phenomena mentioned may be the result either of congenital or acquired syphilis.

SYMPTOMATOLOGY.—Tickling sensations in the trachea, a disposition to cough, and occasional expectoration of mucus or muco-pus, with more or less alteration of the voice in consequence of congestion of the cords

or the collection of mucus upon them, and other symptoms of catarrhal tracheitis are the common symptoms, except where there is obstruction from growths or from stricture. Condylomata of considerable size or marked stenosis of the trachea cause dyspnœa proportionate to the obstruction of the tube; this is usually associated with cough, expectoration, and occasionally with paroxysms of suffocation due either to acute swelling of the parts or to collection of tenacious mucus at the seat of stricture. When the stricture is very close, so as constantly to interfere with respiration, marked constitutional symptoms may result. By inspection of the trachea, lesions in its upper part may usually be seen, but those farther down often escape observation, and can only be detected by careful physical exploration of the neck and chest.

Diagnosis.—The diagnosis must be based upon the results of a careful laryngoscopic examination, and the exclusion of diseases liable to cause compression of the trachea, as, for example, substernal tumors or aneurism.

Prognosis.—The probable duration of the affection can never be accurately estimated, for under appropriate treatment some of the lesions may disappear, and the patient may remain well for years. When decided narrowing of the trachea has taken place, the result is likely to be fatal within a few months. Death may occur from exhaustion from apnœa due to swelling, or suddenly from impaction in the stricture of tenacious mucus.

Treatment.—Constitutional remedies are of prime importance. Mercurials or moderate doses of potassium or sodium iodide should be tried thoroughly. Where these fail, large doses of potassium or sodium iodide are often necessary. An excellent method of administering them is to begin with a dose of gr. xx. three times daily, largely diluted with water or milk; increase the dose each day steadily by five to ten grains, until the maximum dose of from ʒ i. to ʒ ii. is reached; this may be continued two or three days, and then decreased to twenty grains. After two or three days, the dose should be again increased as before. Such large doses are not to be recommended except in extreme cases. Ten, fifteen, or twenty grains three or four times daily are sufficient for most patients, but occasionally a case which would improve promptly under large doses steadily progresses under the smaller quantity. Insufflation of iodol or iodoform into the trachea, daily or three times a week, will be found beneficial in the hyperæmic stage and when ulceration is present. If the stricture is high, O'Dwyer's laryngeal tube may be employed to dilate it; but if low in position, tracheotomy must be performed, and a canula which will reach below the obstruction must be inserted and worn. König's long flexible canula is especially adapted to this purpose.

CHAPTER XXVIII.

DISEASES OF THE LARYNX.—*Continued.*

FRACTURE OF THE LARYNX.

Fracture of the larynx is a comparatively rare accident. Up to the year 1868 only fifty-two cases had been recorded in medical literature. In most instances the thyroid cartilage is the seat of fracture, the cricoid being broken only by unusually extensive and dangerous injuries.

Anatomical and Pathological Characteristics.—It is probable that ossification of the laryngeal cartilages renders them more brittle and liable to fracture, and that, as suggested by Panas, premature senility, a result of chronic alcoholism, is sometimes a predisposing factor (*Annales des Maladies de l'Oreille*, March, 1878).

Etiology.—A direct cause is usually a blow, fall, or compression. As a result, extravasation of blood, œdema, or displaced fragments of the cartilage may so obstruct the air passages as seriously to impede respiration.

Symptomatology.—The usual symptoms are cough, dyspnœa and expectoration of mucus tinged with blood, tenderness or actual pain in the parts, and external swelling and deformity. Subcutaneous emphysema of the neck is apt to follow early, in some cases extending to the arms and trunk, and on manipulation crepitation may be easily felt.

Diagnosis.—The diagnosis may be made from the history of violence and the symptoms just indicated.

Prognosis.—The accident is always dangerous, and judging from the monograph by Henoque, fracture of the cricoid is nearly always fatal (*Gazette hebdomadaire*, 1868, No. 3,940); indeed, there are up to the present time but three or four cases of recovery known. If tracheotomy were promptly performed, probably the number of recoveries would be larger. Unfortunately, owing to the vital character of the structures involved in the injury, many patients die in spite of the operation; or, if recovery follows, they are subject for the rest of their days to troublesome or dangerous deformity of the parts.

Treatment.—Unless the symptoms are very slight, tracheotomy should be performed at once, and even if dyspnœa be absent the operation is advisable, since not infrequently by a slight movement the glottis becomes suddenly closed and suffocation results. If the cartilages are much crushed, it will be best to lay open the whole length of the larynx

and endeavor to replace and fix the fragments in proper position. Leeches and cold applications should be applied to the neck to prevent extensive inflammation. It is probable that intubation of the larynx by O'Dwyer's method would work well in some cases.

DISLOCATION OF THE LARYNX.

Attention has recently been called to luxation of the crico-thyroid articulation, by H. Braun, of Königsberg, according to whom it occurs unilaterally upon either side, and may take place daily or at intervals of weeks or months (*Berliner klinische Wochenschrift*, October, 1890). It may occur during deep inspiration, but more commonly during the act of yawning. Probably a loose capsule is the predisposing cause, and the sterno-thyroid and crico-thyroid muscles are the active agents. Intense pain and a feeling of anxiety are the chief symptoms, a slight prominence being produced at the inner border of the sterno-cleido-mastoid muscle on a level with the lower border of the thyroid cartilage. Reduction may be easily effected by digital pressure outward and backward, or by a few efforts at deglutition.

FOREIGN BODIES IN THE LARYNX.

Foreign bodies of great variety from time to time have been found in the larynx, generally entering from the mouth while the patient is coughing or laughing during mastication, but sometimes they enter from the œsophagus in consequence of sudden inspiration during the act of vomiting, and in rare instances, especially in military service, they penetrate from without. The objects most frequently found are pieces of bread, meat, bone, and other substances taken into the mouth during a meal. In children, peas, beans, coins, buttons, and similar substances which have been put into the mouth in play, or drawn in through blow-guns, are most likely to be found. Pins, fruit-seeds, and coins are sometimes found in adults. Soldiers upon the march, in drinking dirty water, have occasionally taken in leeches which have become lodged in the larynx. Artificial teeth, or natural teeth which have become loosened, have sometimes become lodged in the larynx during sleep; other substances which were in the mouth on going to bed are apt to be drawn in in the same way.

Symptomatology.—The symptoms vary greatly with the size, shape, and position of the object, and with the irritability of the larynx. A large body, or any object which has become impacted in the larynx in such a position as to cause clonic spasms of the glottis, is apt to cause immediate death; on the other hand, small bodies may remain indefinitely without very much annoyance.

I once saw a patient two years of age who had drawn into the larynx half a peanut kernel, which after remaining for two months was coughed out, having caused in the mean time no symptoms other than cough and hoarseness.

Usually, even small and smooth bodies give rise to much discomfort and troublesome cough, while sharp or irregular bodies excite severe paroxysms of cough and dyspnœa due to spasm of the glottis, and in many cases produce hemorrhage. Sometimes a body which causes little discomfort in the larynx at first, upon changing its position gives rise immediately to severe symptoms. Even where irritation is not sufficient to excite spasm of the glottis at once, the inflammation which supervenes within from twenty-four to thirty-six hours may cause extensive swelling, with narrowing of the glottis, which may be suddenly occluded by spasm of the laryngeal muscles. The fright which attends this accident often tends to increase the dyspnœa.

DIAGNOSIS.—The diagnosis will depend upon the history of the case, the symptoms already mentioned, and the results of laryngoscopic inspection when this is practicable; but children, on account of fright, sometimes will not give an accurate history, and adults may greatly exaggerate their symptoms. In the former, laryngoscopy can seldom be accomplished, and even in adults it is often difficult because of irritability caused by the foreign body, though this may generally be relieved by spraying the throat with a solution of cocaine.

PROGNOSIS.—In many cases death occurs immediately from closure of the glottis either by the body itself or by the spasm which it excites, and life is always in danger so long as the body is in the larynx. Frequently the immediate effects of the accident pass off, but the inflammation which the foreign substance excites causes closure of the glottis in from twenty-four to forty-eight hours by swelling or spasm. Sometimes the body suddenly changes its position with a similar result, and even after its removal there is still danger until acute inflammation has subsided.

TREATMENT.—A patient seen at the time of the accident should be immediately placed with the head at least forty-five degrees below the body, and should be slapped vigorously upon the back in the hope of causing expulsion of the foreign body; but if in this position respiration ceases, the head should be raised at once which possibly may so change the position of the object as to allow of respiration. If subsequently respiration should suddenly cease in consequence of change in the position, similar measures should be adopted. If by these methods respiration is not re-established, the patient should be placed upon the back, preferably with the head lower than the body, and artificial respiration should be kept up until medical assistance arrives, even if this is delayed for half an hour. In cases not immediately fatal, the physician may try inversion of the patient with vigorous slapping upon the back in the hope of causing expulsion of the foreign body. If this does not

succeed, unless suffocation is imminent, a laryngoscopic examination should be made where practicable and an effort made to remove the object with forceps. If all these methods fail, unless the body is very small and the symptoms slight, tracheotomy should be done as soon as possible, and another effort at removal made either through the tracheal opening or through the mouth, whichever is deemed best at the time.

In cases of angular bodies firmly impacted, it is occasionally, though rarely, necessary to lay open the whole length of the larynx for their removal. Sometimes a body which has been firmly fixed may be removed by the methods already suggested after the inflammation and swelling have been reduced by external applications. Bodies which have been impacted in one or both ventricles will not infrequently require crushing before they can be extracted. This has at times been accomplished through the natural passages. When tracheotomy has been done and the foreign body extracted, the tracheal tube should be allowed to remain four or five days until swelling has subsided; and it should not then be taken out until the physician, by corking the canula for several hours, has assured himself that laryngeal respiration is easy.

FOREIGN BODIES IN THE TRACHEA.

Foreign bodies enter the trachea quite as commonly as the larynx, for the reason that small substances, as a rule, immediately pass through the glottis. Isolated cases of this accident have been recorded from a very early period, but the first extensive treatise upon the subject was by Lewis, in 1759, though the subject was not treated exhaustively until the publication of the late S. D. Gross' work on Foreign Bodies, in 1854. Foreign bodies in the trachea are due to the same causes, and occur in the same way, as the similar affection of the larynx.

Symptomatology.—The symptoms will necessarily vary with the character of the body which has been introduced, as well as with the irritability of the tracheal mucous membrane. Patients have occasionally drawn foreign bodies of considerable size into the trachea without causing any symptoms which would suggest to them that such an accident had occurred. Large bodies or fluid drawn into the trachea may cause immediate death, or severe dyspnœa, which, growing gradually worse, induces pallor of the general surface with lividity of the lips and nails, cold sweating, and all of the symptoms of suffocation, which become more and more pronounced until death supervenes. Sometimes the symptoms are comparatively slight at the time of the accident, but a few hours later, owing to a change in the position of the body, to swelling of the mucous membrane, or to spasm of the glottis, sudden death may occur; or, the symptoms of suffocation soon subsiding, the patient may breathe easily again for a variable length of time until the

paroxysm is renewed, possibly with fatal effect. If the body is small and smooth, it may pass through the trachea and drop into the bronchial tubes, and unless soon removed it will ere long set up inflammation. Coins sometimes are lodged edgewise in the trachea and give rise to little or no discomfort, but they may suddenly become turned across the tube and cause suffocation. As a rule, bodies of moderate size soon set up irritation and inflammation resulting in cough by which the object may be thrown out or become lodged in the larynx with disastrous results; or the inflammation may finally extend to the lungs, causing pneumonic abscesses or, eventually, phthisis. Rarely, concretions form about small bodies, greatly increasing the difficulty which they cause. Kernels of corn, beans, and similar substances may be greatly enlarged by swelling, from absorption of moisture, and they sometimes germinate. In cases where severe dyspnœa immediately follows the accident, but suddenly passes off without expulsion of the body, we infer that it was first impacted in the larynx and subsequently drawn into the trachea. Frequently movable bodies in the trachea may be felt by the patient as they pass up and down during the acts of respiration or cough, and these movements may sometimes be felt by the finger over the trachea. Angular bodies cause more or less pain; smooth or small bodies may cause no sensations whatever. Bodies lodged in the trachea cause more or less diminution of the respiratory murmur, or a slight râle which may be heard over the entire chest. Usually the foreign substance drops into one of the bronchial tubes, about five out of eight gravitating to the right side; as a result, there is deficient movement and feebleness of the respiratory murmur over the corresponding side. Sometimes the body, or the mucus collecting about it, causes bronchial râles heard on one side only. These signs, when found, are very important from a diagnostic point of view, but are not universally present, even though the body be lodged in the bronchial tube, especially in the case of buttons or coins turned edgewise.

Vocal fremitus is also diminished over the obstructed lung, and there may be slight dulness on percussion, due to collapse of some of the air vesicles or to collection of mucus in the bronchial tubes. By laryngoscopic examination the foreign body can sometimes be detected in the trachea.

Diagnosis.—Usually there is a suggestive history, but it is not always possible to tell whether the body has been ejected or not. When the foreign substance can be seen or felt in the trachea, or when with a history of the accident the difference of the physical signs upon the two sides of the chest indicates obstruction of a bronchus, we may be positive of our diagnosis. There are frequently cases where it is impossible to diagnosticate the presence of small or smooth bodies which have been drawn into the trachea; in these we are obliged to wait for time to decide.

Prognosis.—Where the immediate danger has been survived, the greatest risk occurs between the second day and the end of the first month; during the succeeding month the mortality notably diminishes, but later it again increases. As already indicated, the prognosis is always serious so long as the foreign body remains in the air passages, the gravity depending upon the size and nature of the body, the amount of dyspnœa, and the changes set up in the lungs. When it is ejected or removed, recovery is usually rapid. Foreign substances have sometimes been coughed up weeks, months, or even years after the accident, the patient in the mean time having suffered more or less from the irritation which they produced.

For the encouragement of those in whom the body cannot be found, a case mentioned by Gross may be cited, in which a boy three years old drew a piece of bone into the trachea, which remained in the lung and was finally ejected during a fit of coughing six years later. A child was once brought to me who had drawn a button into the trachea. I did tracheotomy, but the button could not be obtained. The wound was kept open for several weeks and then allowed to heal, and about a month later the button was expelled during a fit of coughing.

Treatment.—The indications are to remove the body as soon as possible. This may sometimes be done by inverting the patient and slapping him upon the back, as recommended for foreign bodies in the larynx, or by Padley's method which consists in placing a strong bench with one end upon a couch, with the other upon the floor, and causing the patient to sit on the upper part with his knees fixed over the end, and while taking a deep breath to lay himself quickly back supinely upon the bench (London *Lancet*, Vol. II, 1878). The inspiration opens the glottis, and the supine position favors the expulsion of the foreign body. If it should happen to lodge in the larynx, the patient's hold upon the bench with his knees enables him quickly to regain the upright position, so that the body will again fall back into the trachea. Children may be held up by the feet, or the child's body may be allowed to hang from the nurse's lap, the back being slapped in the mean time. When attempting either of the above methods, the surgeon should be ready to perform tracheotomy at once, for sometimes the body becomes firmly impacted in the glottis and suffocation would immediately ensue unless the windpipe were opened. It is needless to say that the methods named are only likely to succeed where the body is small and smooth, as in the case of coins, buttons, peas, and beans, and but recently inhaled. The methods just recommended may sometimes be tried with advantage after tracheotomy has been done, providing the body cannot be found and removed by forceps. In most cases tracheotomy will be necessary, and the surgeon should advise it at once when he is sure that a foreign body is in the trachea, remembering that delay is always dangerous; yet he should not fail to inform the friends that some patients recover without opera-

tion. In children chloroform is usually preferred as an anæsthetic, but in adults local anæsthesia may be produced by cocaine. Tracheotomy having been done, the foreign body will frequently be coughed out immediately, but if not, it should be sought with instruments. For this purpose Trousseau's tracheal forceps are well adapted, but I have had better results with Carl Seiler's laryngeal tube forceps (Fig. 180), with which upon one occasion I was enabled to remove a swollen kernel of corn that was deep in the right bronchus, and on another, a small spicula of bone from deep in the left bronchus.

A peculiar accident occurred a few years ago to the son of one of our well known physicians. The boy was playing with a blow-gun, in which he had a shawl-pin used as a dart. By a forcible inspiration the shawl-pin was drawn, head foremost, into the trachea, from which it was removed after tracheotomy with great difficulty because of its length and its position with the point upward.

If the body cannot be found, it is recommended that the edges of the tracheal wound be stitched to the integument, or that ligatures be

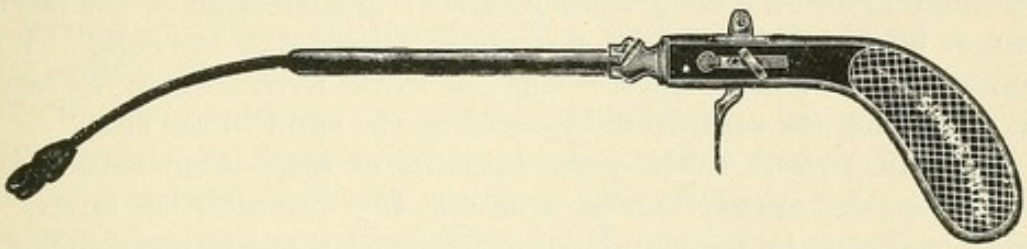

FIG. 180.—SEILER'S TUBE FORCEPS (2-5 size).

passed through the trachea and fastened with an elastic behind the neck, in order to keep the wound open. This device will answer very well for two or three days, but it is not applicable where the tracheal wound must be kept open for several weeks; in such instances I would recommend that a large tracheal canula be left in the wound, but that it be removed from time to time and efforts be made to remove the foreign body either by inversion or by forceps. After the foreign substance has been extracted, the trachea should be kept open for three or four days to allow all inflammation to subside, and to be sure that no other particles remain. The canula may then be removed and the wound allowed to heal. When tracheotomy has been done within a few hours after the accident has occurred, and where the body has been easily removed, the tracheal wound may be allowed to close at once. In the event that a foreign body becomes impacted in the bronchi so low that it cannot be removed by tracheotomy, the question of bronchotomy will arise; but notwithstanding the brilliant results of modern surgery, experience up to the present time is against it, as the danger to life far overbalances the chances of success, and there is a possibility that the object may eventually be expelled spontaneously.

SPASM OF THE GLOTTIS.

Synonyms.—Laryngismus stridulus; spasmus glottidis; suffocative laryngismus; spasmodic, cerebral or false croup.

Spasm of the glottis is a condition in which there is a temporary, complete or incomplete, spasmodic closure of the glottis or vestibule of the larynx, preventing free inspiration. It is characterized in the former case by cessation of the respiratory movements, and in the latter by stridulous respiration, almost identical with that of true croup or that of whooping-cough.

It is a purely nervous disease, and was formerly believed always to result from cerebral disorders. It is now known to be due also to direct or reflex peripheral irritation from a great variety of causes; for example, pressure on the recurrent laryngeal nerve, the presence of irritating substances in the alimentary canal, or irritation of the gums in dentition. Lubet-Barbon (*Revue mensuelle des maladies de l'enfance*, Paris, *Annual of the Universal Medical Sciences*, 1892) states that adenoid hypertrophy in the naso-pharynx is nearly always present. The attack is very likely to occur during acute catarrhal inflammation of the larynx, and may be excited by mental or physical irritation of the child. With nursing babes it is frequently brought on by the entrance into the larynx of a little milk and sometimes by dandling the child in the arms.

Symptomatology.—The great majority of cases occur between the ages of four and twenty-four months, and very few after the latter. It is most common in boys, and more frequent in poorly nourished children than in those well cared for. The attack usually comes on suddenly in the night, when the child awakens in fright from great dyspnœa or temporary suspension of breathing. After a few respirations it cries out, and soon falls asleep as though nothing had occurred. In severe cases the symptoms are more violent; the breathing suddenly becomes difficult, inspiration is prolonged and stridulous, and in a few moments the respiratory movements cease in consequence of complete closure of the glottis; the face, which was flushed, becomes pallid, and this is speedily followed by lividity; the eyes roll, the veins in the neck become turgid; and there are spasmodic contractions of the hands and feet. General convulsions sometimes ensue. In mild cases the attack often does not recur until the following night. The severer the paroxysms, the greater will be the rapidity with which they succeed each other. In some severe cases they follow each other in rapid succession, or there may be an almost endless spasm which does not relax until life is extinct. In the more common form of the affection the child may appear perfectly well the following day and there may be no return of the paroxysm, but usually it is repeated the next night or even within a few hours. As a rule, there is no fever, but profuse sweating, especially of the head, is a common symptom.

Diagnosis.—The disease is not likely to be mistaken for any other

except true croup, from which it may be diagnosticated by the absence of fever and the intermittence of symptoms between the paroxysms.

PROGNOSIS.—The attacks last but a few minutes, but they may recur after a few hours or the following night, or in severe cases may be speedily repeated. In the milder forms, recovery is common, but others are often fatal, and sometimes during the first paroxysm, which may last but one or two minutes. In cases depending upon disturbance of the digestive organs or slight irritating causes, the prognosis is favorable, providing the paroxysms do not last too long or follow each other quickly; whereas in those resulting from cerebral disease, or in those where the intervals between the paroxysms are short, the prognosis is grave. As a rule, the greater the interval between the paroxysms and the slighter the individual attacks, the better the chances of recovery.

TREATMENT.—During the paroxysm, flagellation, and the dashing of cold water in the face, are the most common remedies.

To terminate the spasm and prevent its recurrence, in the majority of cases nothing is better than ℥ xv. to xxx. of the compound syrup of squills, which should be repeated every fifteen minutes until vomiting occurs. Tickling the fauces with a feather or the finger is sometimes sufficient to excite vomiting, apomorphine in minute doses may be injected subcutaneously, or turpeth mineral may be given for the same purpose in doses of gr. ss. to ij. or even more. Teaspoonful doses of powdered alum act promptly and efficiently. To relieve the paroxysm a hot bath or a sitz bath at 95° F. may be employed, or chloroform may be carefully administered. An enema of tincture of assafœtida, ℥ xx. to xxx., ad ℥ i. of warm gruel or milk is sometimes a most useful remedy to prevent recurrence of the attack. Tincture of castor and musk are also valuable for the same purpose. The cause of the spasm must be sought and removed. It is most commonly found in some derangement of the digestive organs associated with slight catarrhal laryngitis. The spasm may be caused by an enlarged thymus gland, especially in young children. It has been known to arise from irritation of the prepuce. It is not infrequently caused by hysteria or cerebral or cerebro-spinal disease. Subsequent to the paroxysm, vegetable tonics, cod-liver oil, and the bromides are generally beneficial.

SPASM OF THE LARYNX IN ADULTS.

Spasm of the larynx is much less frequent in adults than false croup in children, and is most commonly observed in nervous women.

ETIOLOGY.—Spasm of the larynx is sometimes a pure neurosis, but may also be produced by irritation of the larynx by foreign bodies, or by œdema, or by laryngeal tumors. Sometimes it results from irritation of the recurrent laryngeal nerve, and in some cases a paroxysm comes on during sleep, without apparent cause.

SYMPTOMATOLOGY.—The paroxysm comes on suddenly. There is stridulous inspiration, speedily increasing dyspnœa, and in severe cases temporary arrest of respiration, which may be followed by expectoration of a considerable quantity of viscid mucus. On inspection at the time, the mucous membrane of the larynx is usually found slightly congested, but it may appear perfectly healthy, and the vocal cords are seen to separate for an instant, and then to suddenly draw together.

DIAGNOSIS.—The diagnosis rests upon suddenness of onset, the peculiar obstruction of respiration, and the exclusion of foreign bodies or tumors by inspection.

PROGNOSIS.—The attacks are of short duration, and are seldom, if ever, dangerous excepting when resulting from foreign bodies.

TREATMENT.—Inhalations of steam impregnated with soothing remedies as conium, belladonna, or stramonium, or inhalations of the smoke of burning stramonium, are useful in relieving the tendency to spasm when the attacks are recurring with frequency. The inhalation of a few whiffs of chloroform will give speedy relief in most cases. After the attack, general and nerve tonics are indicated. For this purpose a pill containing one grain each of zinc valerianate, quinine valerianate, and iron, is an excellent combination. Potassium, sodium, or ammonium bromide may also be administered to relieve the irritability of the larynx. To prevent the spasm of the glottis which occurs in some patients during and after applications to the larynx, the patient should hold his breath during the application and for a second or two afterward and then recommence breathing slowly, through the nose.

IRRITATIVE COUGH.

A dry, hacking, and sometimes paroxysmal cough is apparently of nervous origin and not infrequently accompanied by hyperæmia of the mucous membrane. The reflex form may be associated with disorders of the digestive organs or of the uterus; it is sometimes violent during dentition, and it may also result from varix or enlarged glands at the base of the tongue, enlargement of the tonsil, or elongation of the uvual. The cough is most frequent in the morning, and is usually referred to the region of the trachea.

TREATMENT.—Any of the associated marked conditions should receive appropriate treatment, and sedatives or antispasmodics in the form of troches and sprays should be given to check the tendency to cough.

NERVOUS COUGH.

By nervous cough we refer to a peculiar cough most frequently manifest in hysterical women, but sometimes occurring in men. It is usually characterized by a resemblance to the cry of one or other of the

lower animals, most frequently the yelping of a dog (Cohen: "Diseases of the Throat and Nose"). It is apparently purely of a neurotic origin, the most careful examination failing to detect any definite lesion. No very satisfactory method of treatment can be suggested, though electricity has sometimes proven effectual. Tonics, especially strychnine, arsenious acid, quinine, and iron, are useful in some cases.

ANÆSTHESIA OF THE LARYNX.

Anæsthesia of the larynx consists in more or less complete loss of sensibility of the mucous membrane, usually characterized by dysphagia, which results from the tendency of food, especially liquid, to drop into the trachea during deglutition. The anæsthesia may be unilateral or bilateral; it may be almost complete over the entire surface, even extending into the trachea, or it may be confined to that portion of the larynx about the vocal cords.

ETIOLOGY.—The affection seems to result form hysteria in a few cases, but is generally caused by diphtheria or bulbar paralysis. In some instances it has been due to tumors, hemorrhages, or deposits at the base of the brain (McBride: *Edinburgh Medical Journal*, July, 1885; and Schech: Diseases of the Nose and Throat); it may follow erysipelatous and variolous affections of the throat, and has been observed in cholera.

SYMPTOMATOLOGY.—The most important symptom is spasmodic cough on deglutition, caused by liquid or food entering the trachea and coming in contact with the sensitive membrane beyond the affected area. The epiglottis is generally found erect, and imperfectly closes the larynx during deglutition.

DIAGNOSIS.—A history of diphtheria or bulbar paralysis, with occurrence of spasmodic cough on deglutition, and the absence of obstructions in the pharynx or œsophagus as determined by inspection and by the passage of an œsophageal bougie, are strongly suggestive of this condition. Palpation with the laryngeal probe without causing appreciable sensations renders the diagnosis certain.

PROGNOSIS.—Except in cases of bulbar paralysis or other cerebral disease, recovery may generally be expected in from four to six weeks. In extreme cases, unless measures are taken to prevent the passage of food into the trachea, it is apt to cause fatal pneumonia. When associated with bulbar paralysis, death results within a few months.

TREATMENT.—The employment, three to six times a week, of either the galvanic or induced electric current, or of static electricity is to be recommended. If either of the first two are used, the electrodes should be applied six or eight times at each sitting. Probably the most important treatment consists of the internal use of strychnine in large and increasing doses, until its physiological effects are appreciated, as recom-

mended for paralysis of the vocal cords. When there is marked difficulty in swallowing, the patient should be fed through the œsophageal tube, to prevent the entrance of food into the windpipe. Owing to the anæsthesia, special care is necessary to avoid the passage of the instrument into the larynx.

HYPERÆSTHESIA, PARÆSTHESIA, AND NEURALGIA OF THE LARYNX.

Increased or perverted sensibility of the larynx, or intermittent pain in the organ, without structural lesions, is most frequently observed in preachers and others accustomed to excessive use of the voice.

Simple neuralgia is very rare, and most cases which formerly would have been classed under this head are now recognized as rheumatic.

Anatomical and Pathological Characteristics.—There may or may not be congestion of the mucous membrane; in some cases even pallor is present, especially when the condition is associated with phthisis. If hyperæsthesia results from excessive use of tobacco or alcohol, there is usually congestion. Frequently there is disease of the glandular structure of the pharynx and larynx, or base of the tongue.

Etiology.—Hyperæsthesia usually results from excessive use of tobacco or alcohol, repeated subacute inflammations of the larynx, gastric disturbances, tuberculosis, pharyngitis, or over use of the voice.

Paræsthesia is commonly caused by debility, nervous prostration, hysteria, or hypochondriasis, and often follows the lodgement for a short time of some foreign substance in the throat. It is sometimes one of the early symptoms of phthisis pulmonalis. It is also a symptom of enlarged glands or varicose veins at the base of the tongue. Neuralgia is attributed to similar causes, but is more often due to anæmia, gout, and rheumatism.

Symptomatology.—In hyperæsthesia, the larynx is so abnormally sensitive that cough is excited by slight irritation, such as the inhalation of cold air, smoke, or dust, or the contact of certain substances in deglutition. It is frequently attended by various sensations, as of burning, prickling, dryness, rawness, and constriction; and occasionally by spasmodic action of the muscles of the larynx and pharynx, the former occurring with respiration, the latter with deglutition. The most frequent sensation in paræsthesia is that of a silver, or other large or small foreign body in the throat. Numbness and coldness are sometimes experienced. The so called *globus hystericus* is a familiar form of the affection. In neuralgia, the pain is often intermittent and unilateral and may be accompanied by areas or points of tenderness. Cough may be troublesome.

Diagnosis.—The diagnosis must be based upon the symptoms, and the absence of physical signs.

PROGNOSIS.—The affections may be expected to continue for a long time, but, in the majority of cases, recovery eventually takes place.

TREATMENT.—When hyperæmia is present, it should be reduced by stimulant or astringent applications. Diseased glands and enlarged veins of the pharynx or base of the tongue are best destroyed with the galvano-cautery. Applications to the larynx once or twice daily, by spray, of solutions of morphine or cocaine, though the latter should not be used freely, or a combination of morphine, carbolic acid, and tannic acid with glycerin and water (Form. 93, 139), are often serviceable.

Where cough is troublesome, troches of lactucarium or of cannabis indica and codeia (Form. 29, 33) or other sedative preparations are especially useful. Sometimes the inhalation of a few whiffs of chloroform, which may be carried in a small bottle in the pocket, gives great relief. Internally, the iodides and colchicum are indicated when a rheumatic or gouty diathesis exists, and camphor monobromide, chloral or aconite, or the bromides, gr. x. to xv., three or four times daily are especially useful for prolonged sedative effects. The various bitter and ferruginous tonics are frequently indicated, and good hygienic conditions are particularly important.

CHOREA LARYNGIS.

Chorea laryngis is an extremely rare affection of the larynx, characterized by regular monotonous recurrence, during waking hours, of a peculiar sound, often resembling a short bark or yelp, associated with, and dependent upon violent inco-ordinate involuntary movements of the vocal bands. The affection is accurately described by Ziemssen, but the first published uncomplicated case appears to be that reported by George M. Lefferts (Transactions of the American Laryngological Association, 1879). Cases have also been reported to the same association by F. I. Knight, of Boston, and E. Holden, of Newark, N. J.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—The disease is a neurosis the seat of which appears to be either in the brain or spinal cord, but the exact lesion has not been determined. The larynx is liable to be slightly hyperæmic, but presents no other physical changes.

ETIOLOGY.—In most of the cases reported there has been no assignable cause for the affection, which has come on in persons otherwise perfectly well. It is sometimes attributed to hysteria, with which it is liable to be confounded.

SYMPTOMATOLOGY.—The affection may be a part of general chorea, but the term *chorea laryngis* should be limited to those cases in which only the laryngeal muscles are involved. There are no constitutional symptoms, the patient complains merely of the frequent recurrence of some peculiar sound at regular intervals during the waking hours. In some this is attended by spasmodic cough, which may be excited by the

act of swallowing. Upon laryngoscopic examination, there is often found some congestion of the larynx, and in the intervals between the production of the peculiar sound the motions of the cords may be perfectly natural, or they may quiver and tremble, and the adductors and abductors may be in constant motion; but, even then, on phonation the cords as a rule act naturally; sometimes, however, during this act their movements are irregular, speech being correspondingly altered. At the time the peculiar sound is produced, the cords are generally driven suddenly and sharply together, sometimes two or three times in succession; this concussion probably accounts for the hyperæmia, and it is immediately followed by a long inspiration after which the parts may remain natural until time for the next sound to occur. These peculiar sounds always cease during sleep.

DIAGNOSIS.—The affection is most likely to be confounded with hysteria, from which it is distinguished by the following points:

CHOREA LARYNGIS.	HYSTERIA.
May accompany general chorea.	Absence of general chorea.
Occurs regularly during waking hours.	Occurs at irregular periods.
Violent, prolonged, inco-ordinate, and involuntary movements.	Short spasms; may be voluntary and regular; never long-continued.
In typical cases, confined to larynx.	Seldom or never confined to larynx.

PROGNOSIS.—Under appropriate treatment most cases recover within a few months.

TREATMENT.—Local applications of electricity have been tried in many cases, but are of doubtful value. Applications of astringent sprays, such as used in chronic laryngitis, are beneficial in reducing the hyperæmia, but the main reliance must be placed upon general tonic treatment, especially the administration of arsenious acid. F. I. Knight mentions one case in which the symptoms immediately subsided upon the exhibition of full doses of quinine (Transactions of the American Laryngological Association, 1883). Bromides have been found of some benefit in diminishing the frequency of the paroxysms. Strychnine has rendered little, if any, service.

SPASM OF THE VOCAL CORDS.

Closely akin to chorea laryngis is a spasmodic affection of the vocal cords most frequently observed in nervous overworked professional men past middle life. In this affection there is commonly congestion of the larynx, but no other visible change from the normal condition. The etiology and pathology are not understood, but the condition appears to be due to functional alteration of the nerve centres. In cases I have observed the individuals have been able at times to talk in a natural voice,

but suddenly, without control, the voice rises to a high pitch, in consequence of spasm of the adductor and tensor muscles, and is apparently produced with much effort and straining of the laryngeal muscles. In this latter respect the symptoms differ materially from those attending paralysis of the crico-thyroid muscles, in which there is a somewhat similar change in the voice.

The affection is likely to continue for years and is very refractory.

TREATMENT.—The treatment from which most relief is to be expected consists in good hygienic surroundings, including rest and pleasant travel, and systematic vocal culture.

At first the larynx should be given, as nearly as possible, perfect rest for several weeks, the patient talking but little and that only in a whisper. After a time he should be given very short but increasing exercises in reading at regular hours two or three times a day, as a sort of vocal gymnastics. The reading should be in a low unvarying tone and must be stopped as soon as the voice breaks.

At first these lessons may not exceed one or two minutes in duration, but they may be gradually prolonged a minute or more each day as the voice becomes more stable, and after the patient is able to read for half an hour in monotone, gradual changes may be tried in the pitch and intensity of the voice. During this time the congestion of the larynx may be removed by the use of weak astringent sprays, as for example zinc sulphate gr. i.–iij. ad ℥ i. At the same time the nervous system should be fortified by sedatives and tonics conjoined with abundant rest, regular exercise, and the removal of all sources of direct or reflex irritation.

FALSETTO VOICE.

Falsetto voice is a rare symptom, usually observed in young men who, although fully developed in every other respect, retain an abnormally high pitched, puerile voice.

It is due to the misuse or non-use of muscles controlling the lower register, which should be brought into activity about the age of puberty. The condition is usually outgrown within a few months, or at most years, after puberty; but it sometimes persists to middle or even advanced life. It is purely functional and may generally be speedily cured if proper methods are adopted; but if left to themselves such patients often suffer for many years from the mortification entailed by the childish or feminine voice.

TREATMENT.—The work of the physician consists in demonstrating to the patient that he has a chest voice and inducing him to use it.

The method recommended by J. C. Mulhall, of St. Louis (Transactions of The American Laryngological Association, 1888) I have found perfectly satisfactory in several cases. At first a thorough laryngoscopic examination is made, and then the patient is assured that the vocal apparatus is normal and that if he will carefully follow directions he will with a little training be completely cured.

He is then caused to depress the chin firmly on the neck, and asked to imitate the physician, who sounds a deep chest tone. The imitation is usually prompt and easy. The patient is thus shown that he has another voice, and by repeated exercises taught to use it. The depression of the chin is merely to direct the patient's will more easily to the proper muscles, and may soon be omitted in subsequent exercises.

A cure may often be effected within a few minutes, though in other cases more prolonged training is necessary. A few lessons have always proven sufficient in my experience.

Sometimes the cure is delayed by the patient's fear to use his newly found voice, or by embarrassment in using it before his acquaintances.

LARYNGEAL VERTIGO.

Laryngeal vertigo is a rare affection characterized by momentary loss of consciousness, occurring during a fit of coughing. It is usually observed in men past middle life. The attack generally comes on suddenly, with short spasmodic cough, which is immediately followed by giddiness. In most instances, during the attack, the patient becomes unconscious for a few seconds: but this speedily passes off, so that mental confusion remains only a short time, excepting in a small percentage of cases. Usually there are no other evidences of nervous disease. During the attack, the face may be unnaturally pale, though in most cases it is congested, and in a few there are twitchings of some of the muscles; but in none has there been frothing of the mouth or biting of the tongue, as in epilepsy. In the majority of cases, the larynx has been found hyperæmic.

Most cases have been relieved, at least temporarily, by the application of astringents to the pharynx and larynx, counter irritation over the larynx, and the administration of bromides internally.

A very full exposition of the whole subject has been given by F. I. Knight, in the Transactions of the American Laryngological Association for 1886.

CHAPTER XXIX.

DISEASES OF THE LARYNX.—*Continued.*

PARALYSIS OF THE THYRO-EPIGLOTTIC AND ARY-EPIGLOTTIC MUSCLES (DEPRESSORS OF THE EPIGLOTTIS).

A PARALYSIS in the domain of the superior laryngeal nerve is characterized by dysphagia especially of fluid, and when complete and bilateral, by anæsthesia of the laryngeal mucous membrane. It is usually attended by paresis of the crico-thyroid muscles.

ETIOLOGY.—The paralysis named is most commonly caused by diphtheria, occasionally by progressive bulbar paralysis, and rarely by enlarged glands and inflammation of the areolar tissue beneath the angle of the jaw.

SYMPTOMATOLOGY.—In consequence of this paralysis, the epiglottis remains erect during deglutition, and fluids or particles of food find their way into the larynx and trachea, where they cause sudden paroxysms of cough and dyspnœa attended by pain if anæsthesia is not also present. Particles of food aspirated into the smaller bronchi are apt to excite pneumonia. Though there are no characteristic signs of this affection, upon inspection the epiglottis may be seen to maintain an erect position during the imperfect acts of deglutition made with the mouth open and the tongue protruded, and upon palpation anæsthesia is often detected.

DIAGNOSIS.—When the affection follows diphtheria, it is usually associated with paralysis of the palate or pharynx and anæsthesia of the larynx. The symptoms and signs, taken in connection with dysphagia, paroxysms of cough and dyspnœa, and the appearance of the epiglottis, together with the absence of other signs, establish the diagnosis.

PROGNOSIS.—In complete paralysis of both superior laryngeal nerves there is considerable danger, but unilateral paralysis is not very serious. In the former, death may result from pneumonia caused by aspiration of foreign substances into the lung; but if this accident is escaped, the cases due to diphtheria usually recover.

TREATMENT.—The greatest care should be taken to prevent the entrance of foreign substances into the trachea. Feeding should be accomplished either by the œsophageal tube, or by having the patient during deglutition assume a position with the head lower than the

body. Ferruginous and bitter tonics are indicated, but strychnine in large doses as advised for anæsthesia of the larynx is of most value.

PARALYSIS OF THE CRICO-THYROID MUSCLES (EXTERNAL TENSORS OF THE VOCAL CORD).

As a separate affection, paralysis of the crico-thyroid muscles is rare. It is either unilateral or bilateral in its occurrence, and is characterized by dysphonia or aphonia. It commonly results from diphtheria, exposure of the neck to cold draughts, or from overstraining the voice in singing or shouting, especially during inflammation of the larynx. It has been caused by injury to a small branch of the superior laryngeal nerve in ligating the common carotid artery, and it is sometimes associated with paralysis of the adductors and internal tensors of the cords. Complete paralysis of these muscles is very rare.

SYMPTOMATOLOGY.—The voice may be very hoarse and inadequate to the production of the high notes, or altogether suppressed. Sometimes during ordinary conversation there is a peculiar sliding rise in the pitch of the voice, which the patient is unable to prevent. Prolonged use of the voice may be fatiguing or even painful. There are also symptoms of coexistent anæsthesia of the larynx. Sometimes by placing the finger over the crico-thyroid muscle at the lower lateral portion of the larynx during phonation, its non-contraction may be readily recognized. In some instances there is congestion, in others a pearly, translucent appearance of the vocal cords, which also have visible longitudinal relaxation.

In well marked cases the glottis presents a peculiar wavy outline (Fig. 181), with a slight depression of the central portion of the cords in inspiration and a corresponding elevation in expiration and vocalization; the vocal process can seldom be seen. When the affection is unilateral, the corresponding cord remains on a higher level than its fellow.

DIAGNOSIS.—In moderate cases the diagnosis must rest largely upon the symptoms; where the paralysis is decided, the subjective symptoms and the appearance of the glottis, together with lack of tension of the crico-thyroid muscle, leave no doubt.

PROGNOSIS.—Most cases recover after a short time, from rest alone, but the restoration of the voice may be aided by appropriate treatment.

TREATMENT.—In slight cases, wet compresses or mild counter irritation is all that is necessary. In those more marked, daily applications over the muscles, of the faradic or galvanic currents will be found beneficial. Strychnine and other tonics are also indicated in some cases. When anæsthesia of the larynx coexists, food should be introduced through an œsophageal tube to prevent its passage into the trachea.

PARALYSIS OF THE THYRO-ARYTENOID MUSCLES (INTERNAL TENSORS OF THE VOCAL CORDS).

Paralysis of the thyro-arytenoid muscles is a common affection, which may be either unilateral or bilateral. It is often associated with paralysis of the crico-thyroids and the adductor muscles of the cords. It is characterized by harshness and high pitch of the voice, with fatigue and sometimes pain in its use, and is most frequent among singers.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—The cords are often congested, sometimes swollen, and the edges are not accurately approximated but leave an elliptical chink between them in phonation, which accounts for the hoarseness or aphonia.

ETIOLOGY.—The affection usually results from over-use of the voice when the larynx is inflamed, or at the period of adolescence when the voice is changing, but it may be caused by a simple cold, fatigue, or strain of the muscles, and occasionally by diphtheria or hysteria.

SYMPTOMATOLOGY.—There may be fatigue or even pain upon use of the voice, with dysphonia, or, in case other muscles are involved, aphonia.

FIG. 181.—BILATERAL PARALYSIS OF THE CRICO-THYROID MUSCLES (MACKENZIE).

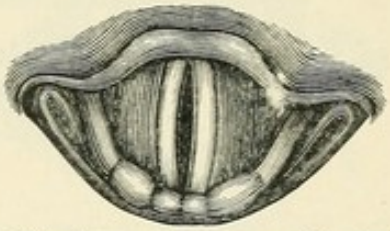

FIG. 182.—ACUTE LARYNGITIS. Paralysis of the thyro-arytenoid muscles.

Upon inspection during phonation, an elliptical chink about a line in width is usually observed between the vocal cords (Fig. 182), which, together with other portions of the larynx, are liable to be congested. When the arytenoid muscle is also paralyzed, the laryngeal picture is peculiar, an elliptical chink appearing in front of the vocal processes, and a more or less triangular opening behind them (Fig. 183).

DIAGNOSIS.—The diagnosis is based upon the history, symptoms, and laryngoscopic appearance.

PROGNOSIS.—When associated with simple laryngitis, provided the paralysis is not complete, recovery usually takes place within a short time, but some cases extend over several months, and occasionally the paralysis is permanent.

TREATMENT.—In over-fatigue and in cases resulting from acute inflammation, rest for the voice, with soothing inhalations or feeble astringent sprays, are most beneficial. In some instances, especially where fatigue is the cause, prolonged rest for many months is necessary. When the affection has already extended over several weeks, astringent or stimulating sprays to the larynx should be used; but if contraction of

the muscles is not readily induced in this way, the galvanic or faradic current should be employed for a few moments daily. Bitter and ferruginous tonics may be useful, but of all remedies strychnine in large doses is most beneficial.

BILATERAL PARALYSIS OF THE LATERAL CRICO-ARYTENOID MUSCLES (ADDUCTORS OF THE VOCAL CORDS).

Synonyms.—Functional aphonia, hysterical or nervous aphonia.

In bilateral paralysis of the lateral crico-arytenoid muscles, the vocal cords act imperfectly and are not approximated accurately during attempted phonation. It is characterized by loss of voice, and is most commonly observed in young women. It is often associated with paralysis of the arytenoid muscle, and sometimes the posterior crico-arytenoid muscles of both sides.

ETIOLOGY.—The affection is caused by hysteria, anæmia, general debility, phthisis, and sometimes by simple catarrhal inflammation in

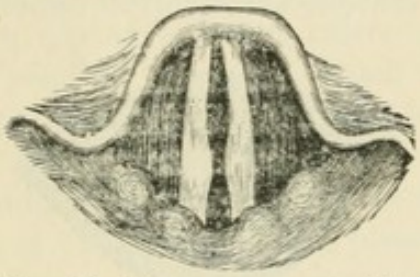

FIG. 183.—PARALYSIS OF THE THYRO-ARYTENOID MUSCLES AND PARTIAL PARALYSIS OF THE ARYTENOID.

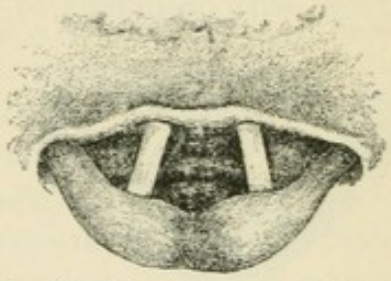

FIG. 184.—PARALYSIS OF THE LATERAL CRICO-ARYTENOID MUSCLES. Attempted phonation.

which the congestion disappears, but the paralysis remains. It is probably due in some instances to lead or arsenical poisoning.

SYMPTOMATOLOGY.—Functional aphonia often comes on suddenly without apparent cause, but sometimes is excited by shock or fright. Occasionally a patient who has retired in perfect voice finds herself unable to speak in the morning. In other cases resulting from an acute cold, hoarseness comes on, gradually growing worse for twenty-four or thirty-six hours, until the voice is lost. Occasionally exposure to a draught of air marks the beginning of the disease. Not very rarely the affection is intermittent, the voice failing and returning every few days for a time. In some of these instances it is possibly of malarial origin. One peculiar feature of many cases is that while voluntary movements of the cords may be lost, the reflex often remain, so that, although the patient cannot speak, she may cough, sneeze, or laugh aloud. Sometimes such patients talk aloud in their sleep, but are unable to do so when awake. When the paralysis is complete, no sound is caused by laughing or coughing.

The larynx is often paler than natural, but in catarrhal cases it is

congested. Upon attempts at phonation, the vocal cords remain in the respiratory position (Fig. 184) or move but imperfectly toward the median line; sometimes one is more completely paralyzed than the other. Usually on attempted phonation the cords are approximated to within about one-eighth of an inch of each other, and in not a few cases the edges may touch for a moment, and a short sound of *a* may be emitted at the time, though the patient is otherwise unable to talk. In complete paralysis, the glottis remains widely open without movement of the vocal cords during attempted phonation, and where the abductors are also involved the cords maintain the cadaveric position midway between phonation and inspiration. J. Solis Cohen remarks that sometimes this form of paralysis is associated with loss of voluntary control over the diaphragm, and then not only is the loud voice lost, but the patient is also unable to whisper (Diseases of the Throat, second edition).

DIAGNOSIS.—The affection may be confounded with cases in which the loss of voice is due to feeble respiratory action, or those in which

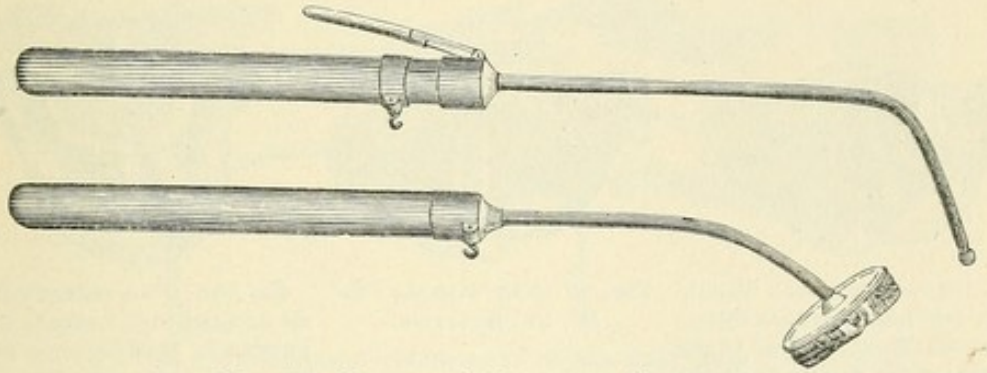

FIG. 185.—MACKENZIE'S LARYNGEAL ELECTRODES.

approximation of the cords is impeded by swelling of the inter-arytenoid folds, or by morbid growths, cicatricial tissue, or disease of the crico-arytenoid articulation. The history and symptoms, together with the laryngoscopic appearance just described, leave no room for doubt as to the diagnosis.

TREATMENT.—In hysterical cases the voice may frequently be restored by very indifferent measures, such, for example, as simply introducing a mirror, or throwing a mild astringent spray into the larynx; but in many cases prolonged use of the faradic current to the affected muscles, applying one electrode within the larynx and the other without, will be necessary to effect a cure. In most instances I have found astringent or slightly stimulating applications to the larynx every second day, combined with the administration of tonics, most effective; and of all tonics for this purpose, nothing can compare with strychnine in full doses. It is well to begin with about gr. $\frac{1}{30}$ three times daily, steadily increasing the dose until constitutional effects are produced, which may not happen until the patient is taking as much as gr. $\frac{1}{10}$ or

even gr. $\frac{1}{8}$ at a dose. When the physiological symptoms occur, the dose should be somewhat decreased, and then continued in an amount just short of producing spasmodic contraction of the muscles, until recovery is complete; or the quantity may again be increased, in the manner before mentioned.

UNILATERAL PARALYSIS OF THE LATERAL CRICO-ARYTENOID MUSCLE (LATERAL ADDUCTOR OF THE VOCAL CORD).

In unilateral paralysis of the lateral crico-arytenoid muscle one cord remains abducted during attempted phonation, thus rendering the voice hoarse or shrill. There is no lesion of the larynx itself, but the recurrent laryngeal nerve is generally involved.

ETIOLOGY.—The affection is caused in most cases by pressure upon the recurrent laryngeal nerve, as by an aneurism of the aorta, cancer of the œsophagus, malignant tumor of the neck, or enlargement of the

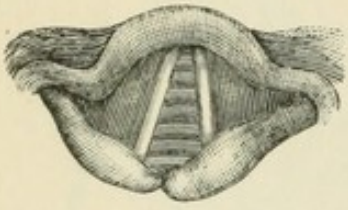

FIG. 186.—UNILATERAL PARALYSIS OF THE LEFT LATERAL CRICO-ARYTENOID MUSCLE. Due to the pressure of an aneurism on the left recurrent laryngeal nerve.

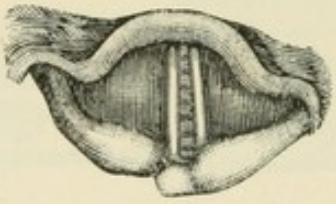

FIG. 187.—THE SAME AS FIG. 186, IN PHONATION.

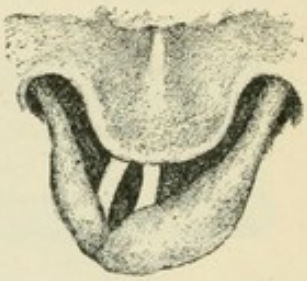

FIG. 188.—UNILATERAL PARALYSIS OF THE RIGHT LATERAL CRICO-ARYTENOID MUSCLE, WITH SWELLING OF LEFT ARY-EPIGLOTTIC FOLD. Phonation—left cord moving far beyond the median line

deep cervical glands. It is sometimes caused by chronic lead or arsenical poisoning, by exposure to cold, or muscular strain, and not infrequently by hysteria.

SYMPTOMATOLOGY.—There are usually no constitutional manifestations but the symptoms and signs of a tumor pressing upon the recurrent nerve may frequently be detected. There is slight or considerable impairment of the voice with loss of volume, and, when paralysis is complete, aphonia. The sounds produced by coughing, sneezing, or laughing are always altered more or less, and these acts are sometimes unaccompanied by sound. In phonation, the affected cord remains at the side of the larynx (Fig. 187), and the supra-arytenoid cartilages cross each other, the one from the sound side passing in front. The mucous membrane covering the affected cord is often found congested. When caused by pressure of a tumor, dysphagia is frequently present.

DIAGNOSIS.—The diagnosis is readily made by laryngoscopic examination.

TREATMENT.—The cause of the difficulty must, if possible, be found and removed. Local treatment is of little or no value. In a few instances, evidently functional, which had existed for a number of months, I have brought about a cure by the administration of large doses of strychnine when many other remedial measures had failed.

PARALYSIS OF THE ARYTENOID MUSCLE (CENTRAL ADDUCTOR OF THE CORDS).

In paralysis of the arytenoid muscle, owing to the non-approximation of the inner surfaces of the arytenoid cartilages in phonation, there is gaping of the posterior or inter-cartilaginous portion of the rima glottidis, with consequent impairment of the voice. Congestion of the larynx is usually present, for this form of paralysis most frequently results from acute or subacute laryngitis.

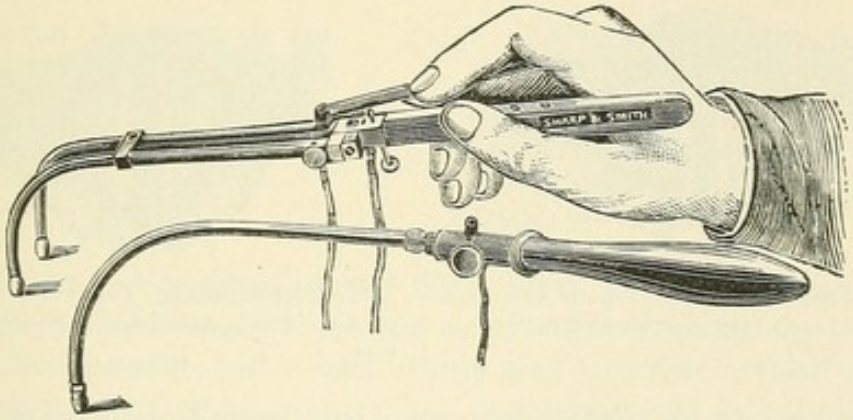

FIG. 189.—ZIEMSSEN'S DOUBLE AND SINGLE LARYNGEAL ELECTRODES.

SYMPTOMATOLOGY.—Hoarseness and fatigue in talking are prominent symptoms. Inspection reveals a triangular opening at the posterior part of the glottis during phonation.

DIAGNOSIS.—The diagnosis is readily made by inspection.

TREATMENT.—Stimulant inhalations and astringent applications appropriate for the laryngeal inflammation which coexists are indicated. In this, as in other forms of paralysis of the laryngeal muscles, if of long standing, faradization of the affected muscles and the administration of strychnine should be tried.

BILATERAL PARALYSIS OF THE POSTERIOR CRICO-ARYTENOID MUSCLES (ABDUCTORS OF THE VOCAL CORDS).

Bilateral paralysis of the posterior crico-arytenoid muscles is a dangerous affection of the larynx in which the vocal cords are not drawn aside during inspiration, but remain near the median line, closing the glottis and causing stridulous respiration and great dyspnœa, without alteration of the voice.

Anatomical and Pathological Characteristics.—The affection is generally due to disease of the central nervous system, but may be produced by morbid processes which involve both pneumogastric or both recurrent laryngeal nerves. The recurrent nerves and their branches, and the muscles themselves, have been found atrophied. In a few cases the muscles have been found atrophied, though the brain and nerves have appeared healthy.

Etiology.—The condition, as before stated, is usually caused by disease of the central nervous system, and is evidently sometimes caused by syphilis, the lesion of which may be central or along the course of the nerve, or in the muscle itself. It is frequently due to pressure upon the pneumogastric or recurrent nerves by goitre, enlarged bronchial glands, or aneurism. Cancer of the thyroid gland or of the œsophagus may have a similar effect. Occasionally the paralysis seems to result from simple catarrhal inflammation, or from hysteria.

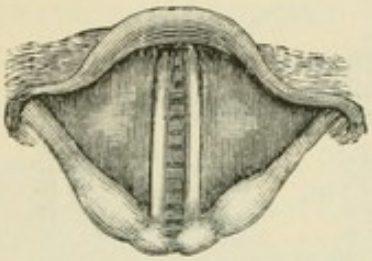

Fig. 190.—Bilateral Paralysis of the Posterior Crico-Arytenoid Muscles—Inspiration.

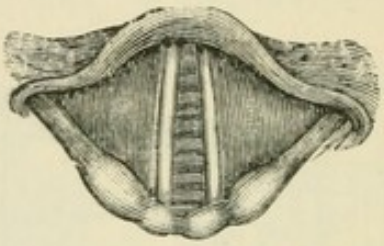

Fig. 191.—Bilateral Paralysis of the Posterior Crico-Arytenoid Muscles—Expiration.

Symptomatology.—The symptoms will depend upon the nature and extent of the lesion. Since the filaments of the recurrent nerve supply antagonistic muscles, those distributed to either the adductors or the abductors may be most involved, but experience shows that the latter are usually implicated first. Where the function of the nerve is completely destroyed, the muscles of both sides are paralyzed and the cords remain in the cadaveric position, offering no impediment to respiration, though the voice is lost. When the abductor filaments alone are affected, the voice remains, but inspiration is greatly obstructed, and extreme dyspnœa supervenes upon the slightest exertion. A feeling of suffocation may occur not only on exertion, but occasionally from spasm of the adductors, especially during sleep. Expiration is quiet and unobstructed. When the abductor muscles alone are paralyzed the voice is not lost, but it is usually weak; if the adductors are also implicated to a certain extent, there is constantly a waste of air in phonation and the patient in talking becomes quickly exhausted on account of the great labor thrown on the expiratory muscles; cough and expectoration are also difficult. Loss of strength, emaciation and febrile excitement, are frequently though not always present. On inspection of the larynx, the vocal cords are seen very near the median line; during

respiration the rima glottidis will measure from one to two lines in width (Figs. 190, 191).

On inspiration, the lips of the glottis are sucked downward and inward below their normal plane, and with expiration, are forced upward, the glottis being somewhat dilated, so that the air escapes freely. The vocal cords and mucous membrane of the larynx may be of a normal color or slightly congested.

DIAGNOSIS.—In adults the true nature of the disease is at once suggested by prominent inspiratory stridor; the characteristic appearance of the glottis on inspiration leaves no doubt as to the diagnosis, except as between this condition and *adhesion of the inner surfaces of the arytenoid cartilages*, which sometimes so closely resembles it that in the absence of previous history a differential diagnosis may be impossible. This affection may be distinguished from *spasm of the adductors* as follows:

BILATERAL PARALYSIS OF THE ABDUCTORS.	SPASM OF THE ADDUCTORS.
Inspiratory dyspnœa constant; may be increased during sleep.	Inspiratory dyspnœa temporary; diminished or absent during sleep.
Vocal cords immovable.	Vocal cords more or less constantly varying in tension.

PROGNOSIS.—The duration and final result necessarily depend upon the nature of the lesion; where the paralysis is decided, the prognosis is always unfavorable, and a fatal result may occur at almost any time unless tracheotomy or intubation has been done. It is only in a few cases, of catarrhal, syphilitic, or hysterical origin, that good results can be expected from medicinal treatment.

TREATMENT.—The great danger from suffocation renders it necessary to adopt some preventive measure. For this purpose, an O'Dwyer intubation tube may be introduced and worn while the influence of internal remedies is being tried; but if this does not succeed, tracheotomy had best be performed. Except when these patients can be closely watched it is not safe to let them go, even for a single day, without one or the other of these operations. Faradization should be tried, and such remedies used as are most likely to remove the cause, such as astringent and stimulating sprays in the catarrhal conditions, strychnine and other tonics in the hysterical form or where there appears to be functional interruption in the central nervous system, and the iodides in the syphilitic variety or when the pressure results from enlarged glands or goitre.

UNILATERAL PARALYSIS OF THE POSTERIOR CRICO-ARYTENOID MUSCLE (ABDUCTOR OF THE VOCAL CORD).

In unilateral paralysis of the posterior crico-arytenoid muscle, one vocal cord remains in the median line during inspiration, with consequent dyspnœa and stridulous respiration. It is due to lesions similar to those which cause bilateral paralysis, but it most frequently results from peripheral causes, as, for instance, catarrhal inflammation, or the implication of one pneumogastric or recurrent laryngeal nerve by malignant disease, aneurism, or other morbid growths.

SYMPTOMATOLOGY.—The symptoms are obstructed inspiration, stridor and dyspnœa, and slight alteration of the voice. There are also present more or less irritative fever and the symptoms of the primary disease. On inspection the affected cord is seen to remain stationary at

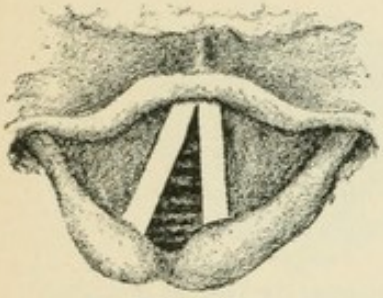

FIG. 192.—UNILATERAL PARALYSIS OF THE LEFT POSTERIOR CRICO-ARYTENOID—INSPIRATION.

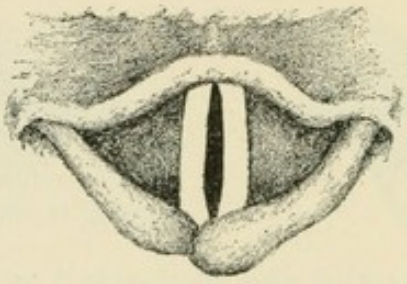

FIG. 193.—UNILATERAL PARALYSIS OF THE LEFT POSTERIOR CRICO-ARYTENOID—PHONATION.

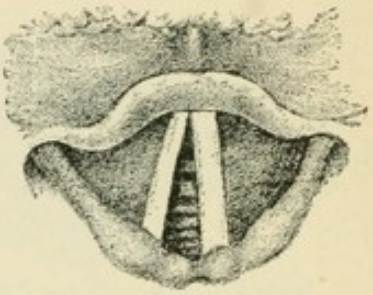

FIG. 194.—ANCHYLOSIS OF RIGHT VOCAL CORD—SPECIFIC—PHONATION.

or near the median line, while the movements of the other are normal or slightly exaggerated.

DIAGNOSIS.—The symptoms and laryngoscopic appearance leave no question as to the diagnosis.

PROGNOSIS.—The affection is much less dangerous than bilateral paralysis, but it is usually best to give a guarded prognosis, since it is impossible to tell how soon the disease which has implicated one nerve may involve the other. When due to simple catarrhal inflammation, hysteria, or syphilis, recovery is the rule.

TREATMENT.—If possible, the cause should be removed. Faradism or galvanism and constitutional treatment similar to that recommended in paralysis of both muscles should be employed.

ANCHYLOSIS OF THE ARYTENOID CARTILAGES.

Anchylosis of the arytenoid cartilages is a rare affection, the diagnosis of which may be attended with great difficulty, since it closely simulates paralysis of the abductors or adductors of the vocal cords. It should be suspected whenever we find immobility of one or both cords, with irregularity of the cartilages; and should always be looked for when

a patient convalescing from typhoid fever complains of the symptoms of laryngeal disease.

TREATMENT.—If the condition interferes with respiration, attempts should be made at dilatation by Schrötter's sound or O'Dwyer's intubation tubes, and tracheotomy must be done if necessary.

ATROPHY OF THE VOCAL CORDS.

Atrophy of the vocal cords is extremely rare, and so far has not been proven by post-mortem evidence. The cords merely have a shrunken appearance, or they may be so narrow that although nothing intervenes to prevent inspection they cannot be brought into view.

Diseases of the Nose.

CHAPTER XXX.

DISEASES OF THE NASAL CAVITIES.

INFLUENZA.

Synonyms.—Epidemic catarrh, epidemic catarrhal fever, grippe.

Influenza is a specific epidemic fever, characterized by catarrhal inflammation of the mucous membrane of the air passages or digestive tracts, and by marked and sometimes profound disturbances of the nervous system. It occurs in epidemics, which spread rapidly over an entire continent and attack the greater portion of the population irrespective of age, condition, or sex, except that infants enjoy nearly complete immunity from the disease, although young children are frequently attacked.

Anatomical and Pathological Characteristics.—No definite lesions can be described as peculiar to this disease, for in most fatal cases death results from some complication. There are usually signs of inflammation in the mucous membrane of the air passages and digestive tract, and not infrequently in the serous membranes covering the brain or lining the thoracic or abdominal cavities. Usually upon opening the chest, the lungs are found to contain here and there depressed spots of lobular consolidation. The mucous membrane of the larynx, trachea, and bronchial tubes is congested, swollen, and more or less covered with frothy or muco-purulent secretion. The bronchial glands may be enlarged and softened. Firm, whitish clots are often found in the right side of the heart. In many instances the gastro-intestinal mucous membrane is distinctly congested and swollen in patches.

Etiology.—The disease is evidently caused by some powerful morbific agent in the atmosphere, but whether an irritating gas or a specific micro-organism has not been determined. Generally speaking, the disease cannot be communicated from one to another, and, though some observations seem to indicate its contagious nature, this is still an open question.

Symptomatology.—The affection is sometimes preceded for twenty-four or forty-eight hours by general malaise, but usually it comes on suddenly with chilly sensations or distinct rigors alternating with flashes of heat and attended by severe headache, pain in the back and limbs, constriction of the chest, and muscular weakness. This is usually followed by the ordinary symptoms of acute coryza, with sore throat, fre-

quent hacking cough, and in many cases dyspnœa, even without any affection of the lungs themselves. There are paroxysms of sneezing and sensations of stuffiness in the head, the eyes are suffused, and not infrequently the inflammation extends to the Eustachian tubes and middle ear.

Severe frontal headache is one of the most common symptoms, and often there is great soreness of the muscles, attended in many cases by sharp neuralgic pains; extreme prostration and great despondency, wholly disproportionate to the severity of the attack, are often observed, and actual delirium or mental vagaries are present in many cases. Dizziness is frequently experienced on rising suddenly. Most epidemics of the grippe have been characterized by great restlessness and insomnia, but in some the opposite condition has been quite pronounced. As the disease becomes established, the face is often congested, and occasionally jaundice, associated with hepatic tenderness, occurs.

The fever rises rapidly to 101° or 102° F., or sometimes even to 104° or 105° F.; it is of a remittent character, usually attended by profuse sweating. Charles Warrington Earle (*Archives of Pediatrics*, March, 1892) states that in some children with influenza a high temperature persists for a long time during convalescence. In others he has observed a subnormal temperature, which in one instance, in the axilla, ranged from 93° to 98° F., for six days, although convalescence progressed favorably. The pulse commonly ranges from 90 to 100, though sometimes it runs much higher. In the milder forms of the disease, the catarrhal inflammation does not extend below the larynx; but in those of a slightly severer grade, which I have witnessed during the recent epidemics, a severe inflammation of the trachea often occurs, and not infrequently the inflammation extends beyond, giving rise to bronchitis or catarrhal pneumonia. These changes are attended by more or less dyspnœa and cough, and are usually preceded by hoarseness. The cough occurs in paroxysms, usually worse at night or in the early morning, and is at first attended by a frothy or clear expectoration, which later becomes mucopurulent and often quite offensive. The discharge from the nares is at first thin and watery as in an ordinary cold; later it becomes mucopurulent, and epistaxis is not uncommon. The tongue is usually coated, and the appetite lost; frequently there is tenderness, or colicky pains occur which may be attended by nausea, vomiting and diarrhœa. In many instances there is acute congestion of the kidneys; the urine is often scanty and not infrequently it is suppressed for a few hours.

Inspection of the nares usually reveals hyperæmia and swelling of the mucous membrane; and the mucous membrane of the fauces is similarly affected. Upon examination of the chest, the signs of bronchitis are generally present, even in comparatively mild cases, and all too frequently the evidences of pneumonia or pleurisy will be obtained.

DIAGNOSIS.—Influenza is not apt to be mistaken for any disease ex-

cept acute non-specific rhinitis or inflammation of the larynx, trachea, or bronchi, from which it does not materially differ except in its epidemic nature and the severity of the symptoms. Isolated cases of the latter frequently precede an epidemic of influenza four or five weeks, presenting much the same symptoms and possibly due to the same cause; but it must not be forgotten that severe catarrhal inflammations of the upper air passages are common, independent of the peculiar conditions which cause influenza. Usually the history of an epidemic, the severe headache, mental depression, muscular pains, and sudden onset of the attack render the diagnosis easy. The symptoms and signs of complicating disorders will not differ essentially from the usual manifestations of these affections, except so far as they may be modified by the fever and nervous prostration attending the epidemic disease.

PROGNOSIS.—The catarrhal symptoms usually begin to subside in three or four days, and in mild cases the patient will not be confined to the house more than forty-eight to seventy-two hours; indeed, many persons continue their avocations in spite of the disease. When the disease is more severe, convalescence may not be established for a week or ten days, and in some the affection may be even more prolonged. This is especially the case when the affection is complicated by tracheitis, bronchitis, or pneumonia, but in uncomplicated cases convalescence is usually fully established within ten or twelve days, even in the more severe forms of the affection. When occurring in the very young or the aged, or in persons greatly debilitated from any cause, or in persons suffering from chronic pulmonary, cardiac, or renal disease, influenza must be regarded as a grave affection; and when its various complicating disorders are considered, it will be found that a considerable number of cases, probably three or four per cent, prove fatal. When it attacks pregnant women, abortion is liable to follow. The experience of the epidemics through which we have passed during the last two years shows that functional disease of the heart, protracted fevers of a typhoid character, pleurisy, and pulmonary tuberculosis are common sequels of influenza. Rheumatoid or neuralgic pains not infrequently continue many weeks after the subsidence of the acute symptoms.

TREATMENT.—No positive directions can be given for the prevention of the disease; but as it has been observed that those who are exposed to the outer air suffer most from the affection, it is wise, during epidemics, for children and those enfeebled by age or disease to remain as much as possible indoors, hoping thereby to escape. As the main symptoms indicate great nervous depression, it is well during an epidemic to fortify the system against an attack by tonic doses of quinine and nux vomica. Large doses of quinine are said sometimes to abort the attack, and the same has been claimed for opiates, or opiates in combination with quinine, or ipecacuanha. During the progress of the disease, rest in bed and gentle laxatives, refrigerant drinks, moderate doses of

quinine, and small doses of opium or other anodynes to relieve the cough are recommended. To relieve the pain in the inception of the disease no remedy has seemed to me more valuable than phenacetin; later, large doses of potassium bromide, which is peculiarly efficient in allaying irritability and quieting cough, together with extract of nux vomica, extract of hyoscyamus, quinine, and camphor, have proven most beneficial. The irritability and inflammation of the mucous membrane may sometimes be greatly relieved by the inhalation of steam, or steam impregnated with various soothing vapors, as of opium, belladonna, or hyoscyamus. When rheumatic symptoms are present, colchicum and the salicylates, together with alkalies, have been found most useful. Complicating diseases should be treated upon general principles, and in protracted cases the nutrition should be carefully attended to. If convalescence is delayed, a change of climate will frequently be of great advantage.

RHINITIS.

SIMPLE ACUTE RHINITIS.

Synonyms.—Acute coryza, acute nasal catarrh, acute cold in the head, acute rhinorrhœa.

Simple acute rhinitis is an inflammation of the nasal mucous membrane, sometimes of one passage, but usually of both, often extending into the maxillary or frontal sinuses, the lachrymal ducts, and Eustachian tubes. It is characterized by paroxysms of sneezing, hypersecretion, and more or less obstruction of the nares. In infants it causes marked difficulty of breathing, particularly during sleep or nursing, and is occasionally attended by attacks very closely resembling laryngismus stridulus. The disease occurs in all climates and seasons and among patients of all ages and all classes of society, but it is somewhat more frequent among children, some of whom apparently have a congenital predisposition to it. It is said to be more frequent among persons of nervous temperament and in those subject to rheumatism, yet it is usually independent of diathesis.

Anatomical and Pathological Characteristics.—The mucous membrane becomes swollen, red, and at first dry, but is soon bathed in a profuse secretion of serum, which a little later becomes sero-purulent and is loaded with an excess of salines, which are very irritating to the nostrils and upper lip. In exceptional cases an excess of fibrin collects in irregular masses, as a membranous layer, which is most often found in the coryza of new-born infants or in that accompanying the exanthemata.

Etiology.—The most common cause is exposure to cold when the body is overheated, but not infrequently it results from exposure to undue heat, or the inhalation of dust or irritating fumes or vapors.

Fraenkel believes that infantile coryza is generally due to direct infection from the vaginal secretions at the time of birth. Among the occasional causes may be mentioned exposure to the rays of the sun, impetigo or eczema, measles, scarlet fever, typhoid fever, tertiary syphilis, iodism, facial erysipelas, or extension of inflammation from the conjunctivæ, pharynx, or larynx; and it is said to be caused in some instances by the cure of chronic discharges, such as those of otitis and ophthalmia, or bleeding hemorrhoids.

SYMPTOMATOLOGY.—The affection often comes on with a feeling of general malaise, which may last for two or three days, but more frequently there is aching of the back or limbs for only a few hours. Often constitutional symptoms are not present, and the onset is marked merely by an attack of sneezing, with more or less stopping up of the nose and hypersecretion of a thin, irritating serum, which, after one or two days, becomes thicker and bland. The nostrils and upper lip become red and irritated from the secretion and frequent use of the handkerchief. The nasal passages are so stopped that the patient is obliged to breathe through the mouth, with great discomfort, particularly while he is eating and during sleep.

The general symptoms vary from slight disturbance to severe pain and headache, with sleeplessness, mental and physical debility, fever, and derangement of the digestive organs. There is sometimes a slight chill at first, but the earlier symptoms usually consist of sensations of dryness or irritation in the nose and a disposition to sneeze. Within a few hours there is stopping up of the nares, with obtunding of the senses of smell and taste, more or less pain, and frequently extension of inflammation along the lachrymal ducts, causing redness and sensitiveness of the conjunctivæ. If the inflammation extends along the Eustachian tubes, there is a sense of fulness, possibly with pain in the ears, and often abnormal auditory sensations and partial deafness. The inflammation may travel down the pharynx, causing sore throat, or it may involve the antrum, frontal sinus, or ethmoidal or sphenoidal cells, causing correspondingly severe pain in the cheek or forehead, or deeper seated.

Occasionally the disease is intermittent, lasting for two or three days, and then subsiding, to be renewed after an equal length of time. Any or all of the symptoms excepting the secretion may be absent. The inflammation frequently attacks one side, not involving the other for two or three days or until its course is completed in the first. Exceptionally the cervical lymphatic glands become enlarged and sore. The body temperature may rise two or three degrees and the pulse be correspondingly accelerated. Obstruction of the anterior nares gives the voice a nasal tone; but if the swelling is mainly in the posterior part of the nares, the general character of the voice is normal, while the articulation is defective, the letter *m* being sounded like *b*, and *n* like *d*.

The secretion, which at first was thin, serous, and irritating, after a time becomes thicker, whitish, yellowish, or greenish, according to the intensity of the inflammatory process, and the cold is said to have broken. The secretion may amount to several ounces in twenty-four hours. The frequent use of a handkerchief after a time becomes painful, but, as the secretion becomes thicker, irritation gradually subsides. There is often an unpleasant catarrhal odor to the breath; and when the nose is completely obstructed, the tongue becomes dry and brown from the continued mouth-breathing. The appetite is not infrequently impaired.

Upon inspection, the mucous membrane is found to be swollen and congested, and sometimes, though not commonly, here and there are small, dark-brown stains, indicating extravasation of blood beneath the mucous membrane; or slight abrasions of the surface may be noticed. Early in the attack the thin secretion may be seen moistening the entire mucous membrane or flooding the floor of the nasal cavity; later, fine, cobweb-like shreds of mucus are often seen stretching from side to side across the nasal chamber, and more or less of the thicker secretion, mucous or muco-purulent in character, will be found collected in the nasal cavities, especially at the lower and back parts.

Diagnosis.—Acute rhinitis is not likely to be confounded with any affections excepting hay fever, inflammation of the antrum or frontal sinuses, or the commencement of measles. In any case the history, the character of the discharges, and the appearance of the parts will soon settle the diagnosis.

Prognosis.—Attacks of acute rhinitis sometimes last but a few hours, but usually they continue for from three days to a week, and sometimes two or three times as long. The stage of dryness generally continues two or three hours, that of free, thin secretion from twenty-four to forty hours. The thick secretion commonly continues two or three days, when it gradually grows thinner until the end of the attack. The affection usually terminates by resolution; in children at the breast, and in the very aged and infirm, it has occasionally proved fatal. Frequently repeated attacks are liable to eventuate in a chronic catarrhal condition of the nasal mucous membrane. The inflammation may leave obstruction of the lachrymal ducts or the Eustachian tubes, or chronic inflammation of some of the adjacent sinuses, and it sometimes seems to be the starting point for nasal polypi. Where these growths already exist, they are often found to enlarge greatly during acute attacks of coryza.

Treatment.—Prophylactic treatment includes daily sponging of the chest with cold water or salt and water, bathing the feet every morning in cold water, care respecting sufficient warmth of the clothing; and avoidance of sudden exposure, damp clothing, wet feet, and in a word all things which have been found to excite the inflammation. In the beginning an attack may frequently be aborted by moderately large

doses of opium, quinine, alcoholic stimulants, or the ammonium salts. Morphine gr. $\frac{1}{8}$ to $\frac{1}{4}$ or its equivalent, atropine gr. $\frac{1}{120}$ with morphine gr. $\frac{1}{8}$, pulv. ipecac. comp. gr. x., quinine gr. vi. to x., or a hot sling taken at bed time will frequently abort the disease. It may also be checked in a similar way by one or two doses of ammonium carbonate, gr. x. to xx.; ammonium chloride, gr. xx. to xxx.; liquor ammoniæ acetatis, ℥ i.; tincture of belladonna, ♏ x. to xx.; tincture of euphrasia officinalis, ♏ x. to xx.; ammoniated tincture of guaiacum, ʒ i.; or an emetic dose of antimony. These are best administered at bedtime, and their action may be favored by a hot foot bath containing a handful of mustard. Sometimes the disease is speedily aborted by frequent inhalations of chloroform, or the vapor of ammonium carbonate, camphor, iodine, or carbolic acid. But, as a rule, the most satisfactory abortive treatment consists in the administration of a comparatively large dose of quinine, and the application to the nose, either by spray or powder, of a small quantity of cocaine. Where opiates are well borne, one or two small doses of atropine and morphine act well.

If the cold has existed for twenty-four hours, it can seldom be aborted, and must then be simply carried through to a speedy termination, with as little discomfort as possible to the patient. Total abstinence from liquids, as recommended by C. J. D. Williams, is said to be efficient in curing attacks of acute rhinitis (Cyclopædia of Pract. Med., London, 1833), the coryza beginning to dry up in about twelve hours after liquids have been suspended, and ceasing completely in from twenty-four to thirty-six hours. Williams, however, allowed a tablespoonful of milk or tea twice a day, and a wine glass of water at bed time. If the disease was not aborted, Morell Mackenzie recommended five drops of the tincture of opium every six or eight hours. Ten drops of the spirits of camphor on sugar may be effectively taken in the same way. Five grain doses of potassium nitrate, twenty minim doses of the spirit of nitrous ether, or two drachm doses of solution of ammonium acetate repeated from time to time are often useful in cutting short the disease.

Turkish baths are sometimes very efficient, though extreme care is necessary to avoid taking subsequent cold. Jaborandi and other diaphoretics have a similar effect, and diuretics and cathartics may expedite the cure; however, these should only be given when the patient can be kept indoors. Inspiration through the nose of warm aqueous vapors or sprays of mild solutions, gr. ij. ad ℥ i., of ammonium chloride or carbonate, or sodium bicarbonate, or potassium carbonate, or boric acid, gr. viij. ad ℥ i., are sometimes very grateful to the patient, and seem to aid much in prompting resolution.

As a rule, the most satisfactory course of treatment will be found in the administration at first either of the morphine and atropine or of a comparatively large dose of quinine or of nux vomica and the application to the nares of a one or two per cent solution of cocaine in water, or better

still in oil, or the insufflation of a powder of four per cent of cocaine in sugar of milk and starch. In the latter case it is well to use also a spray of liquid albolene or benzoinol three or four times daily.

Occasionally persons are met in whom oily sprays of any kind aggravate the disease. In these the solution of boric acid is apt to be most soothing.

If the disease is not aborted at once, the cocaine may be continued in small quantities three or four times a day. The spray of liquid albolene should be continued during the attack, and the patient may be given with advantage, four or five times daily, small doses of cannabis indica and hyoscyamus, with medium doses of camphor and quinine, or quinine and phenacetin, or quinine and camphor mono-bromide. If opiates are given, care should be taken to keep the bowels open; and in any event it may sometimes be desirable to give gentle laxatives.

ACUTE RHINITIS IN INFANTS requires especial care to keep the nasal passages open. This may be done best by the application of sprays of liquid albolene, or, in cases where there is extensive secretion, by syringing the nose with a warm alkaline solution. The washing must be performed very carefully, and it must not be forgotten that often even very mild solutions are irritating to the nares and give the child pain. Whenever it is deemed necessary to syringe the nares in a child, it should be placed upon the face, and the warm solution introduced slowly, so that it may run out again from the opposite nostril, and not be drawn into the larynx. Excepting opium, most of the remedies recommended in the treatment of the disease in adults may be used in smaller quantities for children, but usually it is best to rely upon oily sprays and small doses of quinine, with medium doses of the solution of ammonium acetate. Tincture of euphrasia officinalis given in small and frequent doses is said to be peculiarly effective in the onset.

TRAUMATIC RHINITIS.

Inflammation of the mucous membrane is not infrequently excited by dust and vapors of chlorine, iodine, or other irritating substances suspended in the atmosphere. It may also arise from the entrance of larger foreign bodies, or may follow direct injuries to the nose. The inflammation is not peculiar, and the remedies indicated for acute simple rhinitis are equally applicable here, except in case of fracture, when the parts must be replaced, and retained by nasal plugs and external splints. Hemorrhage should be controlled by the measures suggested for epistaxis, and if abscesses result the pus should be promptly evacuated.

The acute rhinitis due to the pollen of plants or other irritating particles will be considered under the head of hay fever, but there is a form dependent upon the specific effects of potassium bichromate, arsenious acid, and mercury which deserves special notice here. It is characterized by ulceration leading to perforation of the cartilaginous septum.

The ulcer is at first small and round, but subsequently enlarges and assumes an oval shape. Since it does not extend to the lower and anterior part of the cartilage, the bridge of the nose never falls in. Ulcers are also occasionally found on the turbinated bodies, but are less extensive than those on the septum.

SYMPTOMATOLOGY.—The symptoms produced by the bichromate are tickling and sneezing, accompanied by profuse secretion; this is at first watery, but subsequently it becomes thick and greenish, and later contains crusts or particles of sloughing mucous membrane, and finally pieces of cartilage; but it is never offensive. Hemorrhage frequently occurs in the course of ulceration. The symptoms produced by the other substances are said to be similar; and whichever of these substances is the cause, the symptoms seem to result entirely from local irritation.

TREATMENT.—Persons employed in trades where they are likely to suffer from this affection should constantly wear plugs of wool in the nostrils. Where perforation has once taken place, it is difficult to prevent the formation of a large opening, but ordinary treatment will soon check the surrounding inflammation. Those who have once suffered from this variety of traumatic rhinitis are said afterward to enjoy immunity from common catarrh.

CHRONIC RHINITIS.

Synonyms.—Rhinitis chronica, chronic catarrh, chronic coryza.

Chronic rhinitis is a chronic inflammation of the nasal mucous membrane characterized by dryness and the collection of crusts, or excessive secretion and discharge from the nostrils or naso-pharynx, with frequent inclination to hawk and clear the throat. Both conditions may be characterized by stoppage of the nares and interference with respiration. It is an affection found in nearly all climates and among all classes of people, and is most pronounced in the fall, spring, or winter months, when the temperature and moisture of the air are most changeable. It is most frequently met with near the northern seashore or on the borders of large lakes, yet it is prevalent even in some dry climates, especially where the air is filled with dust, as, for example, in Colorado and New Mexico. On the borders of the Great Lakes and at the seashore it is much more common among people who live within two or three miles of the water than among those farther inland, apparently owing to the greater exposure of the former to sudden changes, and to fogs and the damp, chilly winds, especially in the spring, when the southerly land winds have become warm and balmy, while the northerly winds sweep over water often still containing ice, and colder than the land. The affection is most frequent in children and young adults between the ages of ten and thirty-five years, but it often occurs in infants, and not infrequently in people past the prime of life. Persons follow-

ing outdoor vocations become less susceptible to the influence of sudden atmospheric changes, and are therefore less liable to this disease.

For convenience of description, chronic rhinitis may be divided into four varieties: *first*, simple chronic rhinitis, consisting of catarrhal inflammation with little or no swelling; *second*, intumescent rhinitis, a phase of the disease in which there is frequent swelling of the mucous membrane of the turbinated bodies or upper portion of the septum in one or both nares, which may come on speedily in one or the other side, and, after a time, may as quickly disappear, so that often when the nose is examined the cavities appear of normal size, though one or both may have been completely closed a short time before; *third*, hypertrophic rhinitis, an inflammation associated with more or less actual hypertrophy of the tissues; *fourth*, atrophic rhinitis, usually the sequel of the hypertrophic variety, in which the mucous and submucous tissues are wasted away, and as a result the nasal cavities become abnormally large. All these varieties usually originate in the same manner and frequently run the same course for a considerable period. The first variety is often but a commencement of the second, the second of the third, and the third of the fourth; but there are occasional instances in which either the second or third variety may begin or terminate without the supervention of the forms which generally follow, and there are occasional cases in which neither variety can be traced to any antecedent affection.

SIMPLE CHRONIC RHINITIS is a catarrhal inflammation of the mucous membrane attended by little or no swelling and characterized generally by great irritability and susceptibility to acute exacerbations. It is attended by congestion and by excessive watery or muco-purulent secretions.

ETIOLOGY.—The disease may be induced by the frequent repetition of any of those conditions which cause an ordinary cold. It may result from inhalation of irritating substances, exposure of the throat, back, ankles, or of the whole body to cold, or the inhalation of damp, chilly atmosphere. A predisposition to inflammation of the mucous membrane may be inherited, or acquired by frequent attacks of the acute disease. Debility and a depressed condition of the nervous system often directly favor the onset of the affection, and in many cases hyperæsthesia of the terminal nerve fibres in the Schneiderian membrane is apparently the predisposing cause. In some cases it is favored by a scrofulous or dartrous diathesis.

SYMPTOMATOLOGY.—There is usually a history of frequently recurring attacks of acute inflammation which have finally resulted in constant irritation that is likely to have continued for months or years before the patient has applied for relief. Itching, burning, and tickling sensations in the nose are common, and sneezing usually occurs on the

slightest provocation. Headaches and pain in the eyes are frequent symptoms. Not infrequently there is loss of the sense of smell, and partial deafness; and occasionally the sense of taste is obtunded. Profuse lachrymation is an occasional symptom, and in most cases there is a profuse watery discharge from the nose, recurring upon the slightest irritation such as breathing of cold air. In some persons, after a time, the secretions become muco-purulent and of a more or less offensive odor.

Usually the general health is not perceptibly impaired, but sometimes it is poor, with derangement of the digestive organs manifested by capricious appetite and a sluggish condition of the bowels. When the secretion is thin and watery, the mucous membrane will generally be found congested, of a bright red color, the surface moist, and a considerable amount of secretion collected in the lower part of the nasal fossæ. Frequently cobweb-like threads of mucus will be seen stretching from side to side of the nasal cavity, and occasionally small, opalescent, transparent, or yellowish granulations will be seen studding the anterior ends of the inferior turbinated body. These are about a millimetre in diameter and appear like solid masses, but when brushed over with the probe, they are found to be small drops of fluid. The nasal cavity normally is from three to five millimetres in width but in more than half of the cases examined, deviation of the nasal septum is present, or a cartilaginous or bony spur will be found projecting from one or both sides. These, however, may have no relation to the catarrhal condition, and are of no consequence as long as they do not obstruct nasal respiration. In most instances the mucous membrane of the naso-pharynx is congested, and here and there collections of tenacious secretions will be found adhering to its surface; or these may collect to be removed from time to time by the act of hawking. In rare instances the nasal cavity remains of normal size and free, excepting when obstructed by dry and decomposing secretion; if this be removed, the mucous membrane is found irregularly congested and of a bright red color in spots, or pale and anæmic. In most of these cases, the atrophic condition is present, but in others there are evidences of hypertrophy.

Diagnosis.—The diagnosis may be easily made by inspection and palpation of the part, with a consideration of the history. This form of chronic rhinitis is only likely to be mistaken for hay fever. The latter comes on at certain periods of the year, and is repeated season after season; while the former comes on at any time, and is apt to be continuous, with frequent exacerbations. Upon inspection of the part, the nasal mucous membrane is found congested, and palpation with the probe frequently reveals here and there sensitive spots, similar to those which are present in most cases of hay fever; but the hypertrophic or atrophic changes usually present in chronic rhinitis are not so common in hay fever.

Prognosis.—The affection runs a tedious course, sometimes lasting

for many years. Some cases eventually recover spontaneously, but others go on from bad to worse, and finally terminate in some of the other forms of chronic catarrh.

TREATMENT.—The treatment of this variety of rhinitis is tedious and often unsatisfactory, but usually considerable relief may be given and in some cases a cure effected by local applications. In the treatment, two objects are to be kept in view, viz., relief of irritability, and the checking of excessive secretion. If the secretions are profuse and watery, the nares will be kept clean, so that washes are unnecessary. In this class of cases, soothing powders or sprays are most efficacious, and mild astringents will often be found useful to toughen the membrane. All applications should be so mild as not to cause smarting for more than five minutes, and should, after brief discomfort, give a feeling of relief. The susceptibility of the mucous membrane varies greatly in different cases; therefore the mildest preparation should always be used in the beginning. Oily sprays are of utility in most cases. Those most commonly in use are derivatives from coal oil, such as oleum petrolina and liquid albolene; melted vaselin is also used for the same purpose. However, the effects of these are but tentative, and therefore they should only be prescribed for the patient to use at home two or three times daily. In some cases of profuse secretion I have obtained most excellent results by having the patient apply twice daily a spray containing ♏ x. of terebene ad ℥ i. of liquid albolene. Indeed, this has seemed more effective than any other local application.

A sedative powder consisting of about five or ten per cent of boric acid, twenty-five per cent of iodol, five per cent of starch, and sugar of milk to make one hundred grains, with occasionally one per cent of cocaine, may in some cases be applied in addition to the spray once or twice daily with much benefit. Certain patients in whom there is marked hyperæsthesia of the nasal mucous membrane, upon going into the wind or dust are subject to attacks of sneezing, accompanied by excessive secretion, necessitating almost constant use of the handkerchief. There is consequently soreness of the nose, which becomes the source of much annoyance. This is the most obstinate variety of simple chronic rhinitis, but fortunately it is rare. In searching for the sensitive spots a probe should be passed to the back part of the nasal cavity and drawn forward over the various parts of the mucous membrane; as a sensitive spot is touched, the patient winces from the pain or inclination to sneeze or cough, and sometimes says that the probe pricks or burns. The most effective treatment is superficial cauterization of the sensitive areas, as practised in the treatment of hay fever. Sedative powders and sprays should be used in the intervals between the cauterizations, which should not be made oftener than once in five to seven days. The cauterizations destroy the terminal fibres of the hypersensitive nerve, but are not deep enough to destroy the mucous membrane.

CHAPTER XXXI.

DISEASES OF THE NASAL CAVITIES.—*Continued.*

RHINITIS.—*Continued.*

CHRONIC RHINITIS.—*Continued.*

INTUMESCENT RHINITIS, also known as chronic catarrh, and by some considered as one of the forms of hypertrophic rhinitis, is the most frequent of all varieties of chronic rhinitis; it is characterized by intermittent swelling of the Schneiderian mucous membrane, with more or less occlusion of the nasal passages. The swelling may involve both cavities at once but usually affects one side at a time and may change in a few moments to the opposite naris. This is most noticeable when the patient is lying upon the side, the undermost cavity being occluded, but the swelling generally changes to the opposite naris within a few minutes after the patient turns over.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—The mucous membrane is usually congested, but is occasionally pale, and upon one or both sides may be swollen. The tumefaction is most frequently found over the inferior turbinated bodies, but it sometimes involves the middle turbinals and that part of the septum directly opposite. Frequently no swelling whatever is found at the time of examination, though the history clearly shows that it is present several times during the day or night. The swelling interferes with nasal respiration and favors accumulation of secretion in the nasal and post-nasal cavities, consequent partly upon deficient evaporation, and partly upon increased activity of the secreting glands.

In most cases the pharynx, and in many the larynx, finally becomes the seat of chronic inflammation; and in many cases partial deafness results from swelling of the mucous membrane in and at the mouth of the Eustachian tube. The pharyngitis and laryngitis, dependent in part upon extension from the nares, are chiefly the results of mouth-breathing, which becomes necessary when nasal respiration is obstructed.

ETIOLOGY.—The causes are those of simple chronic rhinitis.

SYMPTOMATOLOGY.—In most cases there is a history of unusual susceptibility to colds affecting the nasal cavities. These attacks are most common in the spring and fall months, though in some persons they are more frequent in winter, or occasionally even in warm weather.

After a variable time, during which the attacks of cold in the head have grown more and more frequent and prolonged, the affection finally becomes fixed and the patient is annoyed much of the time, especially at night, by obstruction of nasal respiration attended by hawking and efforts to clear the throat, particularly in the morning or after eating. When tenacious mucus adheres to the upper surface of the palate, the violent effort to dislodge it often causes vomiting. Often the patients are annoyed by slight hacking cough, and by frequent hoarseness, especially on attempting to sing. By Raulin, of Marseilles (*Revue de laryngologie, d'otologie et de rhinologie*, *Annual of the Universal Medical Sciences*, 1892), this is attributed to muscular fatigue caused by excessive vibrations of the vocal bands in an effort to compensate for the loss of resonance caused by the nasal obstruction. In such cases the voice has often been speedily restored by reducing the hypertrophies of the septum or turbinated bodies. Nevertheless many persons who suffer from all the symptoms of nasal obstruction become so accustomed to it as scarcely to recognize the fact, and when questioned, affirm that they have no difficulty in breathing through the nose. They claim to sleep well, and assure the physician that the throat is not dry in the morning, that they always sleep with the mouth closed, notwithstanding the fact that inspection shows the nares to be more than half closed by swelling. Many complain of headache especially in the morning, of pains in the eyes, of frequent hawking to clear the throat, or a slight hacking cough, of dropping of mucus into the throat from the naso-pharynx, and of obstruction in the nares, especially upon taking cold, which they contract very easily.

The symptoms in mild cases usually disappear during the summer months, or upon change of climate, even though it be but a slight change. This is peculiarly noticeable when patients leave the vicinity of our northern lakes, especially in the spring and early summer when the waters are icy or cold. In some there may be little difficulty in temperate weather; but in extremely cold or extremely warm weather, or upon slight exposure to draughts, or change of temperature as in going from a warm to a cold room, or the reverse, or even from the shade into the bright sunshine, there is a tendency to sneeze, followed by speedy closure of one or both nares. I have seen one patient suffering from this form of catarrh who would always sneeze upon going into bright gaslight. Sometimes the inhalation of smoke or of odors from certain plants, or drugs, will irritate the mucous membrane and excite excessive secretion, with swelling. Many patients experience sensations of itching or tickling in the mouth, or a feeling of dryness, fulness, pressure, or stuffiness in the nose, as the principal symptoms. Often the pharynx feels dry or uncomfortable, especially in the morning, and sometimes obstinate pricking or neuralgic pains are experienced in the fauces.

Occasionally the patients are annoyed by repeated attacks of redness

and inflammation of the end of the nose. In many instances the voice is thick or nasal, and it often becomes hoarse from the accompanying laryngitis, so that patients are usually unable to sing or shout, and easily become fatigued upon prolonged talking. Such persons are generally obliged to keep the mouth partially open much of the time, particularly when walking in the wind or during active exertion, and they are frequently in the habit of yawning or taking deep respirations to make up for the constantly deficient supply of oxygen.

The secretions may or may not be increased; they may be thin and watery or thick and tenacious, or they may dry into crusts which are removed every two or three days from the nostrils or naso-pharynx. In the nose these crusts are most likely to collect upon the anterior part of the septum, or the anterior ends of the middle turbinated bodies. Frequently fine cobweb-like shreds of mucus will be seen stretching from the turbinated bodies to the septum, as in simple chronic catarrh. If the secretions collect and remain for any length of time, they become partially decomposed and offensive, giving the peculiar catarrhal odor, familiar even to the laity. The tongue is commonly coated with a white or yellowish fur, especially at its base, and the digestive system is so frequently disturbed as to lead to the belief that in some cases it is the direct cause of this disease. Gaseous eructations from the stomach, and constipation, are frequent concomitants.

Upon inspection, the mucous membrane is usually found congested, though occasionally it may be paler than normal; and one or both nasal cavities are found to be from one-third to two-thirds closed by swelling of the inferior turbinated bodies. In many cases, no swelling is observed at the time of the examination; but on the other hand the nares may be completely obstructed. Swelling of the soft tissues over the septum is not infrequently observed, especially running horizontally along its upper half, and it is not unusual to find similar swellings running vertically from half to two-thirds the whole height of the vomer near its posterior border. The swollen membrane at the upper part of the septum is usually of a slightly deeper hue than normal; that seen with the rhinoscope at the posterior border is of a grayish color. The posterior ends of the inferior or middle turbinated bodies are sometimes found much swollen and of a grayish hue; but this is more commonly present in hypertrophic rhinitis. By examination with the probe, exquisitely sensitive spots are frequently detected, as in simple chronic rhinitis. Whenever swelling is present, the soft tissues may be easily pressed down until the bone is felt beneath, but the dent thus formed quickly disappears as the probe is removed. Upon palpation, in this way, the mucous membrane over the septum will often be found swollen two or three millimetres in thickness, and that over the turbinated bodies from two to five millimetres. In uncomplicated cases of this affection, upon the insufflation of one or two grains of a four per cent powder of cocaine, or spraying the nares with a weak solution of the same

drug, the swelling will speedily subside and the cavities appear of normal size. Sometimes this occurs spontaneously during the examination, from the fright caused by suggestions as to the proper treatment. Sometimes the swelling will promptly disappear upon exercise, and it is not uncommon for patients to find that they can breathe much more easily after going upstairs, or for them to say that they have to get up and walk about in the night in order to breathe.

DIAGNOSIS.—The affection is to be distinguished from simple chronic rhinitis, from hypertrophic rhinitis and from nasal mucous polypi.

Intumescent rhinitis is differentiated from *simple chronic rhinitis* by swelling of the mucous membrane, and the occurrence of frequently repeated nasal obstruction.

It is distinguished from *hypertrophic rhinitis* by the intermittent character of the swelling instead of permanent occlusion of the nares; by the smooth surface of the membrane in place of an uneven, nodular appearance, and by disappearance of the swelling under the action of cocaine, which does not affect true hypertrophy.

We find that *nasal mucous polypi* are of lighter color and more mobile; a probe may be readily passed upon either side of them, whereas it can only be passed upon one side of the swelling in intumescent rhinitis, and, although in the latter affection the swollen tissue may be compressed, the enlarged body cannot be moved upon its base as can a polypus. Again, cocaine diminishes the swelling in intumescent rhinitis, whereas it renders the mucous polypus, in most instances, more prominent by diminishing the swelling about it.

PROGNOSIS.—If left to itself, spontaneous recovery from the disease occurs in a few cases, but usually it extends over months or years, and eventually terminates in hypertrophic rhinitis, though occasional cases appear to pass directly into the atrophic form. The frequent occlusion of the nares leads either to pharyngitis or laryngitis, or both; in many cases, throat-deafness results from involvement of the Eustachian tube, the inflammation extending not infrequently to the middle ear. The general health suffers from imperfect oxygenation of the blood; and although to the casual observer the patients may appear well, they become easily fatigued, are unable to stand exercise, and are often subject to illness upon slight exposure. These tendencies may not be recognized until the marked improvement in the patient's general condition, under appropriate treatment of the nasal affection, demonstrates that they have been present.

TREATMENT.—Prophylactic treatment is of the greatest importance in all persons predisposed to catarrhal affections. They should avoid exposure to draughts or cold or to undue heat, especially in badly ventilated rooms, and so far as possible the inhalation of air containing irritating substances. Woolen underclothing should be worn the year round. The daily practice of invigorating exercise, with cold sponging of the body, followed by vigorous friction, and bathing the feet morn-

ings in cold water, are often useful adjuvants in the prevention of colds. Acute rhinitis occurring in individuals thus predisposed should be cured as speedily as possible. In all cases the condition of the digestive organs should receive careful attention. In the early stages the regular use by the patient of sedative remedies, and the occasional application of mild astringents or stimulants to the nares, constitute the best means for the cure of the disease.

The milder stimulating applications, which may be made two or three times per week, consist of aqueous solutions of zinc sulphate, carbolic acid, and zinc chloride (Form. 94), of sufficient strength to cause smarting or discomfort for not more than ten minutes. Aqueous solutions may be employed for home use two or three times daily, such as: boric acid gr. x. ad ℥ i., or listerine ♏ xl. to lx. ad ℥ i., or sodium bicarbonate and biborate āā gr. iss. to ij. ad ℥ i., or distilled extract of hamamelis or of pinus canadensis ♏ xxx. to l. ad ℥ i. A saturated solution of boric acid in camphor water is also a useful soothing application. Oily preparations such as oleum petrolina or liquid albolene containing camphor gr. i. to ij., menthol gr. ss. to i., oil of cloves ♏ iij. to v., or terebene ♏ viij. to xij. ad ℥ i. (Forms. 105, 106) are generally more beneficial than the aqueous solutions. The oleaginous liquid alone may be used as a soothing application to prevent the contact of irritating substances with the mucous membrane. In addition to these, the sedative powders already mentioned in speaking of simple chronic rhinitis (Form. 166) may also be employed once or twice daily with benefit in certain cases.

Cocaine in any quantity should never be used continuously, not only because of the danger of forming the cocaine habit, but because when used for any length of time it seems partially to paralyze the vasomotor nerves, thereby causing turgescence of the cavernous tissue and thus increasing the difficulty we are trying to remove; but it will be found most efficient in temporarily removing swelling and relieving the acute exacerbations of this affection. Cocaine is most conveniently employed in powder (Form. 166), which may be blown into the obstructed nostril two or three times in twenty-four hours, in quantities not to exceed one-thirtieth of a grain of cocaine at a dose. Even in this quantity it should only be used for a few days, and it is seldom necessary then excepting at night or early in the morning.

For the application of powders to the nares, I give patients a short glass tube about four millimetres in its internal diameter and four inches in length, flattened and expanded at one end, but round at the other (D B, Fig. 195).

A small quantity is worked into the round end by moving it about in the powder; the end of a piece of rubber tubing about nine inches in length is then slipped over the same end of the glass tube; its flattened end is placed in the nostril, the other end of the rubber tube between the lips, and the patient gives a short, quick puff, which blows the powder into the naris. The rubber tube is made of the common

drainage or nursing-bottle tubing with a calibre of about three millimetres. When the physician makes the application himself, it is best to use a hand-insufflator (Fig. 195). Any application which is made as often as two or three times a day should not cause smarting or discomfort for more than three to five minutes, and should make the patient subsequently feel better, instead of worse; but stronger applications, as already recommended, may be made every two to five days. The sprays may be applied by means of any suitable atomizer. The atomizer which I have found most satisfactory is shown in Fig. 196.

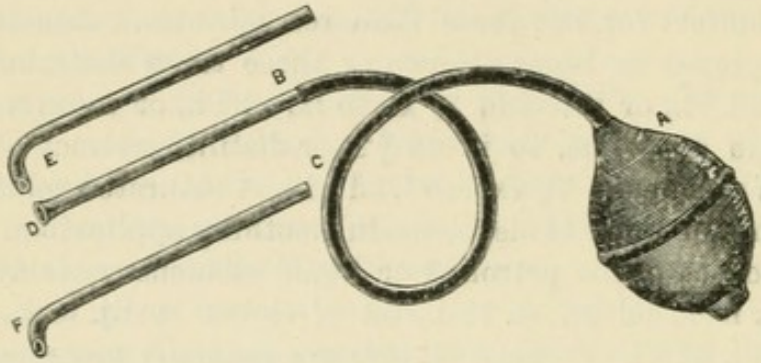

FIG. 195.—POWDER BLOWER. Three glass tubes (½ size). Straight tube for nasal, bent tubes for naso-pharyngeal or laryngeal applications.

When secretions collect in large quantities, the patient should wash the nose once or twice daily with an alkaline solution, or with a salicylate solution (Form. 187). An excellent alkaline solution may be made by dissolving an even teaspoonful of sodium bicarbonate in a half-pint of lukewarm water, or one-half of a teaspoonful each of sodium bicarbonate and sodium chloride in the same amount of water. In some instances sodium chloride alone, in the same proportion, seems to answer a better purpose. This I recommend in cases where the sodium bicar-

FIG. 196.—DAVIDSON'S OIL ATOMIZER, No. 50 (¼ size).

bonate causes an uncomfortable sensation of dryness. After the nose has been thoroughly cleansed, the applications already recommended should be made. In fully developed cases of intumescent rhinitis these remedies will give the patient temporary relief, but can seldom if ever effect a cure, and they should therefore only be employed as an aid to more radical treatment, which consists of the cauterization of the swollen

tissue either by chemical agents or by the galvano-cautery; or in the removal of portions of the tissue with the steel-wire snare. The latter is better suited to the case of hypertrophic rhinitis. Before cauterization, the part should be thoroughly anæsthetized by cocaine, as recommended in speaking of hay fever.

Of the various chemical agents which have been recommended, strong acetic or chromic acid is most useful, and of these two the latter is more generally preferred by laryngologists. It may be employed in solutions of fifty to seventy-five per cent, or preferably a small amount of the acid may be fused upon an aluminium probe (Fig. 197) and employed in the solid form. I always apply it, if at all, in the latter manner, since its effects can be better controlled, and injury to other parts can be more easily avoided. A few of the crystals of chromic acid being placed upon the end of the flat aluminium probe, it is held over the flame in such position that the acid slowly fuses, and then so that it cools upon the desired place. The fused acid is then rubbed over the part to be cauterized, which becomes of a brownish color, and immediately afterward an alkaline spray is thrown into the nostril to neutralize any excess of acid, and to prevent it from being diffused to

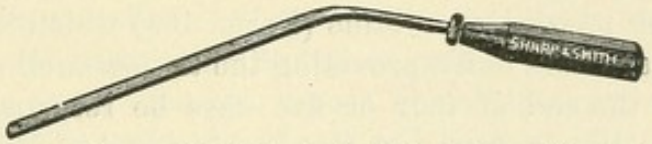

FIG. 197.—FLAT NASAL PROBE (2-5 size). Made of aluminium and bent at an angle of 35°.

other parts. The amount of acid used at one time should not exceed four or five times the bulk of a pin's head or about two-thirds the bulk of a flax-seed. The acid should be applied along a narrow strip of membrane about three or four millimetres in width and from ten to twenty in length according to the depth of cauterization, care being taken not to use too much acid at one time or to cauterize too large a surface. Bosworth prefers touching only at separate points with the acid, claiming that the small eschars, as he expresses it, pin down the mucous membrane to the bone beneath; but in my hands this plan has been less satisfactory than the one already recommended. I would not advise a repetition of cauterization until complete healing has occurred, which will require from ten to twenty days. H. Holbrook Curtis, of New York, who has had excellent results in the treatment of this form of catarrh, informs me that he touches the lower half of the inferior turbinated body along its whole length with chromic acid, which he commonly uses in strong solution, and repeats the cauterization within four or five days.

Chromic acid causes much more pain than the galvano-cautery, a more irritating discharge, and a sore which heals more slowly than that by the latter, while its effects cannot be so accurately controlled. The treatment is therefore more tedious and gives the patient much

more discomfort, and the result is no better than that obtained by the hot electrode.

In using the galvano-cautery I employ an electrode (No. 2, Fig. 91), with a blade about fifteen millimetres in length consisting of No. 21 platinum wire. One, two, or more narrow, linear incisions the whole length of the turbinated body, and deep enough to just graze the bone in two or three places, should be made, one at a sitting, with a sufficient interval for healing to occur before the cauterization is repeated. These lines are usually made at the junction of the middle with the inferior or superior third of the lower turbinated body; and in from ten to fifteen days afterward, a similar cauterization is made upon the other side. In the same length of time subsequently the first cauterization will have healed, and if necessary the treatment may be repeated upon the side first treated.

Immediately preceding or following the cauterization I apply to the nares a solution of ♏ v. ad ℥ i. of oil of cloves in liquid albolene, and after the cauterization follow this by the insufflation of two or three grains of iodol. A light pledget of cotton is then placed in the nostril, and the patient is directed to wear this, changing it as he wishes, for the next forty-eight hours, whenever out of doors. He is also given a four per cent powder of cocaine (Form. 168) which he is directed to use three or four times daily, providing the tissues swell so as to occlude the nares. At the end of four or five days he returns, and a probe is passed between the septum and the turbinated body to prevent adhesion; or if the thick mass of exudate, resembling false membrane, which usually covers the wound, is still present, it is gently removed, and the line of the cauterization touched with a ten grain solution of silver nitrate; or the parts are simply sprayed with a stimulating solution of zinc sulphate and carbolic acid, āā gr. ij. ad ℥ i. The patient is then given, to use once or twice daily, instead of the powder first employed, a similar powder to which has been added twenty-five per cent of iodol.

In most cases two or three times each day after the cauterization the patient also uses at home a spray containing gr. ⅓ of thymol, gr. ss. of carbolic acid, and ♏ iij. of oil of cloves ad ℥ i. of liquid albolene, or, if this causes any irritation, a still milder application. Most patients find this soothing, and it prevents the formation of dry scabs; but for patients to whom oleaginous sprays of any form are irritating, a spray of boric acid, gr. viij. ad ℥ i., will be found most beneficial; though any of the soothing sprays already recommended may be employed to suit the indications of the case or the fancy of the patient. If the soft tissues over the middle turbinated body or the septum swell, they may be treated in the same manner.

In a few cases a single cauterization upon each side will be sufficient to effect a cure, and in the great majority of cases two cauterizations upon

each side are sufficient; but occasionally three, four, or even more will be necessary before the disease is checked. During the treatment, and for a few weeks afterward, it is usually best for the patient to use some of the sedative or slightly stimulant sprays recommended for the treatment of mild cases of the disease. If the treatment is properly carried out recovery may be confidently expected in at least nineteen cases out of twenty. The treatment usually requires from six to twelve weeks with an average attendance at the physician's office of about once a week, though many cases are cured much more promptly, and rare cases demand more extended treatment.

In using the galvano-cautery, I employ a current sufficiently strong to heat the platinum wire to a white heat within two seconds after contact is made. The electrode having been carried to the back part of the tissue to be cauterized, and turned so that the platinum wire rests against the tissue, the circuit is closed, and as soon as the sound of burning is heard, the electrode is drawn slowly forward, or, if the bone is not felt, moved slightly backward and forward until it grazes the bone, and then drawn slowly to the anterior end of the turbinated body, where it should be lifted from the soft tissue before the current is turned off, and then allowed to cool before it is withdrawn from the nostril. If the circuit is broken before the electrode is lifted from the tissues, the eschar is pulled off with it and bleeding results. If the wire is too hot, it cuts like a knife, and much bleeding may follow; if it is only of a cherry-red heat, or if it is too small, it will cut through the mucous membrane too slowly, so that the time necessary for a sufficiently deep cauterization will allow enough radiation of heat to burn surrounding tissues.

Occasionally, in spite of all precautions, adhesions will take place between the two walls of the nasal fossa, though this is not apt to occur except where there is hypertrophy of the turbinated bone, or an outgrowth or deflection of the septum. If adhesions form, they must be cut or broken down, and the parts kept apart by a pledget of wool or bit of rubber or gutta-percha until healing occurs. Sometimes an application of monochloracetic acid will prevent subsequent adhesions.

When patients find it inconvenient to call within four or five days after the cauterization, they are directed to come again at any time that suits their convenience after two weeks, and most of them will progress very well in this way, though there is more liability to adhesion, and occasionally the wound does not heal as it would if proper attention could have been given at an earlier date.

In a few cases too much reaction will follow a cauterization of the extent recommended; in these a line only half way across the turbinated body should be made at once. Usually the treatment causes little or no pain, and no subsequent inconvenience except such as would be experienced from an acute cold in the head. The discomfort following

the cauterization most frequently results from the cocaine; it may often be relieved by a cup of strong coffee or ten to fifteen grains of potassium bromide. Headache occasionally follows, which is best relieved by five or ten grain doses of phenacetin, repeated in one, two, or three hours as needed. Coexisting pharyngeal or laryngeal inflammation should receive appropriate treatment at the same time; though the physician may with perfect candor assure his patient that, as soon as the nasal obstruction is removed, at least four-fifths of the difficulty arising from the other affection will disappear, and that the remaining trouble will probably disappear within a few months even without treatment. In this form of rhinitis a slight change of climate, especially moving from the vicinity of large bodies of chilly water, will often give immediate relief, though the affection is liable to recur as soon as the patient returns to his former abode.

Hypertrophic rhinitis is a common affection, next in frequency to intumescent rhinitis. It is usually characterized by excessive discharge from the nostrils or into the naso-pharynx, with hawking and clearing of the throat, and more or less permanent obstruction of the nares, though it varies much from time to time in consequence of the swelling.

Anatomical and Pathological Characteristics.—The mucous membrane is usually congested, but may be paler than normal, and hyperplasia of the mucous and submucous tissues causes permanent thickening of the turbinated bodies, especially the inferior (Fig. 198), and sometimes also of the septum, usually at its upper part.

Occasionally the bones themselves are likewise hypertrophied, and constantly narrow the lumen of the nares. The condition may be present upon one side only, but commonly involves both. It is frequently associated with deflection or exostosis, or enchondrosis of the septum, in which case the inferior turbinated body upon the concave side of the septum is apt to be much more hypertrophied than its fellow; indeed, the latter will sometimes be found atrophied, so that patients can breathe more easily through the side which appears most obstructed. In addition to hypertrophy, swelling of the soft parts is usually present, so that the nasal cavity is from one-half to two-thirds closed or entirely obstructed.

Etiology.—Hypertrophic rhinitis is usually preceded by frequent attacks of acute catarrhal inflammation, from which intumescent rhinitis is at length developed, finally terminating in true hypertrophy. It is produced by the same conditions that cause the intumescent form of the disease.

Symptomatology.—The patient usually states that for a long time he has taken cold easily, and for several months or years has been annoyed by stopping up of the nose, especially at night or in the early morning, and by excessive discharge from the nostrils, or into the naso-

pharynx, with hawking and clearing of the throat, or hoarseness. More recently one or other naris has been constantly obstructed, so that the mouth must be kept open upon any exertion and during sleep. Frequently the sense of hearing is obtunded; indeed, most cases of deafness are the result of hypertrophic rhinitis. Frequently the general health suffers in consequence of imperfect oxygenation of blood. Often the patient suffers from frontal or occipital headache or a feeling of pressure over the bridge of the nose or forehead, and occasionally the eyes are affected so that reading is painful or impossible, except for

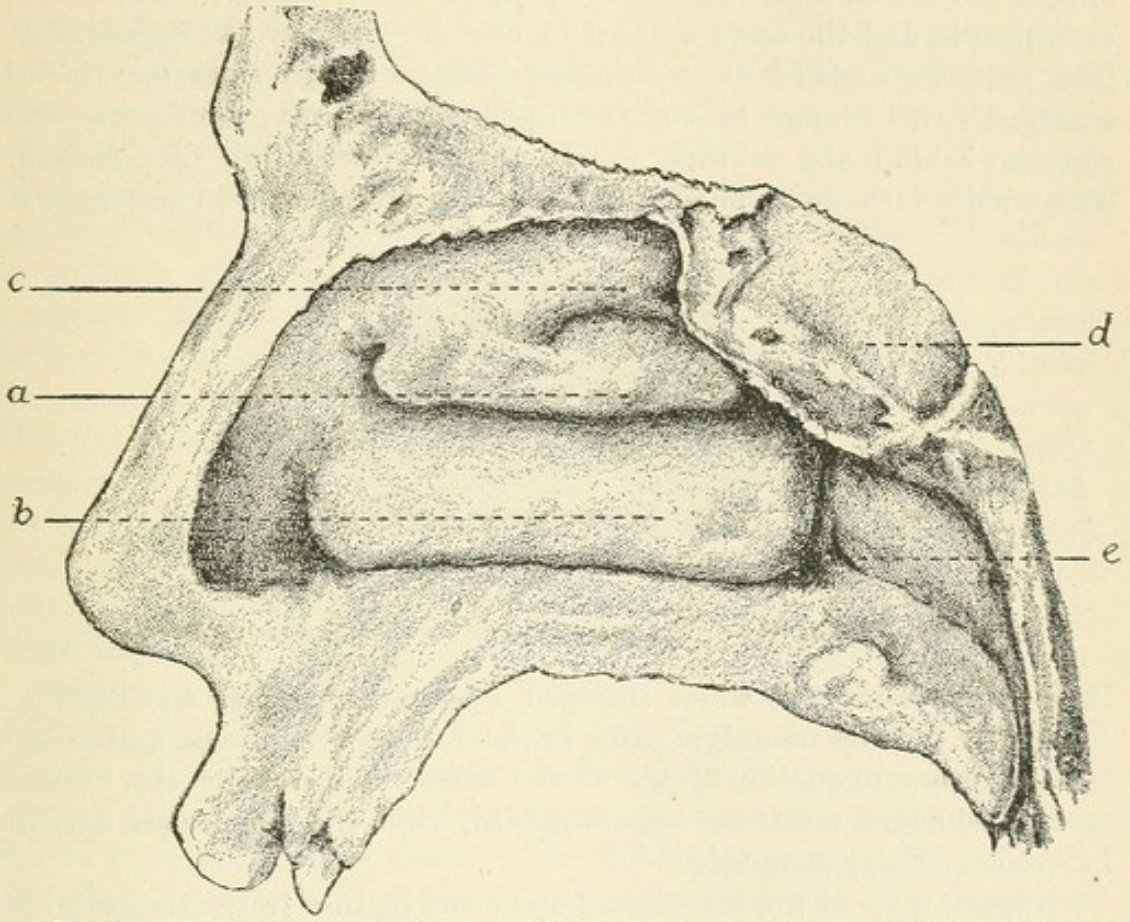

FIG. 198.—HYPERTROPHY OF INFERIOR TURBINATED BODY. Cross-section of head, from frozen section. *a*, Middle turbinated body; *b*, inferior turbinated body hypertrophied; *c*, superior turbinated body; *d*, sphenoid cells; *e*, orifice of Eustachian tube.

a few minutes at a time. There is usually some dysphonia and dyspnœa, the mucous membrane, especially over the inferior turbinated body, is thickened, and its surface is usually more or less uneven in appearance, sometimes presenting distinct nodules. The amount of swelling varies much from time to time, and it may be uniform over the whole turbinated body or limited to portions of it. Thus, it is common to find either the anterior, middle, or posterior portion of the cavity most occluded; or along the upper portion of the turbinated bodies there may be but little thickening while the lower portion touches the septum, the inferior border resting upon the floor of the nares. Whenever the

mucous membrane of the two sides of the nasal cavity is in contact, we usually find a considerable collection of mucus or muco-pus at the lower portion of the fossa. In many cases cobweb-like shreds of mucus will be found extending from side to side as in other forms of rhinitis already discussed, or the dried secretions may have collected in crusts upon the cartilaginous septum, or about the middle turbinated body. Usually the vault of the pharynx is congested, and contains tenacious mucus or dried masses, and the posterior ends of the inferior or middle turbinated bodies are enlarged (Fig. 199). These commonly appear in the rhinoscope of a gray color, but occasionally of darker hue, even purple, and the surface has a nodular or raspberry-like appearance. The posterior ends of the turbinated bodies of both sides may be so enlarged as to project into the naso-pharynx, and may even come into contact behind the septum, nearly or quite occluding the choanæ. The middle turbinated bodies are much less frequently hypertrophied

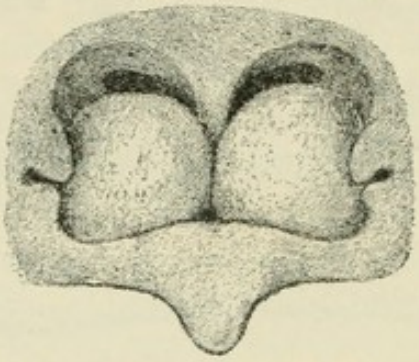

FIG. 199.—HYPERTROPHY OF POSTERIOR ENDS OF INFERIOR TURBINATED BODIES.

than the inferior, but when enlarged they press against the septum, frequently causing neuralgic pains in the forehead and eyes, and sensations of pressure on the bridge of the nose. Occasionally the middle turbinated bodies are found hypertrophied, while the inferior are normal in size or perhaps atrophied.

Hypertrophy of the soft tissues upon the septum in the majority of cases is found at its middle or upper third, running nearly horizontally, or extending vertically near the posterior edge of the vomer.

DIAGNOSIS.—Unless the parts are carefully examined, the affection is apt to be confounded with any of the other causes of nasal obstruction; but by a consideration of the history, and a careful inspection and palpation of the parts, it may be easily distinguished from all diseases except intumescent rhinitis and syphilitic affections of the nose.

The tissues are easily impressed with the probe in *intumescent rhinitis*, and swelling rapidly and completely disappears on application of cocaine, signs not obtained in true hypertrophy.

It is impossible to distinguish hypertrophic rhinitis from *syphilitic disease of the nose*, attended simply by persistent swelling without ulceration, except by careful consideration of the history and watching re-

sults of specific treatment. It is often difficult to get the specific history of syphilitic patients, for reasons already indicated.

Excessive hypertrophy of the anterior or posterior end of the turbinated bodies is distinguished from *mucous polypi* by inspection, and palpation with the probe, which can be passed between a polypus and the external wall, but cannot be so manipulated in hypertrophy. The posterior end of the turbinated body when hypertrophied has much the color of a mucous polypus, but its surface, unlike that of a polypus, is uneven and slightly nodular, and it is usually of a deeper hue and has not the translucent appearance of the polypus.

Prognosis.—Hypertrophic rhinitis left to itself may extend over a period of several years. I have known of no case terminating in less than one year, but have seen one well-marked case where the affection merged into atrophic rhinitis within eighteen months. In many instances the hypertrophy gradually increases or, after a certain point has been reached, appears to remain without change; but in a considerable number of cases, atrophy finally begins and continues until the secretions become much altered, and the cavities greatly enlarged and more or less obstructed by decaying mucus and muco-pus, which cause the offensive odor of ozæna. In more favorable cases, atrophy continues for a time until the nasal cavities once more become free, and then ceases, whereby spontaneous recovery results. There is a common belief with the laity, and among physicians who have been in practice for more than ten or fifteen years, that little or nothing can be done for chronic catarrh by treatment; and this belief was well founded until the advent of the improved methods of treatment in vogue during the last decade.

Although the general health is often somewhat impaired by this affection, there is little or no evidence that it ever terminates in tuberculosis. It is true that patients suffering from chronic catarrh frequently die of tuberculosis, but apparently no more frequently than those free from the nasal disease. On theoretical grounds, it would appear that obstruction of the nares, by interfering with free expansion of the lungs, would sooner or later cause collapse of some of the air cells, with a consequent chronic inflammation and finally tuberculosis. I have seen some cases which seem to substantiate this hypothesis.

Treatment.—Various medicinal substances have been recommended internally and locally for the cure of hypertrophic rhinitis, but none of them are of much value excepting when used in connection with proper surgical measures; and a cure can seldom be effected except by the removal of some portion of the redundant tissue. This may be accomplished by means of chemical caustics, the galvano-cautery, burrs, trephines, scissors, saws, or the snare. Among the chemical agents which have been recommended are the mineral acids, especially nitric and sulphuric, solution of mercury nitrate, London paste, glacial acetic, and chromic acid; all of these have passed into general disuse excepting

acetic and chromic acid. The former, especially, in the form of monochloracetic acid, is useful particularly in cases where there is liability to adhesion of the opposing surfaces after cauterization, and either this or the glacial acetic acid may be used to reduce hypertrophy of the soft tissues, but they are less efficient than chromic acid, which, though an effectual remedy, is open to the objections mentioned under intumescent rhinitis.

Injections of carbolic acid, beneath the mucous membrane, by means of a hypodermic syringe, have been recommended, and the treatment appears to have been successful in some instances.

The majority of cases may be cured by cauterization as already described in the treatment of intumescent rhinitis. I prefer the galvano-cautery for most cases, and make linear incisions, as already recommended, two, three, or more of which may be necessary upon the inferior and possibly the middle turbinated bodies of each side. In cauterization of the middle turbinated body, I frequently use a small loop-like or pointed electrode (No. 3 or 4, Fig. 91), which is thrust into the lower edge of the turbinal in three or four places. In cauterizing the inferior turbinated body I sometimes use the same lance-pointed, slender electrode, and carry it all the way from before backward beneath the mucous membrane without burning through to the surface except at the points of entrance and exit. In seventy-five per cent of cases, not more than two lines are necessary upon either turbinated body, and in only five or ten per cent will more than three be needed. When the middle turbinal is involved, generally one or two cauterizations are all that will be useful, and if they do not succeed, some portion of the bone must be removed.

In hypertrophic rhinitis, Harrison Allen has recommended pressing the incandescent loop of the galvano-cautery into the tissue and drawing it forward until a small piece has been scooped out by the burning wire. In some cases, especially in hypertrophy of the middle turbinated body, when the soft tissue stands out prominently it may be caught and removed by the galvano-cautery écraseur, particularly where there is objection to the bleeding which would follow removal by the cold steel wire. When there is great hypertrophy of the soft tissues, it is far better to remove the redundancy by the scissors or snare. Sometimes with the nasal scissors (Fig. 200) I cut off the lower edge of the inferior turbinated body, but I prefer the snare where the wire can be made to hold. As a rule, in all these operations the parts should first be thoroughly anæsthetized by cocaine, but sometimes the swelling is so reduced by this agent that the snare cannot be made to hold, whereas the redundant tissue could be easily secured before the cocaine had been applied. In such cases it is sometimes best to introduce and tighten the snare first and subsequently to apply cocaine. In those patients who can easily endure pain the snare may be used with-

out cocaine, being gradually tightened until it causes the patient to wince; then after resting two or three minutes it is tightened still more until it again causes pain, when another rest is taken; this process is continued until the mass is cut off. This slow process has the advantage of causing a minimum amount of bleeding. In hypertrophy of the anterior end of the turbinated body, if the snare cannot be made to hold alone, the tissue may be transfixed with a needle, as recommended by Jarvis, of New York, the wire being slipped over the end of the needle and tightened down behind it.

In posterior hypertrophy the snare should be armed with a No. 5 steel piano-wire; the loop, of proper size, should be bent sharply over the end of the canula, as recommended by Bosworth; and then drawn slightly into the canula to straighten it during introduction into the naris. When it has been passed to the back part, the wire is again crowded forward until the bend is brought to the end of the canula, when it springs outward, and may be made to engage the diseased mass.

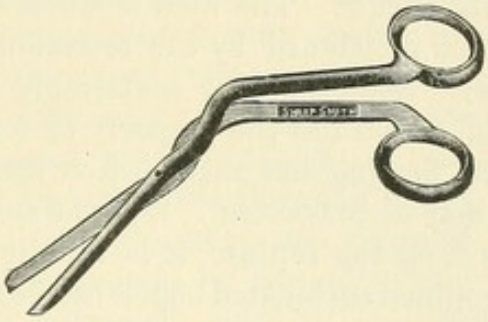

FIG. 200.—INGALS' NASAL SCISSORS (½ size).

The end of the snare should then be pressed firmly against the turbinated tissue, the wire drawn taut, and subsequently gradually tightened by the milled wheel. When this method is practicable, it is to be preferred to the slower process of cauterization, for by it a large amount of the redundant mass is at once removed, and the reaction which follows, as well as the consequent discomfort to the patient, is much less than after cauterization.

When any operation liable to be followed by much bleeding is done, the naris should be tamponed with lint or gauze, as recommended in speaking of epistaxis and the operation for exostosis. Even in cases where the snare or the scissors are applicable, it is usually also necessary to cauterize. It will be seen that the treatment of this affection is essentially the same as that of the intumescent variety of rhinitis, except that here we desire to remove redundant tissue, while in simple swelling we aim to destroy as little tissue as possible. In both instances it should be the effort of the physician *to save as much mucous membrane as would normally cover the parts*, and to form as little cicatricial tissue as possible. In a considerable number of cases of hypertrophic rhinitis the bones are also enlarged so much

that no treatment of the soft tissue can sufficiently remove the obstruction. In these the bony tissue may be removed with saw and scissors, or better with the dental burr (Fig. 201) or the nasal trephine (Fig. 202). These instruments, attached to the electric motor or dental engine, are run beneath the mucous membrane, enough of the bone being removed to allow the soft tissue to contract until sufficient space is obtained. Between the operations the same sedative or slightly stimulating powders and sprays should be employed that were recommended for

FIG. 201.—NASAL BURRS (actual size).

treatment of intumescent rhinitis. If adhesions of the opposing surfaces occur, they must be broken down or cut with scissors, and the surfaces kept apart by gutta-percha, or a rubber plug, or by a pledget of wool, until healing occurs. The wool is much better than cotton, as it becomes larger when moistened by the secretion, whereas the cotton plug becomes smaller. Sometimes by cauterizing the raw surface with monochloracetic acid, which has the property of forming an eschar that usually remains until healing has taken place beneath, subsequent adhesions of the part may be prevented. Where a spur of cartilaginous or bony tissue projects from the septum, it is usually necessary to remove it before the hypertrophied turbinated bodies can be satisfactorily treated; otherwise adhesions are very apt to take place.

Metallic, gutta-percha, or soft-rubber tubes, sponge and laminaria tents, have also been recommended for the cure of hypertrophic rhinitis.

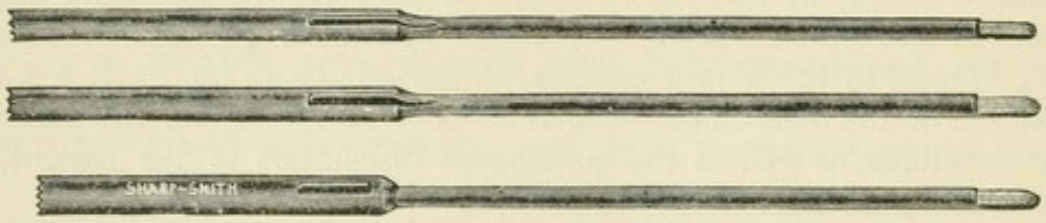

FIG. 202.—NASAL TREPHINES (actual size). Modification of Curtis.

When tents are used which swell by absorbing moisture, they should be allowed to remain for only a short time, and should be moved slightly back and forth frequently as the swelling progresses, to prevent them from becoming fixed too firmly in the cavity. Tubes may be introduced and worn for several hours at a time, providing they do not cause too much pain; theoretically, this procedure is excellent, but practically a tube large enough to affect all of the diseased tissue can seldom be introduced into the nostril. Furthermore, in the majority of cases the nares are so sensitive that tubes cannot be tolerated; therefore, this form of treatment has been abandoned except for some special cases.

Whatever treatment is adopted, the cavity should not be made larger than normal. Frequently patients will urge the physician to make it so large that they will never be troubled again, even upon taking cold; but this procedure is injudicious, and would subsequently be regretted by both patient and physician; for if the calibre is greater than normal, secretions are liable to collect, decompose, and give offensive odors, as in atrophic rhinitis. It is better to do too little than too much; but the patient should not be kept under treatment while we are accomplishing nothing. The physician must not be contented with making soothing applications which give but temporary relief. These can be made quite as well by the patient, and if for any reason the soothing form of treatment seems best, we are to remember that no good will result by seeing the patient oftener than once or twice in a month.

SUBMUCOUS INFILTRATION OF THE SIDES OF THE VOMER is common in chronic rhinitis, especially in the hypertrophic variety; it is characterized by more or less difficulty in nasal respiration and increased secretion. It is often associated with chronic inflammation of the pharyngeal mucous membrane, and sometimes with adenoma of the vault of the pharynx. The altered mucus collects in the posterior nares and drops into the throat or causes frequent hawking. The symptoms are those of post-nasal catarrh. Inspection by the aid of the rhinoscope reveals a yellowish white or gray puffiness on one or both sides of the vomer, near its posterior margin (Fig. 203).

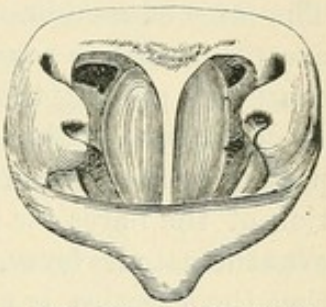

FIG. 203.—SUBMUCOUS INFILTRATION AT SIDES OF VOMER (COHEN).

DIAGNOSIS.—There can be no difficulty in the diagnosis when pharyngeal affections have been excluded and the characteristie appearances just mentioned are discovered.

TREATMENT.—We should contract or destroy the œdematous tissue by means of the galvano-cautery, or we may tear it off with forceps. The former is most effective. Astringents have little effect.

ATROPHIC RHINITIS is a chronic inflammation of the nasal mucous membrane, characterized by abnormal enlargement of the cavities, and the collection within them of drying secretions, giving rise sometimes to an extremely offensive odor. It occurs in all countries and among all classes, but is most frequently found in children or young adults, and according to Greville McDonald (Diseases of the Nose) is most common in girls. I have never observed it in children under eight years of age nor in adults over forty; most cases occur before the twenty-fifth year, very few being observed in patients more than thirty-five years of age.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—The nasal cavities are widened, even to two or three times their normal size, the turbinated bodies appear smaller than normal, and in advanced cases

they may have entirely disappeared. It is not unusual to find the inferior turbinated bodies much smaller than normal, while the middle turbinals are still hypertrophied. As a result of changes in the mucous membrane, involving its blood vessels and glands, the secretion becomes tenacious and of a muco-purulent character; and in consequence of the large size of the nasal cavities it is impossible for the patient to secure a sufficient blast of air for its expulsion; therefore it dries upon the surface, partially decomposes, and thus forms crusts which may completely block the cavities. These crusts are finally separated by the increased secretion beneath them and may then be expelled, but only to be soon replaced by others of the same character.

The pathology of the disease is still a mooted question, and it would be profitless for us to enter into the controversy. I favor the theory that in most cases the atrophy is the result of previous hypertrophy. The mucous membrane is usually anæmic, but seldom if ever ulcerated, excepting that in some instances abrasion of the septum may have been caused by picking the nose.

Etiology.—The cause cannot always be ascertained, but in some persons a history of frequent colds, with more or less complete obstruction of the nares for a considerable period, sometimes dating from an exanthematous fever, and at others from an injury, leads to the belief that the affection is usually preceded by chronic catarrhal inflammation, and favors the theory that atrophy results from an antecedent hypertrophy.

Symptomatology.—The patient is usually in good health at the beginning, but commonly the general condition suffers with the advance of the disease. Usually the nose is broad, the alæ thick, the lips thickened and prominent, and the whole physiognomy is lacking in expression, as is often seen in the strumous diathesis. The eyes are often affected, the sense of smell is usually lost, and partial deafness commonly exists. The secretion, which is of a muco-purulent character is tenacious, and usually there is but little discharge from the nose except at intervals of once or twice a week, when the crusts formed by drying of the secretion are expelled. The breath has an exceedingly offensive odor caused by decomposition of the retained secretion. So great indeed is this that it will often speedily permeate a whole room, though, perhaps fortunately for the patient the sense of smell is usually lost, so that he is spared much personal discomfort. The foulness of this indescribable odor is only second to that of syphilitic necrosis of the nasal bones, and is so peculiar that, when once detected, it becomes a valuable diagnostic symptom.

Upon inspection of the nares, we are at once impressed with the abnormal size of the cavities, unless they be choked by dried secretions. When the crusts are removed, we observe the small size, or absence, of some or all of the turbinated bodies, with perhaps hypertrophy of others, and find that usually we may easily see the naso-

pharynx and often the orifice of the Eustachian tube through the nostril. The secretion which has remained longest in the nose is of a brownish or blackish color; that less old, of a yellowish or greenish hue. In most cases where crusts are found upon the surface, atrophy of the mucous membrane is very apparent, and the odor is offensive. In some cases the secretion is thin, of a purulent character, and may be easily washed away, even though the patient cannot expel it by blowing the nose. Immediately after washing the nares the mucous membrane may appear redder than normal, as the result of the cleansing process, though it is commonly anæmic.

Diagnosis.—The affection is liable to be mistaken for lupus, syphilitic disease of the nose, suppuration of the accessory cavities, and rhinoliths or foreign bodies in the nose. There is usually no difficulty in distinguishing it from *lupus*, because of the external manifestations of the latter disease; but in *lupus vulgaris*, crusts and scabs similar to those found in atrophic rhinitis are formed; these are usually closely adherent to the septum instead of the turbinals; and unlike the crusts in atrophic rhinitis when removed, they leave an ulcerated surface which usually bleeds and is marked in one or more places by the typical lupus tubercle.

On account of the offensive odor, *syphilitic disease* of the nose is especially liable to be mistaken for atrophic rhinitis; but in syphilis, upon examination with a probe, dead bone is often detected, and upon cleansing the part, ulceration or perforation of the septum or hard palate is apt to be found; at the same time there may be falling in of the bridge of the nose, which does not occur in simple atrophy.

An offensive odor arises from *suppuration of the accessory cavities*, but unlike atrophic rhinitis this is almost always unilateral; the corresponding naris is not likely to be enlarged, and the sense of smell is seldom lost; therefore the patient can generally appreciate the odor sooner than those about him.

An offensive odor, with profuse discharge from one side, arises from *rhinoliths* or *foreign bodies* in the nose; but after the parts are cleansed, offending bodies may be readily detected by inspection or palpation with the probe.

Prognosis.—If left to itself, atrophic rhinitis continues for many years; but it is seldom observed after the thirty-fifth year. As the history shows that even with the most indifferent care most patients eventually get well, it is probable that there is a spontaneous tendency to recovery about middle life. Under appropriate treatment, most cases may be cured within from six to twenty-four months, if the patient will give it proper attention. In nearly every case the offensive odor may be speedily relieved, and it will not reappear if perfect cleanliness is observed. We cannot hope, however, to cure the anosmia, and the deafness associated with atrophic rhinitis is seldom remediable. Restoration of the atro-

phied structures can seldom be expected, though I have seen a few cases in which undoubted atrophy, with great enlargement of the nasal cavities, has so far disappeared as a result of treatment, that the nares became of normal size, and in one case even smaller than desirable. Therefore I agree with Moure, of Bordeaux, who holds out hope of regeneration of atrophied structures in some cases. Impairment of the general health resulting from constant inhalation of the fetid air from the nose, and probably from partial absorption of the secretion is speedily remedied as the local disease is relieved.

TREATMENT.—Judging from the great importance attached by various authors to special forms of local treatment it is probably of little consequence what remedies we employ, so that they be used in such manner as to keep the nares cleansed and disinfected, and the mucous membrane slightly stimulated. Cleanliness must be insisted upon, otherwise any form of treatment will be of little avail. It is maintained by some that this cleansing must be done by the physician, to which there is no objection, providing he has sufficient time and it does not entail too much expense upon the patient; but it is entirely unnecessary

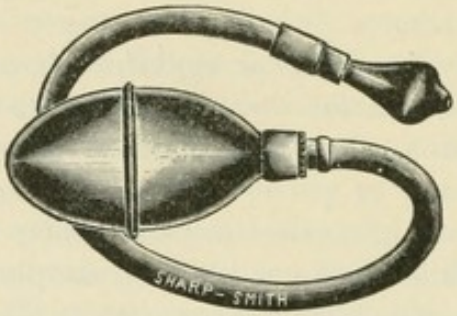

FIG. 204.—INGALS' NASAL SYRINGE (⅓ size).

for the physician to perform these ablutions if he will insist that the patient do it himself. The patient should be directed to wash the nose thoroughly two, three, or four times daily, using from half to one and a half pints of fluid each time, as may be found necessary to accomplish the object. In some cases it is sufficient for the patient to snuff fluid through the nose from the palm of the hand. In others it is better to use some form of nasal syringe (Fig. 204) or the nasal douche, though the latter should be avoided if possible, on account of the danger of causing deafness by forcing fluids through the Eustachian tubes to the middle ear. In using any form of nasal syringe or douche, but little force should be employed, the mouth should be kept open, and the patient must be careful not to swallow during the washing process. As a rule, the solution should be warm, though with some patients the stimulation of cold douches answers an excellent purpose. Pure water is sometimes sufficient, though usually it is better to use solutions of some of the sodium salts, of which sodium chloride or bicarbonate, or the salicylate mixture (Form. 187) may be employed in the proportion

of a heaping teaspoonful to a pint of luke-warm water. Sea salt may be used in place of the common article, but is no better. Carbolic acid, listerine, or other antiseptics in small quantity may be added to this solution if desired. After the part is thoroughly cleansed, various remedial agents may be employed, the object being to slightly stimulate the mucous membrane with the hope of improving its nutrition, increasing the glandular secretion, and preventing suppuration and decomposition. For the latter purpose iodoform is an excellent agent, though too offensive for use in private practice.

In hospital and dispensary work no remedy has given me more satisfaction in atrophic rhinitis than a powder consisting of equal parts of iodoform and boric acid, which is thrown freely into the nasal cavities two or three times a week, after the parts have been cleansed by the patient as directed. In private practice, europhen or iodol may take

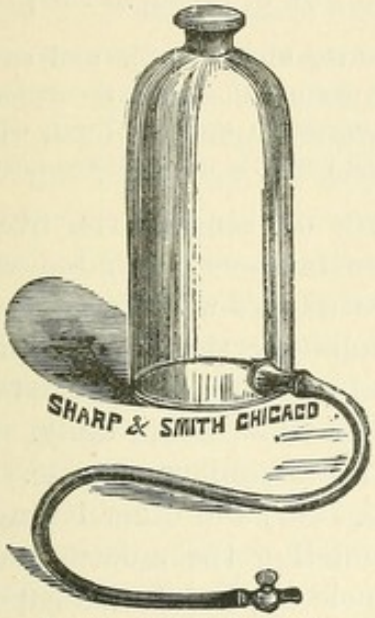

FIG. 205.—NASAL DOUCHE.

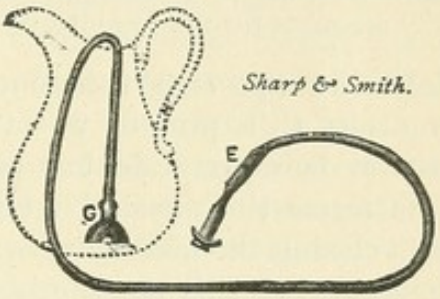

FIG. 206.—TRAVELERS' NASAL DOUCHE.

the place of iodoform. I use the latter much alone, and also variously combined with mercury bichloride, myrrh, gum benzoin, berberine, boric acid, aristol, and cocaine, with sugar of milk as a base (Form. 170 to 172 and 181).

Powders are used when there is free secretion, and sometimes, even though there is much dryness of the part, they have a most satisfactory effect, especially if associated with the oleaginous sprays of carbolic acid, menthol, oil of cloves, or other similar substances in liquid albolene; the rule being that whatever application is made should not cause the patient discomfort for more than five or ten minutes. The powders and sprays I generally give in the following strength, to be used by the patient two or three times daily: mercury bichloride, from one-tenth to one-fifth of one per cent; iodol, twenty-five per cent; boric acid, ten per cent; aristol, five to eight per cent; gum benzoin or myrrh, twenty per cent; berberine muriate, ten per cent; cocaine, two

or three per cent. The sprays contain of menthol one-tenth to one-fifth of one per cent, carbolic acid one-fifth of one per cent, oil of cloves one-half to one per cent (Form. 104 to 106). Ichthyol used as a spray in five per cent oily solution is reported to have given good results in these cases. Where the secretion is profuse and of a muco-purulent character, from one-eighth to one-half grain to the ounce of mercury bichloride in an aqueous solution is an excellent remedy. Similar applications should be made by the physician sufficiently strong to cause discomfort for half an hour. It is best for the patient at first to visit the physician once or twice a week, in order that he may be certain that the cleansing process is properly accomplished and that the applications are of proper strength, but after a short time twice a month is usually sufficient. In mild cases from one to two per cent of cocaine added to the powder which the patient uses at home has appeared to have a most beneficial action in stimulating the flow of blood to the parts.

The effects of cocaine in causing contraction of the blood vessels and cavernous tissue is well known; it is also true that if used continually for a considerable length of time, it frequently increases the congestion and swelling, which probably accounts for the benefit sometimes derived from its use in these cases.

McDonald (*op. cit.*) recommends tincture of sanguinaria, five to thirty drops to a pint of warm water; also tampons saturated with glycerin or boro-glyceride, but especially Gottstein's wool tampons, or what he terms the physical method of stimulating the circulation by partially closing the nostrils with cotton wool and causing the patient to inhale through this obstructing mass two or three hours daily. He also recommends a simple nasal respirator for a similar purpose. D. Bryson Delavan (*New York Medical Journal*, 1887) and other laryngologists report satisfactory results from stimulating the mucous membrane with the electric current, the positive pole applied to the nape of the neck, the negative to the mucous membrane by means of a piece of copper wire enclosed in a pledget of moistened cotton, with a current of from four to seven milliamperes. In addition to the local remedies, great benefit is often derived from constitutional treatment. Quinine, iron, strychnine, arsenious acid in some form, and iodine are most beneficial. The latter, in moderate doses just sufficient to excite nasal secretion, is frequently found most advantageous. Good diet and proper clothing should always be supplied, and a change of climate will sometimes be found beneficial.

CHAPTER XXXII.

DISEASES OF THE NASAL CAVITIES.—*Continued.*

HAY FEVER.

Synonyms.—Hay asthma, rose cold, June cold, autumnal catarrh, rhinitis hyperæsthetica, catarrhus æstivus.

Hay fever is one of the neuroses occurring periodically and characterized by irritation and inflammation of the mucous membrane of the eyes, nose and air passages, attended by profuse secretion and asthmatic attacks. Isolated cases may occur at any time of the year, but in this country the affection usually prevails from about the middle of August until the latter part of September, or until the early frosts; though a considerable number of cases are observed in May, June, and July, and occasional instances even in mid-winter. In England it is most prevalent in June and July. It is rather more common in men than in women. It occurs at all ages, but is most frequent before the prime of life; I have seen it in children five years of age, and have known it to afflict those as old as eighty or ninety. Seldom found among the working classes, it attacks preferably those of education and cultivation, and residents of towns and cities rather than dwellers in the open country.

Anatomical and Pathological Characteristics.—The inflammation generally affects the nasal mucous membrane and conjunctivæ, but often extends to the frontal sinuses, and may be severe in the fauces or entire respiratory tract. The membrane is usually highly congested and swollen, but in some cases, although swollen, it is much paler than normal. Though its pathology is not fully understood, the affection apparently results from a peculiar irritability of the nervous system, sometimes being manifested by constitutional symptoms and again by localized abnormal sensibility either in the whole or a part of the respiratory mucous membrane.

Etiology.—Heredity and nervous temperament predispose to this affection, but a great variety of substances may excite the attack where the predisposition exists. William H. Daly, first pointed out the relation between hay fever and certain morbid conditions in the nasal passages (Transactions of the American Laryngological Association, 1881). Subsequently his observations were repeated, and his conclusions confirmed, by Roe, Hack, J. N. Mackenzie, Sajous, and others; and although the disease is not so uniformly dependent upon the condi-

tion of the nasal mucous membrane as some of these authors supposed, yet in most cases such a relation is undoubted. Commonly the attack appears to be brought on by inhalation of the pollen of ambrosia artemisiæ folia, known also as Roman wormwood, rag-weed, or hog-weed, or that of solidago odora, known commonly as golden-rod, but it is frequently excited by dust and smoke, especially in railway travel, and by the emanations of roses and other fragrant plants, or the pollen of certain grasses, as wheat, barley, oats, rye, or even indian corn. It may also be excited by the dust of ipecac, salicylic acid, benzoic acid, and lycopodium, and sometimes it is brought on by exposure to heat or light, or by over-fatigue. So strong is the neurotic influence in this disease that imagined exposure to influences which had formerly excited an attack have been sufficient to induce the return of the paroxysm; for example, an artificial flower or even the painting of a full-blown rose has brought on an attack of the disease.

Symptomatology.—The attacks often come on the same date of succeeding years, regardless of the temperature, the conditions, or surroundings; but in some is a variation of a few days, apparently dependent upon atmospheric conditions or environment. There are two well-marked types, the *catarrhal* and the *asthmatic*. In the former the disease usually comes on suddenly, with irritation of the mucous membrane of the fauces, conjunctivæ, and nares, attended by frequent sneezing; in the latter, asthmatic features are usually developed after the nasal symptoms have existed two or three weeks, but they may come on independently. The asthma in this affection commonly differs from ordinary spasmodic asthma in that the paroxysms are likely to occur during the day-time.

In most instances the patient is made aware of the onset of the disease by a tickling or stinging sensation in the Schneiderian mucous membrane, accompanied by violent sneezing and itching of the conjunctivæ, with profuse lachrymation; or by burning or stinging sensations in the throat, or in some instances by severe neuralgic pains in the eyeballs or back part of the head. Swelling of the conjunctivæ, eyelids, lips, or tip of the nose is frequently present. Constitutional symptoms are often marked by elevation of temperature, aching of the muscles, general malaise, and sometimes great weakness. One of the most uniform concomitants is swelling of the Schneiderian mucous membrane, which causes obstruction of the nares, and thus interferes with respiration, in many cases leading to the asthmatic attacks. Profuse watery discharge from the nose, subsequently becoming mucopurulent, and which is often very irritating, is nearly always present. The mucous membranes affected are usually of a bright red color, though occasionally anæmic.

Diagnosis.—Hay fever may be confounded with simple acute rhinitis or spasmodic asthma. The essential points of difference are the his-

tory, the abrupt commencement, the excessive irritation; and the occurrence of asthmatic paroxysms during the day instead of at night. This history, together with the detection of very sensitive areas of the nasal mucous membrane by lightly touching it with the probe, are sufficient to establish the diagnosis, except during first attacks or in young children, where it is sometimes necessary to watch the patient for some time. Urticaria is frequently observed in connection with hay fever.

PROGNOSIS.—The attacks usually continue, with daily varying severity, from four to six or eight weeks, according to the patient's surroundings and the atmospheric conditions, and not infrequently the patient remains greatly debilitated for several months. The asthmatic attacks may continue several hours or two or three days, and then disappear as suddenly as they came. Some lose susceptibility to the disease with advancing years. The affection is not dangerous to life.

TREATMENT.—In most cases the attacks may be prevented by change of climate—sometimes a change from city to country or *vice versa* is sufficient—but most patients find the greatest relief in cool localities by the northern lakes, in places near the seashore, or at high altitudes; or from a lake or ocean trip, which removes them from the pollen-laden air. In this country, the most favored spots are in the White Mountains of New Hampshire, and in the region about Mackinac, in the northern part of Michigan. Many obtain complete immunity from the disease in the high altitudes of our western states and territories. No locality will be found equally beneficial for all individuals, and some will suffer severely where others have complete relief.

As the disease commonly occurs in neurasthenic persons, nerve tonics and sedatives are especially indicated. It is well to begin the administration of these remedies a month before the attack usually comes on, and to continue them until convalescence is established. To this end the various preparations of quinine, strychnine, or arsenious acid, and asafœtida or some of the preparations of valerian are most serviceable. I have found peculiarly beneficial a pill containing medium doses of brucia phosphate, alcoholic extract of hyoscyamus, quinine valerianate, and camphor monobromate, with or without small doses of sodium salicylate, phenacetin, acetanilid, or asafœtida. These may be given before and during the attack, with the effect of greatly mitigating the patient's sufferings. During the attack, opiates and belladonna in small doses are often of the greatest benefit; for example, five to eight drops of the tincture of belladonna or the deodorized tincture of opium, or both combined; or instead of these from a twelfth to an eighth of a grain of morphine, or from one two-hundredth to one one-hundred-and-twentieth of a grain of atropine, or both together. Atropine in small doses or hyoscyamus is especially beneficial in checking the profuse secretion and tendency to sneeze; the after effects of the latter are less likely to be unpleasant. Local stimulating inhalations, of ammonia,

iodine, or chloroform are sometimes useful, though they must be employed guardedly lest they increase the irritation.

For relief from the itching of the conjunctivæ, weak solutions of lead acetate are especially recommended by Mackenzie. I have found most beneficial a solution of sodium biborate gr. v. to x. ad ℥ i. of camphor water. With this, the eyes may be bathed as frequently as desired. The lips and nostrils may be protected from the irritating effect of the secretion by applying the ointment of zinc oxide, or better the iodol and lanolin ointment (Form. 9), to each ounce of which has been added ten or twenty grains of zinc oxide. The irritation of the nasal mucous membrane may sometimes be largely prevented by wearing plugs of wool in the nostrils to exclude dust and other irritating substances. Bathing the eyes and nose with either of the solutions recommended, or with very hot or very cold water, will sometimes give great relief.

As a local application to the Schneiderian mucous membrane, a spray of a saturated solution of boric acid will sometimes be found very grateful. In some instances it is well to make this solution in camphor water; in others it will be necessary to add to it small quantities of atropine, morphine, or cocaine. The latter remedy gives more immediate relief than any other we possess; but unfortunately its continued use is frequently followed by most serious consequences. With some patients, oily sprays will be found more beneficial. For this purpose a most excellent combination is that of thymol gr. ½, oil of cloves ♏ iij., and liquid albolene ℥ i., to which in some cases a small amount, not more than one-half of one per cent, of the alkaloid cocaine may be added. The strength of this solution may be slightly increased in some cases with advantage, but care should be taken not to make it irritating. A similar spray used five or six times a day will sometimes prevent the paroxysms of this disease. A powder containing three or four per cent of cocaine hydrochlorate (Form. 166) will be found more convenient for general application. In whatever way cocaine is employed, the patient should not use more than one-third of a grain daily, and this should not be long continued. Because of the temporary relief afforded, patients are very apt to use this remedy to excess, therefore physicians should never give written prescriptions containing it, and should insist upon knowing exactly how much the patient is using. I have known several lives wrecked by neglect of this precaution. During an acute attack of hay fever, nasal douches of weak solutions of quinine, salicylic acid, sulphurous acid or other antiseptics have been recommended on the theory that the irritation is due to the local action of microbes. These applications seem to have been beneficial in the hands of some physicians, but in my experience they have been disappointing.

When the attacks are due to sensitiveness of the nasal mucous membrane, the disease may be cured by judicious operative measures. These consist in removing any spur from the septum that may be large

enough to impinge upon the outer wall, the removal of polypi, linear cauterization along the turbinated body to prevent extreme swelling, and, most important, superficial cauterization of all spots found to be extremely sensitive. The superficial cauterizations should simply sear the mucous membrane, leaving it in much the same condition as the integument after a blister; it must not be burned so deeply as to cause any amount of cicatricial tissue. The linear cauterizations are the same as those recommended for hypertrophic rhinitis. The operations on the septum and for polypoid growths are described elsewhere.

The nasal cavity should first be thoroughly examined with a flat probe, the various parts being gently touched and the sensitive spots marked upon a diagram representing the two surfaces of the nares. A solution of cocaine (Form. 140) is then applied by means of a small pledget of absorbent cotton wound on the end of a flat nasal applicator (Fig. 197). The pledget saturated with the solution is carried back to the posterior part of the naris and as it is brought forward is rubbed gently over every part of the mucous membrane to be anæsthetized. This occupies about thirty seconds. A minute later the application is repeated with a fresh pledget. From two to four such applications are generally sufficient.

The cauterization may commonly be done without pain as soon as the patient ceases to feel the probe rubbed lightly over the surface, even though pressure may still be felt.

The part, having been thoroughly anæsthetized, should be sprayed with liquid albolene, and then rubbed over quickly two or three times with a flat, guarded electrode (1, Fig. 91) until a spot about a centimetre in diameter has been seared and appears of a white color. It should not be burned deeply enough to cause an appreciable scar after healing has taken place. The cauterized part should be noted upon the diagram, and after four or five days a similar cauterization may be made in some other part of the nasal cavities, preferably upon the opposite side. These operations should be repeated from time to time until the whole surface has been treated and no part remains peculiarly sensitive to the probe.

After the cauterization, the patient may be given a four per cent powder of cocaine, which may be insufflated into the nares once in three to five hours for the following three or four days. Together with this it is well to give an oily spray similar to that already recommended. These cauterizations may sometimes be repeated every two or three days; but it is generally better to make the intervals longer, otherwise the nares are apt to become quite sore, and the patient experiences much discomfort. When the longer interval is allowed, treatment may usually be conducted without in any way interfering with the patient's vocation, and without serious discomfort. From fifteen to thirty treatments are generally necessary to cover all of the diseased surface. The following year a few spots may be found still sensitive, which were

overlooked previously or not burned deeply enough; or possibly these may result from new development of the disease.

The treatment is best carried out during the warmer portions of the year, either before the usual time of the attack or after it has subsided; for during the attack it is liable greatly to increase the patient's distress. By this method from forty to fifty per cent of the cases of hay fever may be cured, about twenty-five per cent more may be greatly benefited, and the remainder will usually obtain sufficient relief from the nasal symptoms to compensate for the discomfort experienced during the treatment. The treatment is most apt to be beneficial where asthma has not yet developed, and where the general nervous symptoms are not pronounced. Cauterization of the surfaces with chromic or carbolic acid and other caustics has also been recommended. Asthmatic attacks occurring in connection with hay fever call for the same treatment as simple spasmodic asthma. It is always best for the patient to seek a different climate during the season if possible; and this is especially important in those who suffer from debility for several weeks or months after the attack, and in children, in whom we may hope to cure the disease by interrupting for two or three years the vicious habit of the nervous system, which otherwise might last a lifetime.

FURUNCULOSIS OF THE NOSE.

Furunculosis of the nose is a comparatively frequent affection, characterized by the development of small pustules or larger furuncles, the cavities of which vary in diameter from one to five millimetres or more. These suppurative points are attended by redness and great soreness of the end of the nose, and a larger furuncle by constant pain. The inflammation usually originates in the hair follicle. The affection lasts from three to seven days, and, upon discharge of the pus, healing quickly takes place. In many individuals the attack frequently recurs, and in some, one or more of these small abscesses are nearly always present.

Treatment.—As in all other abscesses, the indications are to evacuate the pus; but it is most important to adopt some measure which will prevent a recurrence of the attack. For this purpose remedies calculated to prevent the occurrence of suppuration in any part of the body are indicated, such as calcium sulphide, potassium chlorate, saline diuretics and laxatives; brewers' yeast has also been used for this purpose, with apparent success. Of the above, potassium chlorate has seemed to me most valuable. Local applications of tincture of iodine or solutions of silver nitrate and of various oils and ointments have been employed, with almost uniformly unsatisfactory results; for although the remedies appear beneficial at the time, the affection persistently recurs. It is true that in many cases any of these remedies may be used with appar-

ent benefit; but it is doubtful in such instances whether the patient would not have recovered almost as speedily without them. In obstinate examples the fact remains that local applications, as a rule, do but little good. In two or three cases, under a suggestion for which I am indebted to J. E. Best, of Arlington Heights, Ill., I have seen speedy improvement and permanent cure result from the use, four or five times daily for two or three weeks, of a two per cent aqueous solution of carbolic acid, which should be thoroughly applied with a small swab of absorbent cotton wound upon a toothpick or other applicator.

EPISTAXIS.

Synonyms.—Nose-bleeding, hemorrhagia narium.

Epistaxis consists of hemorrhage from the nose, originating either in the nasal cavities or the adjacent sinuses. It is most frequent about the age of puberty, is more common in early childhood and advanced age than in the prime of life, and occurs more often in men than in women.

Anatomical and Pathological Characteristics.—The mucous membrane may be congested and swollen, or may appear normal; but in most cases erosion, actual ulceration, or a small bleeding point may be found upon the cartilaginous septum. Sometimes the septum is perforated, and the bleeding comes from the edge of the opening. In other cases the mucous membrane is thin and the blood vessels are near the surface, so as to easily rupture upon engorgement from any cause. Occasionally the bleeding comes from the mucous membrane over the turbinated bodies, from the adjacent sinuses or posterior nares, or from the easily bleeding surface of a fibrous or malignant tumor.

Etiology.—Among the local causes are injury from picking the nose, the introduction of instruments, violent sneezing, coughing, straining, the inhalation of irritants, or the presence of polypi or other foreign bodies in the nasal passages. The constitutional causes are alterations of the blood, such as occur in anæmia, plethora, eruptive and relapsing fevers, diphtheria, scurvy, purpura, and hæmophilia; or changes in the walls of the blood vessels accompanying phosphorus poisoning, acute yellow atrophy of the liver, Bright's disease, gout, rheumatism, and occasionally syphilis or chronic alcoholism. The affection is also due in some instances to obstructed circulation through the jugular vein, engorgement of the right ventricle, obstructed pulmonary circulation as in severe bronchitis or emphysema, or to engorgement of the liver or kidneys; and it may result from the effects of strong emotional excitement upon the vasomotor nerves. It is sometimes vicarious, taking the place of menstruation or of the habitual bleeding from hemorrhoids.

Symptomatology.—In the plethoric, and in patients suffering from fever, the bleeding is often preceded by flushing of the face, a sense of

fulness in the head, with buzzing in the ears, and giddiness, and sometimes itching in the nose. It usually begins without apparent cause, frequently even while the patient is asleep, and flows from one side in drops, which follow each other in rapid succession; in severe cases it may run in a small stream. Usually not more than a drachm of blood is lost at one time, although it may seem very much more, to the patient and his friends; but in others, bleeding is rapid and persistent, and sometimes sufficient to prove fatal. A large amount of blood may be lost within a few hours, and the bleeding may continue for several days. Martineau mentions a case in which twelve pints of blood were lost in sixty hours (*L'Union Médicale*, 1868, troisième série, Tome VI). When the bleeding is excessive, syncope is liable to occur, and may prove fatal. Where epistaxis occurs frequently, or continues for several days, serious anæmia may result. Usually bright red blood flows from one nostril only, but it may pass back to the posterior nares and escape around the septum from the other nostril, or run down the throat.

Diagnosis.—Simple epistaxis may be confounded with certain neoplasms, or with ulceration, and can only be distinguished therefrom by careful inspection of the parts.

Prognosis.—Most cases terminate spontaneously within ten or fifteen minutes; but in some the bleeding continues several hours or even days. The cases occurring in children without apparent cause, and those resulting from various injuries to the nose, are seldom, if ever, dangerous. When occurring in old people without provocation, epistaxis indicates degenerative changes in the blood vessels, which are ominous. In subjects of hæmophilia, bleeding is liable to prove fatal. Nasal hemorrhages frequently recurring and lasting several days at a time, unless properly treated, cause dangerous anæmia, and many therefore terminate fatally. In low forms of fever, and in diphtheria, it is a grave symptom. As has been shown by Hughlings Jackson, this symptom occasionally precedes apoplexy (London Hospital Clinical Lectures and Reports, 1866, Vol. III); on the other hand, in malarial fever, in plethora, and in congestive conditions of the brain, the bleeding is sometimes beneficial. Instances are on record in which mania, epilepsy, and asthma seem to have been induced by checking the flow.

Treatment.—In the majority of cases the bleeding does no harm and need receive no treatment. When of a vicarious nature, and where there is evidence of plethora or of obstructed venous circulation, it should not be checked unless long continued. Owing to the fact that most cases stop spontaneously within ten or fifteen minutes, a great variety of methods for checking bleeding from the nose are implicitly relied on by the laity. To aid in checking hemorrhage, the head should be kept erect, applications of cold may be made to the neck or directly to the nose, or the application of hot water at a temperature of 120° to 125° F. As in most instances the blood flows from a small point on the

cartilaginous septum, it is easy to check it by continuous compression of the alæ nasi for ten or fifteen minutes or by direct pressure of the finger upon the septum. Compression of the facial artery is also recommended.

In continued bleeding which occurs from points far back in the nares, other methods must be employed. The insufflation of powdered alum, tannin, or matico leaves will often be found efficient. The alum is liable to cause excessive pain, and tannin also is frequently painful; powdered matico, however, has been found much less painful, and apparently is quite as effective. The application of a spray of tannin gr. x. ad ℥ i. answers well in some cases, or a solution of iron perchloride ♏ xx. ad ℥ i. may be used in the same way; of the two, the tannin is preferable. Injections of ice water, or better, small bits of ice frequently introduced, are often satisfactory. Internal remedies may be given at the same time with more or less benefit. For this purpose the fluid extract of ergot in doses of half a drachm every one to two hours, or ergotine in proportionate quantity, is recommended; also, tincture of opium in doses of from five to eight minims or medium doses of lead acetate, alone or combined with opium.

In the epistaxis of purpura, MacNamara commends a wineglassful of spirits of turpentine in a tumbler of brandy or whiskey punch taken as rapidly as possible (Mackenzie: "Diseases of the Nose and Throat," 1884). Harkin, of Belfast, Ireland, claims to have obtained excellent results (Transactions of the Ninth International Medical Congress, Vol. IV), in preventing the recurrence of epistaxis by counter-irritation over the liver. In persistent bleeding, when simple remedies fail, plugging must be resorted to.

Simple plugging of the nostril with cotton or lint, and holding the head forward until coagulation has taken place, will be sufficient in many cases. When it fails, plugging of the posterior nares must be the resort, or better still, filling the whole nasal cavity with a styptic and antiseptic tampon of gauze or lint. Sometimes the nares may be easily and effectually plugged by an air sack, operated on the plan of Barnes' uterine dilator, but this method is not usually very successful. Compressed sponge or simply strips of sponge may be packed into the nares with the forceps or applicator and will usually quickly check bleeding, but these are removed with difficulty, and occasionally some piece is left behind, causing an infinite amount of trouble, which might be avoided by carefully tying each bit of sponge with a strong thread, and numbering the threads by knots to indicate which should be removed first. One of the most convenient tampons for the nose is made by tying a strong thread to the middle of a bundle of fifteen or twenty ravellings from surgeon's lint, about four inches in length; one or more of these bundles being used. After the naris is filled, all of the threads may be wound about a bit of lint

and tucked into the nostril. This tampon has the merit of causing little pain and of being easily extracted, providing the threads have been numbered as already mentioned. In using any of these, it is well first to blow into the naris four or five grains of iodoform or of a mixture of equal parts of iodoform and boric acid.

A most efficacious method of checking excessive bleeding from the nose, which I adopted some years ago, and one easy of application, consists of saturating a strip of antiseptic gauze about an inch in width by four feet in length with a thick syrupy mixture of tannin in water, to which has been added a little glycerin and a few drops of carbolic acid. This is stuffed into the nose, fold after fold, until the naris is filled. Sometimes to the end first introduced, I attach three or four strong threads about two inches apart. This end is then passed through the naris into the naso-pharynx, the free ends of the thread being left hanging from the nostril. The strip is then rapidly pushed in until the posterior part of the cavity is full, after which the threads are drawn upon so as to pack the gauze firmly into the posterior naris. The whole cavity is then filled with the strip of gauze, any remaining portion being cut off. This is to me the most satisfactory means of plugging the naris, and has proved efficient in the most severe cases where posterior plugging would be indicated. The gauze may be rapidly and easily introduced, and readily removed, and the method obviates the danger of pressure upon the openings of the Eustachian tubes and consequent inflammation of the middle ear. The only disadvantages I have observed are that its removal is sometimes painful, especially after operative procedures in the nose, and the tannin causes some individuals considerable smarting. Walton Browne, of Belfast, Ireland, recommends a similar procedure, the gauze being impregnated with powdered alum instead of tannin, and he says it is not painful (Transactions of the Ninth International Medical Congress, Vol. IV), though from my observation alum appears to cause much more smarting than tannin.

Plugging the posterior nares has long been practised for checking obstinate epistaxis. It is commonly performed with the aid of Bellocq's canula, by drawing through the nose from the throat a strong string to which is attached a plug of cotton or lint of a sufficient size to fill the posterior naris. By traction on the string, this plug is firmly packed into the choana. A plug is then introduced into the nostril, and the string tied about it. Lint is much preferable to cotton for either of these plugs, as the latter tends constantly to become smaller when it becomes saturated with the secretions. A loop at least two inches in length should be left hanging from the plug that is drawn into the posterior naris, or a string should be attached and left protruding from the mouth to aid in removing the tampon. Both sides may be treated in the same way, but the impaction of a large mass into the naso-

pharynx is to be deprecated. It is unsafe to leave the post-nasal plug in position for more than twenty-four hours without renewal, as inflammation of the middle ear or suppuration of the mastoid cells is liable to follow such practice, and occasionally death from gangrene, tetanus, erysipelas, or septicæmia has resulted. To remove the tampon, the pledget should be taken from the nostril, and, when only one side has been stopped, warm water to which has been added a teaspoonful of sodium bicarbonate to each pint should be gently injected through the opposite side to loosen the tampon. The affected side may be carefully washed in the same way, but force should not be used. The string hanging in the pharynx or protruding from the mouth should then be pulled upon, and if necessary, the tampon gently pressed back by a probe until it is released and drawn out through the mouth. An ordinary soft catheter is often more convenient for introducing the string than the Bellocq's canula; it is passed through the nostril into the throat and drawn out at the mouth by forceps; a suitable thread is then attached and drawn back through the naris. A well waxed thread may usually be easily passed through the naris without the aid of catheter or sound.

To prevent recurrence of the attack, the cause must be sought and removed. In the majority of cases this will be found in a bleeding point upon the cartilaginous septum, but occasionally upon other portions of the mucous membrane. Sometimes cauterization of this with solid silver nitrate will be sufficient to cure; but usually it is best to touch it with the galvano-cautery, the point of which should be heated to a cherry-red and quickly touched to the spot several times, until the surface is thoroughly seared. In most cases a single treatment of this kind, provided the exact spot has been found, is sufficient to effect a cure, but in others subsequent cauterization will be necessary.

CHAPTER XXXIII.

DISEASES OF THE NASAL CAVITIES.—*Continued.*

NASAL MUCOUS POLYPI.

Synonym.—Nasal myxomata.

Nasal myxomata are tumors which grow from some part of the mucous surface, producing obstruction of the passages and usually excessive mucous discharge. They are very common, occurring more often in men than in women, but are seldom seen in children under twelve years of age.

Anatomical and Pathological Characteristics.—Mucous polypi are grayish or pinkish in color and semi-transparent; they are round, oval, or pyriform, and vary in size from five to fifty millimetres in diameter. They are somewhat yielding and elastic to the touch, their surface being smooth and often marked by minute blood vessels. They are commonly pedunculated, but sometimes sessile; they are generally multiple, and in about thirty per cent of all cases occur on both sides. Most of them spring from the middle meatus or the external surface of the middle turbinated body, a considerable number from the superior turbinated body and superior meatus, and not a few from the ethmoid cells. They occasionally start in the antrum or frontal sinus, and very rarely, spring from the septum. These tumors are usually covered with ciliated epithelium, beneath which are found a few dilated capillaries. Nerves have not been traced into these growths, but that they contain nervous filaments is demonstrated beyond peradventure by the pain caused by cutting them off. The bulk of the polypoid mass is made up of embryonic connective tissue and a gelatinous substance rich in mucin, the density of the growth depending on the degree in which the connective stroma or mucous substance predominates. Sometimes their structure is fibro-cellular.

Etiology.—Though their ultimate cause is not known, polypi are generally attributed to chronic congestion or to the irritation resulting from denuded bone. Woakes holds that mucous polypi are always associated with necrosis of the ethmoid bone (Nasal Polypi with Neuralgia, Hay Fever, etc., H. R. Lewis, London). While this may be an antecedent in many cases of polypi, either condition not infrequently occurs independent of the other.

Symptomatology.—At first the patient suffers from increased nasal

secretion and more or less occlusion of the nasal passages, which is often aggravated by damp weather, and is increased by colds, to which he is very susceptible. The occlusion is usually more marked in one naris, but the sense of obstruction frequently changes quickly from one side to the other. Nightmare, headache, giddiness, epilepsy, congestion of the fauces, hay fever, asthma, and other reflex disturbances sometimes result from the presence of these growths; but Mackenzie justly remarks (Diseases of the Throat and Nose):

> Whilst fully admitting that many reflex phenomena may arise from diseases within the nose, I must caution the younger specialist that the various complaints referred to as resulting from nasal disease are much more frequently due to other conditions, and that every other possible cause must be eliminated before the nose is incriminated.

Bosworth shows that mucous polypi are found in thirty-two per cent of all cases of asthma (Diseases of the Throat and Nose, 1889, Vol. I).

Patients often experience a sensation as of a movable foreign body in the nose; headaches are comparatively common, and the senses of smell and taste are often obtunded, although in many cases they may be restored by the removal of the growth. The voice is modified in a characteristic manner by the obstruction, and respiration is disturbed, so that the patient may be obliged to breathe entirely through the mouth. A profuse watery and sometimes muco-purulent, though not offensive, secretion from the nose is common. Epistaxis is not infrequent. When the tumor protrudes from the nostril, it is usually much congested. By anterior or posterior rhinoscopy the smooth, glistening, grayish or pinkish, growths may be seen; frequently only one or two large ones are visible, removal of which discloses many more of smaller size. A flat probe may be easily passed upon either side of the tumor, and to the touch it is found soft and elastic.

Diagnosis.—These polypi are to be distinguished from deviation of the septum, thickening of the turbinated bodies, chronic abscess of the septum, foreign bodies in the nose, and from fibrous, sarcomatous, and cancerous growths.

The polypi are readily distinguished from *deviation of the septum* by their semi-translucency and the fact that a probe may be passed between them and the septum.

They are distinguished from *thickening of the turbinated bodies* by their color, which is usually much lighter; by their density, which is much less; by passage of the probe between them and the external wall of the naris, and by their movability.

They are distinguished from *chronic abscess* of the septum by their color and density, by their presence usually in both nares, and by the passage of a probe between them and the septum.

Mucous polypi resemble *foreign bodies*, especially in causing obstruc-

tion and a profuse discharge, but the discharge in the case of foreign bodies is nearly always offensive—not so with mucous polypi. The history of the case, together with inspection and palpation of the nares, will establish the diagnosis.

Fibrous, sarcomatous, and *cancerous growths* in the nasal cavity are usually of deeper color, and more resistant to the touch, they bleed easily, and, the fibrous growths excepted, have a more irregular surface than polypi. The malignant tumors usually grow much more rapidly, often causing considerable pain, much disfigurement, and sooner or later grave constitutional symptoms. We would readily detect *cartilaginous* or *osseous tumors* by the sense of touch.

We frequently see hypertrophy of the mucous membrane associated with myxomata, but, on the other hand, the mucous polypi may cause atrophy of the soft tissues and sometimes even of the bony structures.

Prognosis.—The affection, if not relieved by operative procedure, usually continues for a lifetime, causing the patient much discomfort and annoyance. Although the obstructed respiration must eventually compromise the general health, the affection does not appear to threaten life. Often the tumors remain so small as not to attract the patient's attention, but when they have become large there is no reason to expect retrogression. Spontaneous expulsion of one or more polypi sometimes occurs. They are very liable to recur after removal, and are sometimes very difficult to eradicate. Rarely myxomata are transformed into sarcomata, and according to Schiffers, of Liège, such change occurs only in subjects past the fiftieth year (Transactions International Congress Laryngology and Otology, 1889).

Treatment.—For destruction of the growths the injection of various substances has been recommended, such as zinc chloride, iodine, alcohol, carbolic acid, and solution of iron perchloride; also local applications of saturated watery solutions of potassium bichromate. F. Donaldson, of Baltimore, has also recommended introduction into the tumor of chromic acid on a sharp pointed probe. While these methods have sometimes succeeded, they certainly generally fail, even in the hands of skilful operators.

Evulsion with the forceps, the oldest method, is still most commonly practised by general surgeons, though seldom employed by laryngologists. Sometimes, however, the polypus forceps will be found useful. As commonly performed by surgeons, this operation is very painful, there is much bleeding, often some of the turbinated bones are torn away at the same time, and rarely are the polypi completely removed. Some surgeons advise that the nose be laid open and the parts thoroughly curetted. This would evidently be more effectual than removal with forceps in the old way, but it cannot be more thorough than removal with the snare, followed by cauterization (or, if the operator prefer, curetting), when done under good rhinoscopic illumination, by

which every part can be seen quite as well as if the nose had been laid open. Sometimes polypi may be cut off with forceps or scissors. The galvano-cautery écraseur (Fig. 207) affords the advantage of searing the base and thus destroying it at the time when the tumor is cut off, but it is a clumsy instrument compared with the ordinary steel-wire snare which is the one now generally adopted by laryngologists. When polypi

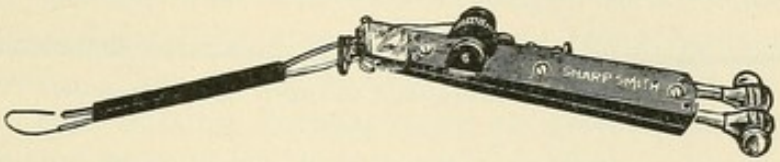

FIG. 207.—GALVANO-CAUTERY HANDLE, WITH ÉCRASEUR ATTACHMENT (¼ size).

bud again after removal, the best treatment is thorough searing with the galvano-cautery while they are still small. The operation which I have found most satisfactory for the majority of cases is done with the steel-wire écraseur or snare (Fig. 208). This is a modification of the snare devised by Clarence Blake, of Boston. Good instruments for the same purpose have been devised by Jarvis and Sajous, and various modifications of these have been made by other laryngologists.

The snare is armed with No. 5 steel piano wire, which in practice has been found to answer much better than other sizes. The loop is passed in vertically, its under edge turned beneath the polypus, and then with a backward and forward movement it is worked up as near the pedicle as possible. The loop is now tightened, and, if thought best, the polypus cut off at once, but usually better results are obtained if it is torn from its base by traction. There is little danger in this way of

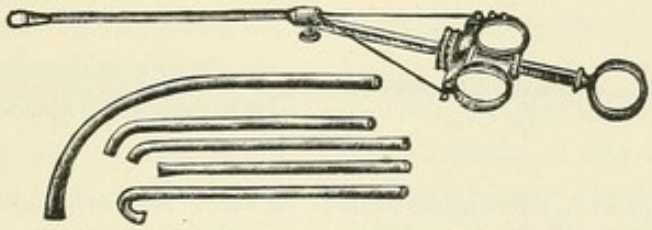

FIG. 208.—INGALS' SNARE, WITH EXTRA TUBES (¼ size, angle 25°).

removing any of the normal tissues, for it is almost impossible to include within the snare anything but the polypus. Where polypi grow from broad bases, and are attached over the whole surface of a turbinated body, the bone may be torn off with the snare if much traction is made. Under such circumstances the experienced operator, noticing the increased resistance of the normal tissue, instead of continuing the traction, will tighten the screw and cut the growth as near its base as possible. Where polypi grow from a large surface of the turbinated body, it is sometimes better to remove the bone to prevent recurrence.

The operator should have at hand forty or fifty applicators (Fig.

209), wound with absorbent cotton, for swabbing out the blood while the operation proceeds, as it is useless to try to catch the tumors when the nose is filled with blood. Whatever operation is performed, the parts should first be thoroughly anæsthetized with a four to ten per cent solution of cocaine, which is best applied by means of a hypodermic syringe fitted with a long, blunt silver nozzle (Fig. 210) bent at the end so that the solution may be thrown up about the base of the

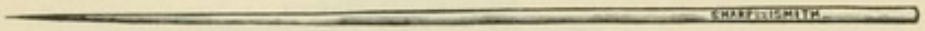

FIG. 209.—COTTON APPLICATOR (2-5 size). Made of copper.

tumors. Sometimes both cavities may be cleared at once, but it is usually preferable to remove what growths may be easily reached, and to complete the operation at one or two subsequent sittings, as this generally gives the patient much less discomfort than one long sitting. It will be remembered that the effects of cocaine disappear in about ten minutes, and after blood has once begun to flow it is difficult to anæsthetize the parts again; furthermore, if too much cocaine is used, its constitutional effects, even if not alarming, are extremely annoying. After the polypi have been removed, the patient should cleanse the nose once or twice daily with the salicylate wash (Form. 187), or with a wash of sodium bicarbonate, a teaspoonful to the pint of lukewarm water.

Antisepsis and healing will be promoted by insufflation two or three times daily of a powder containing twenty per cent of boric acid, fifty per cent of iodol, and sugar of milk sufficient to complete the mixture; together with the use of a spray containing about one minim of oil of wintergreen, two minims of carbolic acid, three minims of oil of cloves

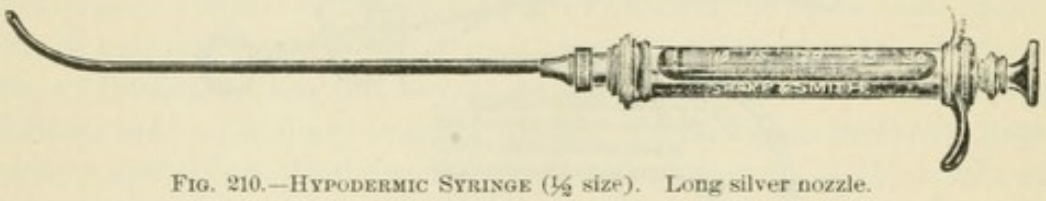

FIG. 210.—HYPODERMIC SYRINGE (½ size). Long silver nozzle.

to an ounce of liquid albolene. If secretion is profuse, ten minims of terebene may be added advantageously. The patient should return in about a week, when it will often be found that sacs which were invisible at the time of operation have filled, and may be removed. He should return again in four or six weeks, so that if the polypi are growing they may be thoroughly cauterized with the galvano-cautery. If the surgeon is not provided with this instrument, chromic acid may be used instead. In some cases mucous polypi do not return after thorough removal, but usually recurrence takes place, and operative procedures must be repeated from time to time until complete destruction of the growths is effected.

NASAL FIBROUS POLYPI.

Synonym.—Fibromata of the nares.

Fibrous polypi are extremely rare in the nares, although not uncommon in the naso-pharynx. Generally, growths in the nasal cavity which resemble fibrous tumors in appearance really occupy a histological position midway between mucous and fibrous polypi, termed fibro-mucous. These growths differ from mucous polypi in being harder and bleeding more easily. They should be removed, when possible, by the natural passages, with cutting forceps, snare, or galvano-cautery écraseur. The latter is best when it can be accurately applied.

NASAL PAPILLARY TUMORS.

Synonym.—Papillomata of the nares.

Nasal papillary tumors, though occurring more frequently than fibrous polypi, are still infrequent, though Hopmann states that small warty growths are more common than generally supposed, and he has met with numerous cases (Virchow's *Archiv*, Band XCIII, 1883). He also states that Schäffer, of Bremen, has observed them quite as frequently. This is different from the observations of Mackenzie, Zuckerkandl, and various other laryngologists, and from my own experience.

Anatomical and Pathological Characteristics.—The true papillary or warty growths are stated by Hopmann to spring invariably from the lower turbinated body, though I have seen one such tumor growing from the septum alone, and another instance in which several of these tumors grew from the septum while others came from the turbinated body directly opposite. They vary in size from two to fifteen millimetres in diameter. In five cases observed by Mackenzie, the tumors were situated on the septum or on the inner plate of the alar cartilage.

Symptomatology.—The symptoms which I have observed were those referable to dry catarrh, with the usual signs of obstruction of the nasal passage when the tumor was large. Hopmann also observed frequent cough and expectoration, which he attributed to the papillary growths.

Diagnosis.—The diagnosis must be based upon the peculiar appearance of the growths, which, unless they are moistened by secretion, is similar to that of warts upon the integument, and upon microscopic examination, which will determine their papillary character.

Prognosis.—The tumors tend to increase in number, and are very apt to recur when removed.

Treatment.—The growth may be destroyed with nitric, acetic, or chromic acid, the cutting forceps or curette, or the galvano-cautery. In one obstinate case under my care, all of these methods were tried

unsuccessfully; the warts repeatedly returned again in four to six weeks after each removal. Finally the patient was given a strong tincture of thuja occidentalis, which he applied to the part two or three times daily. This, with a few applications of chromic acid, finally eradicated the disease.

NASAL VASCULAR TUMORS.

Synonym.—Angiomata of the nose.

Vascular tumors in the nose are extremely rare. In their removal, Jarvis, who judges from his own experience and a tabulated report of sixteen cases by J. O. Roe, of Rochester (*New York Medical Journal*, January, 1886), considers the cold-wire snare safer, simpler, and more satisfactory than the galvano-cautery or other agents (*International Journal of Surgery and Antiseptics*, 1889). In one successful case reported by him, the gradual removal occupied three hours and there was no hemorrhage. Reasoning from analogy only, the galvano-cautery would appear to be the best instrument in such cases.

NASAL OSSEOUS CYSTS.

Osseous cysts of the middle turbinated body have recently been the subject of articles by H. Zwillinger, of Budapest, Charles H. Knight, of New York, and Greville Macdonald, of London.

This variety of tumor is rare, and its etiology, pathology, and symptomatology are not yet fully understood. Charles E. Sajous (*Annual of the Universal Medical Sciences*, 1892) quotes Macdonald as follows: "Whenever an osseous tumor presents itself in the middle meatus of such a size that it is obviously something further than a simple osteophytic periostitis, whether presenting an osseous surface covered only by mucous membrane or whether it is concealed partially or entirely by polypoid growths, the probability is strongly in favor of cyst. When, moreover, these appearances are accompanied by a purulent and fetid discharge, one may safely surmise that he is dealing with a suppurating cyst or abscess of the middle turbinate. The diagnosis is finally substantiated by the removal of a portion of the walls of the tumor by snare or forceps.

"The treatment is simple enough in cases when the tumor has not attained enormous dimensions. The simplest way of effecting removal is to throw a strong snare around the mass and remove as large a portion as possible. The remaining portion of the walls may afterward be broken away with forceps."

I have seen but a single case of the kind, which was easily removed with snare and forceps. The cyst was filled with a soft, yellowish cheesy mass.

NASAL CARTILAGINOUS TUMORS.

Synonym.—Ecchondromata of the nose.

True cartilaginous tumors in the nasal cavities are extremely rare, though a few cases have been reported. Ecchondroses or cartilaginous outgrowths, however, are very common, and will be considered elsewhere.

Anatomical and Pathological Characteristics.—Cartilaginous tumors closely resemble fibrous polypi; they are, however, sessile, generally grow from the cartilaginous septum, and if not interfered with may attain an enormous size, causing great deformity of the face.

Symptomatology.—The symptoms are those of nasal obstruction.

Diagnosis.—The cartilaginous growths, when large, are liable to be mistaken for fibrous polypi, malignant growths, exostoses, or osteomata. Practically we may exclude *fibromata*, because of their rarity. When present, they bleed more easily and are less dense than cartilaginous growths. It is to be observed that *malignant tumors* are softer, bleed easily, and grow rapidly. We readily distinguish *exostoses* and *ecchondroses* by inspection as being simple outgrowths. It is distinctive that *bony tumors* are harder and cannot be penetrated by the needle like cartilaginous growths.

Prognosis.—The prognosis is favorable if the disease is detected early, before great deformity has occurred. There is no tendency to recurrence when the tumor has been removed.

Treatment.—Removal by galvano-cautery écraseur is the most satisfactory surgical operation.

NASAL BONY TUMORS.

Synonym.—Osteomata of the nose.

Nasal bony tumors are usually characterized by obstruction of the nasal passage and severe neuralgic pains. When occurring, they usually develop about the age of puberty, but they are rare.

Anatomical and Pathological Characteristics.—Osteomata are usually ovoid in form, and they vary in diameter from five millimetres to five centimetres. They are distinctly bony formations, sometimes exceedingly dense, yet at others cancellous; but they have little or no connection with the osseous structure of the nose, and are generally attached to the soft tissues by a comparatively small pedicle. They are covered by periosteum and mucous membrane, which is freely supplied with blood vessels and of a pink or red color, and is occasionally ulcerated from pressure.

Etiology.—The etiology is unknown.

Symptomatology.—Early, the bony growth commonly causes intol-

erable itching of the nose, which is soon followed by symptoms of obstruction, with impairment of the sense of smell, and frequent epistaxis. As it begins to press upon the surrounding parts, neuralgic pains sometimes become extremely severe. In some instances, however, the nerves of sensation are paralyzed, and no suffering is experienced. As the growth enlarges, the nose may be distorted, the cheek may become prominent, and the eyeball crowded outward. In some cases continued pressure causes ulceration and finally perforation of the external parts. Such tumors are usually attended by an offensive discharge. By inspection the tumor may be seen. Its density or immovability can be ascertained with the needle or probe.

DIAGNOSIS.—The bony growths may be confounded with exostoses, rhinoliths, or cancer. They may be distinguished from *exostoses* at the outset by their movability, and later by their different form, larger size, and darker color. We can distinguish *rhinoliths* by an absence of mucous covering, and by the ease with which the surface is broken or crumbled by a strong nasal probe or forceps. It has been found that *cancerous tumors* grow much more rapidly and are usually very soft. In all cases they may be easily punctured by the needle. They, like osteomata, cause extreme pain and an offensive discharge.

PROGNOSIS.—If the tumor is seen early enough, it may be readily removed through the natural passages, but, when large, external incisions are necessary and scars remain, unless it can be destroyed by a dental burr. There is no tendency to recurrence.

TREATMENT.—The softer forms may be crushed with strong forceps and the fragments easily removed, but in the hard variety, which is most frequent, this is difficult, if not impossible. If not too large, they may be ground down or drilled through with dental burrs or trephines, and subsequently broken, but, if very large, an external incision is usually necessary for their removal.

NASAL MALIGNANT TUMORS.

Cancerous growths of the nose are characterized by rapid growth, obstruction of the nasal cavities, an offensive discharge, frequent epistaxis, and usually by severe pain.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—They commonly grow from the septum, but sometimes from the outer wall or floor of the nasal cavity. They are usually sarcomatous, but sometimes carcinomatous. They tend to increase rapidly in size, and soon encroach upon surrounding structures. They have a pale, slightly nodular or raspberry-like surface, are of soft consistence as a rule, and bleed freely when touched with the probe; their microscopic characteristics are the same as those of similar growths in other parts of the body.

ETIOLOGY.—The etiology is unknown.

SYMPTOMATOLOGY.—At first there are alteration of the voice, impairment of the sense of smell, and sensations of stuffiness in the nose common to all tumors in this locality. Other symptoms, however, rapidly develop. A greenish, offensive discharge is apt to soon occur, frequent epistaxis takes place, and great pain is often felt in the infra-orbital region. As the disease progresses, the bony structures are pushed in front of it or separated from each other, the eyeball protrudes, and the mass, perforating the base of the skull, may extend to the brain. Deafness, dysphagia, and dyspnœa are all symptoms which may occur in the progress of the case, and ere long constitutional symptoms appear indicated by loss of appetite, the development of fever, and a marked cachexia. Upon inspection, a tumor may be detected, usually of a light pink hue, but sometimes darker, even brown or black; highly vascular, bleeding easily when touched, and commonly soft and friable. Malignant growths ulcerate early; the ulcer presenting raised, ragged edges, and a sanious base.

DIAGNOSIS.—Malignant tumors of the nose are to be distinguished from rhinoliths, impacted foreign bodies, abscess, and benign growths. When the nasal cavity has been cleansed and well illuminated, we find the appearance of a *rhinolith* or *impacted foreign body*, and the sensation it communicates through the probe entirely different from that of a malignant tumor. An *abscess* may be developed rapidly or slowly, but it is almost universally located at the lower part of the septum, is apt to present upon both sides, is covered by normal mucous membrane, does not bleed, is elastic to the touch, and is not attended by the symptoms so commonly found in malignant growths. We may distinguish *benign tumors* by their color, density, slow growth, and other symptoms already described. In malignant growths, after a short time there is an enlargement of the lymphatics, especially those below the angle of the jaw. This does not occur with benign tumors.

PROGNOSIS.—The disease usually runs a rapid course, terminating within six or eight months in death. Sarcomata appear to have been eradicated in some cases where taken early, but carcinomata are always fatal.

TREATMENT.—Astringents and sedatives may be applied as palliative measures, but thorough eradication, when practicable, is the only treatment that affords any chance of success. Partial removal only aggravates the disease and causes its more rapid growth.

R. P. Lincoln reports a case of melano-sarcoma of the lower and middle turbinated bones and floor of the nostril which, returning after several operative procedures, was finally completely cured by the use of the galvano-cautery écraseur with cauterization at the site of removal (Transactions of the American Laryngological Association, 1885).

CHAPTER XXXIV.

DISEASES OF THE NASAL CAVITIES.—*Continued.*

SYPHILIS OF THE NOSE.

A LOCAL manifestation of constitutional syphilis in the nose may be primary, secondary, or tertiary, and may be congenital or acquired. It is characterized in mild cases by simple obstruction of the nares, and in the more severe by extensive ulceration and necrosis of the bones and cartilages.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—The mucous membrane may be thickened in patches or may be ulcerated. Condylomata are sometimes observed, and if the perichondrium or periosteum beneath the thickened patches becomes the seat of suppuration, death of the cartilage or bone is the natural result. This necrosis may also follow extension of the ulceration from the surface. Sometimes the process is one of gradual molecular destruction or slow caries, entirely escaping observation during life. In such cases the bone, gradually devitalized and absorbed, is replaced by exuberant granulations.

ETIOLOGY.—The sole cause is the syphilitic virus, but the severity of the disease often appears to depend upon individual constitutional peculiarities other than syphilitic. According to Mackenzie, the strumous diathesis seems to render the subject particularly liable to severe forms of nasal syphilis; and in countries where the disease is imperfectly treated, as, for example, in Egypt and Mexico, it becomes virulent. Primary syphilis of the nose is very rare. The secondary form is not infrequent in infants, in whom it is usually developed about the third or fourth month; but it is generally overlooked, and passes for what the nurse terms snuffles. Tertiary manifestations are seldom noticed until several years after the initial lesion; but the symptoms are sometimes developed between the sixth and twelfth month, and it is stated that among the modern Arabs, where syphilis is peculiarly severe, the tertiary symptoms appear much earlier.

In the secondary stage of the disease, the congestion of the mucous membrane causes profuse muco-purulent secretion and more or less obstruction of the nares. Mucous patches may occasionally be observed at the angle of the nostrils or upon the anterior portion of the mucous membrane. Evidences of the disease in the mouth and throat and upon the skin are usually present at the same time. In the tertiary stage,

there occurs necrosis of the cartilaginous or bony septum or of the turbinated bodies, accompanied by a most offensive odor of decaying tissue.

Extensive destruction of the nasal bones causes falling in of the bridge of the nose, and the oral cavity may be entered by perforation of the palate. Deep, foul ulcers, with ragged edges and a dirty, gray base, are usually present. Before extensive destruction has taken place, the turbinated bodies are often so swollen as nearly or quite to occlude the nares. The dead bone usually presents a blackish, uneven surface, though in some instances nothing can be seen except an offensive crust of dried and decaying secretion, which must be thoroughly washed away before satisfactory examination can be made; it can sometimes be detected with a probe, by the rough, grating sensation which it communicates; occasionally the lesions are so situated that they cannot be discovered. In rare instances an offensive odor is constantly exhaled, even though the parts are apparently kept perfectly cleansed by frequent ablutions.

DIAGNOSIS.—The secondary stage of the disease is not common, and, when it does occur, is very apt to escape observation. It can be distinguished from *chronic rhinitis* by the history of its sudden onset with very pronounced symptoms; by its very obstinate course; by the discovery of mucous patches or condylomata when these exist; and by the acknowledgment of infection when this can be obtained from the patient. The tertiary affection may be confounded with lupus or simple atrophic rhinitis. We can distinguish *lupus* from syphilis by its occurring at an earlier age than any form of syphilis except the hereditary. Again, in the beginning, the peculiar reddish papules or tubercles of lupus are quite distinct from any syphilitic manifestations; and, later, the marked preference which lupus shows for the cartilage is characteristic.

The offensive odor caused by *atrophic rhinitis* is quite different from the stench of tertiary syphilis. Upon cleansing the parts carefully, no necrosed tissue will be found in ozæna, whereas it is very apt to be present in syphilis. In all doubtful cases, the history, the presence of old cicatrices, or induration of the tongue, pharynx, or larynx, or brownish scars upon the skin or periosteal nodes, and finally the beneficial action of potassium iodide usually enable us to make a diagnosis of syphilis.

PROGNOSIS.—Syphilitic coryza in the adult usually terminates within two or three weeks. Secondary symptoms and those of the tertiary stage in mild cases, as a rule, speedily disappear under proper antisyphilitic treatment. When caries has taken place, and is still progressing, the prognosis is much less favorable, especially in debilitated subjects, in whom even life may be endangered.

TREATMENT.—Syphilitic coryza requires no other treatment than the internal administration of tonics, and the local use of mild alkaline

sprays or washes. Indeed, any secondary symptoms usually require only mild constitutional treatment, and touching of the condylomatous growths or mucous patches with tincture of iodine or silver nitrate. Tertiary syphilis, however, demands active constitutional and local treatment. It is well to begin with potassium iodide in moderate quantity, and steadily increase the doses until the reparative process is well established. To this end, not infrequently the drug must be pushed to its physiological limit. In all cases it or other specific medication should be continued in larger or smaller doses until a complete cure is effected. Small doses of mercury, or of gold and sodium chloride, will sometimes be found especially beneficial. At the same time, bitter or ferruginous tonics are often demanded, and cod-liver oil when well borne is useful. Good nutritious diet should be provided. Local treatment is extremely important. The nose should be thoroughly cleansed two or three times daily with the sodium salicylate wash (Form. 187) or a similar alkaline

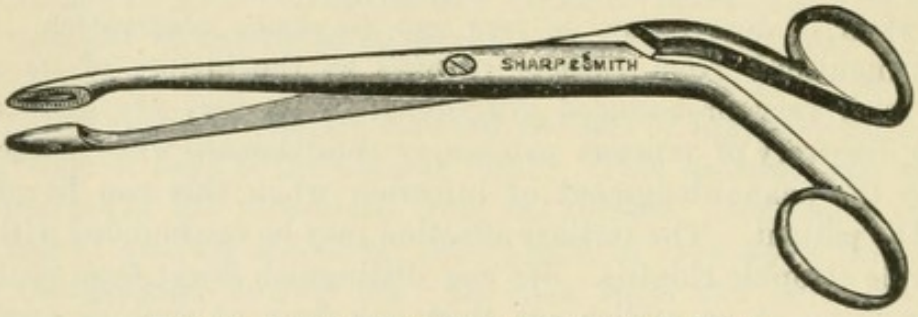

Fig. 211.—Ingals' Nasal Dressing-Forceps (3-5 size).

solution. Under this treatment superficial ulcers usually speedily heal; but where deep ulceration exists, in addition to cleansing, the sores must be touched with some stimulant or caustic. For this purpose the most commonly employed caustic is silver nitrate fused upon the end of an aluminium or silver applicator, but in the majority of cases I prefer strong tincture of iodine to any other local remedy. The applications should be made daily for ten or fourteen days, until evidence of cicatrization appears, and then every other day for a week or more, and subsequently less often. Even large ulcers under this treatment usually heal within three or four weeks. If dead bone is present, it must be carefully removed with forceps (Fig. 211), though it is unsafe to use much force. In the mean time the patient may advantageously insufflate into the nasal cavity twice daily a powder consisting of one part boric acid and two parts iodol or iodoform; or with this, in case there is much swelling, may be combined two or three per cent of cocaine, and five per cent of aristol to correct the offensive odor. Schuster specially recommends scraping the ulcers with a sharp spoon, and afterward destroying any indurated tissue that may remain with the galvano-cautery or silver nitrate (*Vierteljahresschrift für Dermatologie u. Syphilis*, 1877).

When the disease has been checked, if serious deformity exists, it may sometimes be remedied by an artificial nose, or in some cases by rhinoplastic operations, which are described in the textbooks of surgery.

CONGENITAL SYPHILIS OF THE NOSE.

Hereditary syphilis usually makes its appearance in children within the first two or three weeks after birth, and seldom later than the second month; but occasionally not until the child is eight or ten years of age, or at a later period, about puberty.

ETIOLOGY.—The disease appears to be contracted, in many instances, at the time of birth, though commonly during intra-uterine life.

SYMPTOMATOLOGY.—Usually within a week or two after birth the child appears to have a bad cold in the head, the nares are stopped, and there appears a thin, irritating discharge, which soon becomes muco-purulent, causing redness, soreness, and erosion of the nostrils and upper lip. The child is said to have the snuffles. As the secretions become thicker, the nasal cavity is blocked with scabs, which exhale an offensive odor. In some instances caries of the cartilages and bones ensues, not infrequently causing disfigurement for life. Such children are usually small and feeble, suffer from marasmus, and frequently have a copper-colored, papular eruption upon the skin. Mucous patches are probably present in the nose in most cases, but it is hard to get a view of them; similar patches may often be found at the anus or at the angles of the mouth or eyelids.

DIAGNOSIS.—The diagnosis must depend upon the history, the symptoms, the obstinacy of the disease, and the effects of treatment.

PROGNOSIS.—The affection runs a chronic course, with little or no tendency to spontaneous recovery. Such children often die young; but under judicious treatment some may be apparently cured. In a considerable number the disorder may be checked, but it continues to reappear at intervals for many years.

TREATMENT.—Mercurials and potassium iodide are indicated internally, and local treatment is generally desirable, though in young children it is very difficult to carry out. Mackenzie prefers mercury with chalk, which he administers in doses of from one to two grains twice daily, to which he adds, if this causes diarrhœa, one grain of Dover's powder or an additional grain of chalk (Diseases of the Throat and Nose, Vol. II). Erichsen recommends the external application of mercury in the following manner proposed by Brodie (Science and Art of Surgery, London, 1872): a drachm of mercurial ointment is spread upon a flannel roller which is stretched around the child's thigh just above the knee, the ointment next to the skin. This is renewed daily for two or three weeks, after which potassium iodide is administered in milk, cod-liver oil, or malt. Milk and water are the best vehicles for the administration of the drug to either children or adults.

TUBERCULOSIS OF THE NARES.

Tuberculosis of the nares is a rare affection characterized by the formation of tubercles of varying size, with ulceration and a fetid discharge. It is usually secondary, though Tornwaldt has reported a case in which the nasal symptoms preceded any other; and I have seen one case in which no evidence of pulmonary lesion could be discovered for several months after the appearance of the tubercular ulcer in the nostril. Of thirty-eight cases of nasal tuberculosis collected by Michelson, of Königsberg, nineteen showed no tuberculosis of any other organ (*Internationale klinische Rundschau*, Vienna, 1889), and F. Hahn reports five primary cases (*Deutsche medicinische Wochenschrift*, Leipsic, 1889).

Anatomical and Pathological Characteristics.—The tubercular deposit may be observed either as thickening, with or without ulceration of the mucous membrane, or in the form of tumors varying from two to thirty millimetres in diameter. The disease may attack any part, but most frequently the septum is the seat of the trouble. The nodules are generally small and of a grayish white color; the ulcers, which may be single or multiple, have a grayish base and frequently raised edges.

Etiology.—The bacillus tuberculosis is now generally accepted as the ultimate cause of the disease.

Symptomatology.—The affection comes on insidiously, and generally progresses slowly, causing all the symptoms of offensive catarrh. Tubercles or ulcers, as already described, may be detected by careful inspection. The ulcers are not generally painful and at first are not accompanied by constitutional symptoms; but sooner or later tuberculosis of the lungs or larynx is developed, and runs its ordinary course.

Diagnosis.—Tuberculosis may always be suspected when obstinate ulcers or tubercles are detected in the nose, especially in scrofulous patients, or those with recognized tuberculosis of other organs providing syphilis has been carefully excluded. An accurate diagnosis can only be made by finding tuberculosis in other parts or by the detection of the bacillus tuberculosis in the discharges or scrapings from the ulcers.

Prognosis.—The progress of the disease is generally slow, and may extend over many years; but it usually continues until other organs finally become involved, and then runs a more rapid course to a fatal termination.

Treatment.—The nares should be kept clean. Tumors which by their size interfere with respiration should be removed, and ulcers should be thoroughly treated with lactic acid, in strength varying from thirty to one hundred per cent, with or without previous scraping, according to the indications. Treatment of the ulcerated surface by carefully touching it from time to time with the galvano-cautery has been recom-

mended, and is advantageous in some cases. Insufflations of iodol or iodoform are also indicated; but whatever method is adopted, the ulcers are very difficult to heal, and in many cases the treatment does no appreciable good. When pain is present, soothing remedies are required. Of prime importance are all those means by which the system may be fortified against the spread of the disease. It would appear that these cases, if any, might be cured by the use of Koch's tuberculin; but in a single case of the kind in which I administered it, the results were most disastrous, and the progress of the disease was very much accelerated by the presumed remedy.

EMPYEMA OF THE ANTRUM.

Empyema of the antrum, which was accurately described by John Hunter, consists of a collection of pus in the antrum of Highmore, characterized by a purulent discharge having an offensive odor, usually

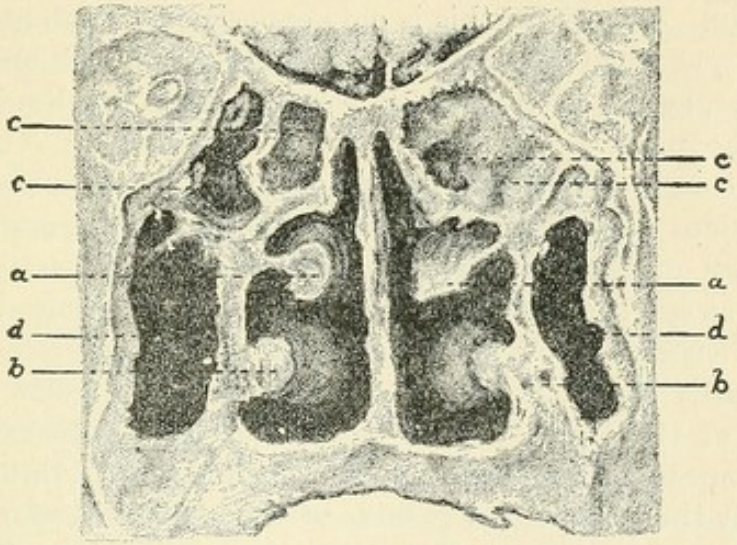

FIG. 212.—CROSS SECTION OF HEAD LOOKING FROM BEHIND FORWARD ABOUT HALF AN INCH IN FRONT OF THE OPENING OF THE NARES INTO THE NASO-PHARYNX. From a photograph of a frozen section prepared by C. H. Stowell, of Washington (4-5 natural size). *a,a*, Middle turbinated bodies; *b,b*, inferior turbinated bodies; *c,c,c,c*, ethmoid cells; *d,d*, antra of Highmore.

from one nostril. It is more commonly found upon the left side, but frequently upon the right, and occasionally on both sides. The antrum, as shown by Giraldes, is sometimes divided by septa of bone, so that in this disease two or more pockets of pus may exist (Des Maladies du Sinus Maxillaire, Paris, 1857). Delavan, in a paper read before the American Medical Association, Section of Laryngology, in 1889, showed that the antra are liable to various irregularities in formation, which accounts for some of the peculiarities presented in the symptoms and signs of the disease. The relations of these cavities to the nares and surrounding parts are accurately shown in Fig. 79, and Fig. 212.

ETIOLOGY.—Disease of the teeth is the principal cause of the affection; but in many instances it originates in morbid changes in the nasal

cavity or adjoining sinuses, such as caries, polypi or granulation tissue in the middle meatus, or suppurative inflammation of the ethmoid cells or middle meatus, the pus from which enters the antrum.

Symptomatology.—The affection usually comes on insidiously and lasts for several months, or possibly years, before it is detected. When it has existed for some time, there may be found considerable disturbance of the general health. In most cases, pain in the cheek is complained of, sometimes radiating toward the ear and frequently attended by supra-orbital neuralgia. But comparatively few of the patients suffer from toothache or swelling of the face, the most common subjective symptoms being more or less obstruction of the nose, a foul smell or taste seemingly from the throat, and discharge from one nostril. The fetor is often appreciated only by the patient himself, and is present in many instances only at certain hours of the day. The discharge also is usually periodical, occurring in considerable quantities two or three times a day, though in many instances there is a continual but slight flux. Sometimes this is only experienced upon assuming certain positions, as when lying upon the affected side, or even upon the sound side, or, again, upon bending forward with the head low down. Sometimes the principal flow is into the naso-pharynx, where it may excite reflex cough, or even nausea and vomiting. Upon inspecting the nares, a purulent discharge is generally observed in the middle meatus, trickling down over the inferior turbinated body. Oftentimes this, on being wiped away, speedily reappears. Polypi or granulation tissue may be seen in a large percentage of cases, and with the probe caries may not infrequently be detected. By tapping over the malar prominence with the tip of the finger, pain or tenderness is usually caused, which is not experienced on the sound side. McBride, of Edinburgh, notes that generally there is marked redness of the gum corresponding to the diseased antrum (*Edinburgh Medical Journal*, April, 1888).

Diagnosis.—The essential points in the diagnosis are the pain, fetor, and discharge from one naris. The affection is liable to be mistaken for disease of the frontal sinus or of the anterior ethmoid cells, or for polypus, ozæna, foreign bodies, syphilis, caries, or disease of the sphenoidal sinus. A useful method of detecting pus in this locality consists of injecting, through the normal opening in the middle meatus, a solution of hydrogen peroxide, which, in case pus is present, will immediately cause a discharge of froth through the opening. Transillumination, as suggested by Voltolini, is often, though not universally, of great value in deciding obscure cases. It is practised by means of a small electric lamp placed in the mouth while the patient is in a dark room. The effect of this is to cause a rosy-red suffusion of the face, cheeks, lips, and inferior eyelid in health, but the cheek and inferior eyelid will remain dark in case the antrum is filled with pus. A three candle power lamp, five to eight volts according to the strength of the

battery used, is best for this purpose. It may be attached to some form of tongue depressor. That shown in Fig. 213, which is inserted into the ordinary galvano-cautery handle, I have found most convenient. The patient may be examined in a dark room, or more easily with the aid of an ordinary photographer's focusing-cloth thrown over the heads of both patient and physician. This method is of peculiar value in detecting cysts of the antrum, which are said to render the illumination even more brilliant than in health, while solid tumors or pus prevent the transmission of light.

Empyema of the frontal sinus, unattended by closure of the duct, is so extremely rare that it may be excluded; when the duct is occluded the external signs are so marked that the affection cannot be mistaken for disease of the antrum.

We frequently find *suppuration of the anterior ethmoid cells* associated with empyema of the antrum; but when occurring by itself it is distinguished from the latter by the position of the pus above instead of below the middle turbinated body, and by the absence of positive signs in the antrum. McDonald recommends as a means of diagnosis the introduction into the antrum, immediately above the inferior turbinated bone,

FIG. 213.—INGALS' ELECTRIC LAMP (½ size). For trans-illumination.

of a strong, curved, hollow needle, to which is attached a small exhaust syringe (Diseases of the Nose, 1890).

Empyema is distinguished from *polypus* by inspection of the nares, but it must be remembered that before any operation has been done, whenever polypi are attended with purulent secretion, pus will usually be found in the antrum at the same time.

An extremely fetid breath, which is appreciated by every one except the patient, is continuously caused by *ozæna.* The fetor in empyema of the antrum is usually noticed only by the patient, and is apt to be intermittent in its occurrence. Inspection of the nares in these cases will readily determine the diagnosis.

An offensive discharge from one nostril may arise from *foreign bodies in the nose,* but they may be easily distinguished from disease of the antrum by inspection, and palpation with the probe.

An offensive odor and excessive discharge from the nares may be caused by *syphilis,* but it nearly always affects both sides, and inspection reveals ulceration, dead bone, or other evidence of disease of the cavity itself, instead of the comparatively healthy appearance found in empyema of the antrum. Caries is also usually detected in syphilis by inspection, and palpation with the probe.

Disease of the sphenoidal sinus is very rare, and when it does occur

the discharge flows into the throat, but not from the nostrils. It would not cause pain in the cheek or interference with the transmission of light; therefore, it may readily be excluded.

Prognosis.—Acute cases sometimes recover spontaneously within a short time, but the affection may extend over many years unless appropriate treatment is adopted. Even under the most approved methods, with free drainage, it is sometimes impossible to check the formation of pus.

Treatment.—Some cases have been cured by washing out the antrum through the natural opening with detergent solutions or with hydrogen peroxide, but usually free drainage must be established. For this purpose, Hunter's method of opening the antrum through the socket of one of the molars is still considered best, the only objection urged against it being the annoyance caused the patient by the offensive discharge into the mouth, and the possibility that particles of food may escape into the antrum. Christopher Heath recommends puncture of the antrum above the alveolus (Transactions Odontological Society, November, 1889). The main objection to this is the difficulty of keeping the opening patent. The antrum may be opened through the

Fig. 214.—Brainard's Bone Drill.

inferior meatus by means of trephine, drill, knife, or a long, curved, strong trocar, as recommended by Krause (*Berliner klinische Wochenschrift*, 1889). The latter position obviates the objection to Hunter's method, but the opening is less easy of access, and is more difficult to maintain until healing has occurred.

My own preference is for Hunter's method, a tooth or a root being extracted when necessary, or an opening being made through the space left by a tooth which has been already lost. Various forms of trephines, drills and dental burrs have been used for making the opening, but in most instances too small an instrument is employed. I use Brainard's conical bone-drill (Fig. 214), which makes an opening nearly a quarter of an inch in diameter. Notwithstanding statements to the contrary, the operation is extremely painful unless an anæsthetic has been used. General anæsthesia may be induced by chloroform, ether, or nitrous oxide gas—the effects of the latter are usually too evanescent—but in most instances the parts may be sufficiently benumbed by injecting into the gum, in two or three places on each side of the alveolus, a solution of cocaine, already recommended (Form. 143). The opening having been made, the antrum should be washed out and a gold or rubber tube introduced to maintain its patency. If this precaution is neglected, the opening is almost sure to close before the disease has been cured. Any good dentist can make a suitable gold tube which can be

fastened with clamps to the adjoining teeth. I have recently used with great satisfaction rubber tubes (Fig. 215) of six millimetres diameter, nineteen to thirty-five millimetres length, and four millimetres calibre, with flanges at each end. With a wire, the end of which has been bent to a right angle, the distance through the alveolus may be measured and a tube of proper length selected. The flange at the upper end of the tube is thinned, by cutting away its upper surface, until it may be squeezed into a gelatin capsule of proper size. This is then oiled and readily passed through the opening into the antrum. A probe is then

FIG. 215.—INGALS' DRAINAGE TUBE FOR ANTRUM. Full diameter; three different lengths.

passed through the tube, the gelatin capsule forced off, the flange opens out, and the tube is thoroughly secure. These tubes are inexpensive and very much more comfortable to the patient than gold. The subsequent treatment consists of keeping the cavity clean, and stimulating the healing process by injections of iodine, zinc, copper, or hydrogen peroxide in watery solution; or by insufflations of boric acid, iodol, iodoform, or aristol; or by solutions, in liquid albolene, of carbolic acid, oil of cloves, oil of cinnamon, or terebene. If septa prevent thorough cleansing of the cavity, it may be necessary to enlarge the opening and break them down. The patient should always stop the opening with a pledget of cotton while eating.

EMPYEMA OF THE SPHENOIDAL SINUSES.

Empyema of the sphenoidal sinuses is so extremely rare that no definite rules for diagnosis or treatment can be formulated. These sinuses, which occupy a position at the upper back part of the nasal cavity, just at its opening into the naso-pharynx, vary in number, size, and form in different individuals (Fig. 216).

SYMPTOMATOLOGY.—Purulent inflammation of these cavities gives rise to a persistent discharge of pus into the nares and naso-pharynx, and not infrequently causes severe headache, with more or less disturbance of the senses of smell and sight.

The anterior wall of the sphenoidal sinus, as shown in Fig. 216, is thin, and in cases of long-continued empyema a spontaneous opening through it might be effected. The finding of pus uniformly in this position, or trickling from it down the sides into the posterior nares, may suggest the true nature of the disease.

TREATMENT.—Other affections being excluded, and the diagnosis established, the anterior wall of the sinus should be carefully perforated, and the cavity drained and treated on the same principles as empyema

of the antrum. Opening has also been successfully effected through the inner wall of the orbit in extreme cases.

INFLAMMATION OF THE FRONTAL SINUS.

Inflammation of the frontal sinus is a comparatively frequent affection, but owing to the dependent position of the duct in most cases the products of inflammation readily escape and spontaneous recovery speedily follows. Sometimes, however, swelling obstructs the duct, and the secretions may be pent up. Such cases I have seen readily relieved by

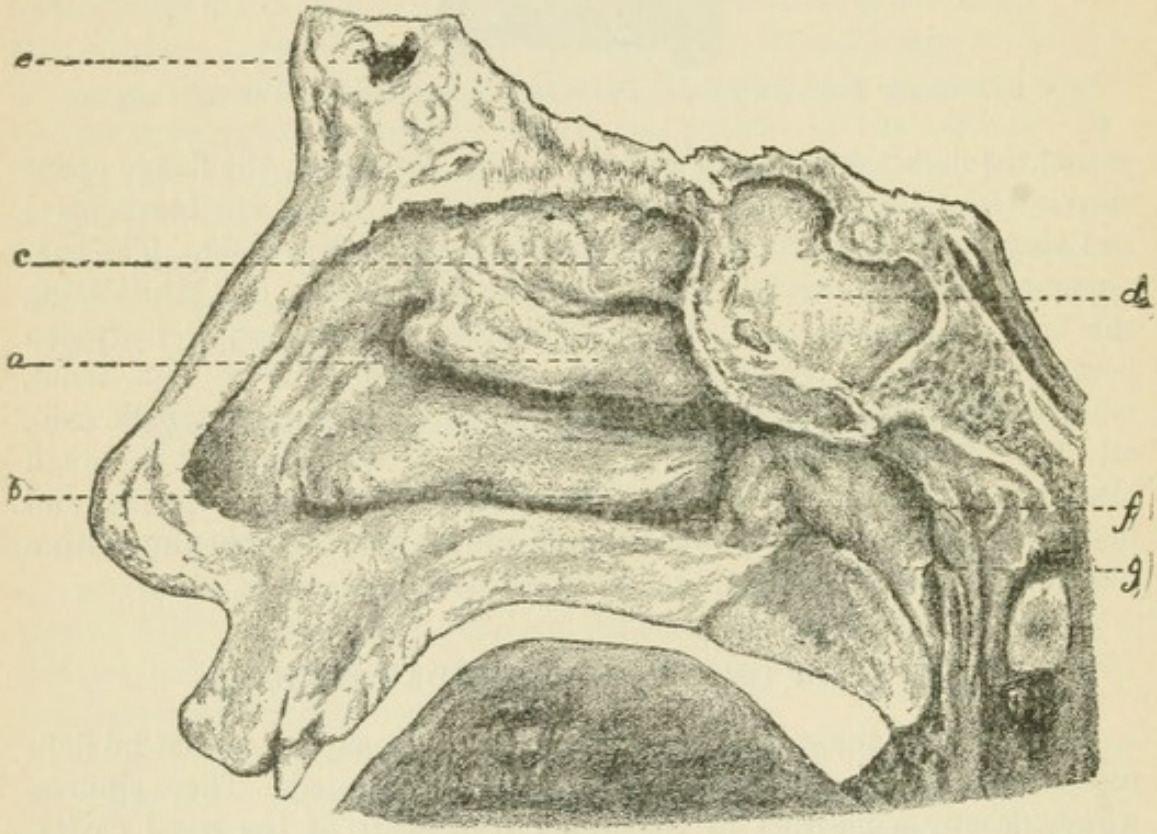

FIG. 216.—CROSS SECTION OF HEAD. From photograph of frozen section prepared by C. H. Stowell (4-5 natural size). *a*, Middle turbinated body; *b*, inferior turbinated body; *c*, superior turbinated body; *d*, sphenoid cells; *e*, frontal sinus; *f*, Eustachian orifice; *g*, naso-pharynx as closed in deglutition.

the local use of cocaine, which reduced the swelling sufficiently to allow free discharge, and, this condition being maintained for two or three weeks, recovery ensued. In some instances, permanent obstruction of the duct occurs, and then empyema of the frontal sinus follows. When this results, the pent-up secretions eventually cause a tumor at the upper inner angle of the orbit, disfiguring the patient, and displacing the globe of the eye.

The occurrence of suppuration will be indicated by rigors, excessive headaches, swelling, redness, and some local œdema and throbbing pain. Violent pain in the course of the supra-orbital and nasal

nerves is a common symptom. In suppuration caused by simple catarrhal inflammation, a small opening made with a drill from the nasal cavity, is usually sufficient to allow the confined secretions to escape; but when it results from syphilis, energetic measures are demanded, otherwise fatal involvement of the brain is likely to ensue. Then the frontal bone should be laid bare, and the cavity opened with a trephine in its most dependent part. Afterward provision should be made for free drainage into the nasal cavity, a drainage tube introduced, and the external wound allowed to heal. Finally, as recovery takes place, the drainage tube is removed through the nose. Other diseases of the frontal sinus come more properly within the domain of general surgery.

CHRONIC SUPPURATIVE ETHMOIDITIS.

A chronic suppurative inflammation of the ethmoid bone and membrane lining its cells is characterized by a persistent, somewhat offensive discharge, and obstinate neuralgic pains in the temples and forehead.

Etiology.—The causes are unknown. In two cases which have come under my observation, I am satisfied that the disease was the direct result of inflammation of the antrum, and not the cause of the latter, as it is believed often to be by McDonald (Diseases of the Nose, 1890). The suppuration results from abscess of the antrum in consequence of the occlusion of the opening from the latter into the nasal cavity, so that it becomes filled with pus which crowds upward and finally flows from the openings which are frequently present between the antrum and the ethmoid cells; by pressure this pus causes necrosis and perforation of the thin bones which separate the two cavities. The relation of parts will be readily understood by reference to Fig. 212.

Symptomatology.—Patients frequently suffer from neuralgic pains in the temple or over the orbit, which are more or less intermittent, and sometimes paroxysmal. Indeed, the symptoms closely resemble some of those attributed to empyema of the antrum; but there may be reasonable doubt whether these symptoms would occur in the latter affection were it not for coexisting disease of the ethmoid cells. There is usually purulent or muco-purulent discharge from the nose, which is often fetid, but not so offensive as in ozæna. This flux may be scanty or very profuse, is generally continuous, and usually comes from one side only. Upon inspection it may be seen filling the middle meatus and running over the middle turbinated body. Often inflammatory thickening of the external wall of the middle meatus is seen, which sometimes communicates through the probe a sensation of bony hardness, but usually it appears and feels more like a polypoid formation or fungous granulation.

Diagnosis.—The affection is to be distinguished from mucous polypi, atrophic rhinitis with ozæna, from suppuration of the antrum, and from

empyema of the sphenoidal and frontal sinuses. It may ordinarily be distinguished from *mucous polypi* by the presence of pus; this must be wiped away, and carious bone which often exists, or fungous granulations are to be carefully sought with the probe. Not infrequenly small polypi are associated with this affection.

Suppurative ethmoiditis must be distinguished from *suppuration of the antrum* by careful inquiry into the history and symptoms and by persistence of the discharge after the latter cavity is known to be healed. We readily distinguish *atrophic rhinitis* by the abnormal size of the nasal cavities, the peculiar stench, and collections of decaying crusts of mucopus. From empyema of the sphenoidal and frontal sinuses this affection is distinguished according to Max Schaeffer (*Deutsche Medinische Wochenschrift*, Leipzig, No. 41, 1890), largely by the position of the pus, which in disease of the frontal sinus covers the more or less swollen mucous membrane of the septum in the superior meatus, and in disease of the sphenoid cells passes down the pharynx, while in ethmoiditis it spreads out in the middle meatus.

Prognosis and Treatment.—It is probable that some of the cases recover spontaneously, but most of them continue for many months, and even years, in spite of the best-directed treatment. The indications are to remove any obstruction which prevents free exit of pus; to keep the parts cleansed, and as nearly aseptic as possible; and by judicious stimu-

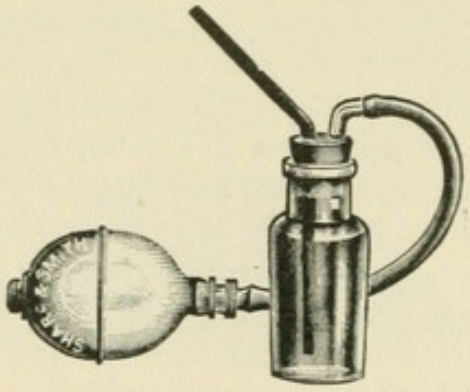

Fig. 217.—Holbrook Curtis' Wash Bottle (⅔ size). Used for the ethmoid cells.

lation to encourage healing. If disease of the antrum exists, it must be remedied before we can hope to cure the disease of the ethmoid cells. Polypoid growths or fungous granulations may be best removed by snare or sharp spoon, or small masses may be touched with the galvano-cautery or with monochloracetic acid. Dead bone must be carefully scraped away, and with the drill, trephine, or forceps the partitions of the ethmoid cells may be broken down to give free exit to the pus; but care must be taken not to excite undue inflammation, which might extend to the brain. I have found the most satisfactory results from injecting into the ethmoid cells, with a long, slender silver canula attached to a hypodermic syringe, about fifty per cent solutions of the hydrogen peroxide, and subsequently oily solutions containing oil of gaultheria

℥ i., oil of caryophyllum ℥ v., terebene ℥ x., ad ℥ i. of liquid albolene, the strength being slightly increased or diminished according to its effect. It should not cause pain for more than half an hour afterward. At the same time the nasal cavity should be washed two or three times daily, by means of the nasal syringe or Curtis' wash-bottle (Fig. 217), with a detergent solution, and a similar oily preparation, or one somewhat weaker may be used as a spray by the patient morning and evening. A powder containing five per cent of aristol, two per cent of cocaine, twenty per cent of boric acid, forty per cent of iodol, with sugar of milk for an excipient, may be advantageously used by the patient once or twice daily as an insufflation.

LUPUS OF THE NARES.

Lupus of the nares is a chronic affection of the mucous membrane usually secondary to lupus of the external surface of the nose, and characterized by the formation of small, irritable nodules which subsequently are the seat of indolent ulceration, followed frequently by a process of slow repair and cicatrization. It generally occurs in young persons of strumous habit, and is most liable to affect girls.

Anatomical and Pathological Characteristics.—Two varieties of the affection are recognized; one known as *lupus non-exedens*, in which atrophy of the affected tissues, including bone and cartilage, occurs without ulceration; the other as *lupus exedens*, which usually begins on the cartilaginous septum in the form of small, red, irritable nodules; these gradually coalesce, forming raised, uneven patches, which ere long become the seat of deep ulceration. This process extends slowly, destroying the soft tissues, cartilages, and even the bones, though repair is often inaugurated before the latter perish. The ulcers are covered with crusts under which the destructive process is going on in some places, while healing may be taking place in others.

Etiology.—Pathologists now generally recognize lupus as a tubercular disease, but the clinical history of the affection still leaves much doubt as to its true nature, and a large part of the profession is still unwilling to accept any dictum concerning it.

Symptomatology.—The disease occurs in young subjects, progresses slowly, causing the physical appearance already described, and it is attended by a discharge more or less profuse and offensive. The ulcers are not usually painful. As a rule, the disease first attacks the skin upon the cheek or nose, but it occasionally commences in the mucous membrane.

Diagnosis.—Lupus is liable to be mistaken for syphilitic affections of the nose, epithelioma, and true tubercular disease. The essential points in the diagnosis are the history, the development of red, irritable nodules, the progressive ulceration, and the slow process of repair.

There is usually a specific history in *syphilis*, which may be obtained by the adroit physician; thickening of the mucous membrane in patches or extensive swelling of the turbinated bodies comes on rapidly and is quite unlike the slowly developing, small, red tubercles seen in lupus. Syphilitic ulceration, though rapid, may usually be soon checked by appropriate local and internal remedies, which make no impression upon lupus.

We cannot always distinguish *epithelioma* from lupus in the beginning, but after a short time the characteristic features of the two diseases render the diagnosis easy.

The small red nodules found in lupus do not precede *true tubercular ulceration*, in which the ulcers are of a lighter color and present few if any of the bright red granulations usually seen in lupus, and show no tendency to repair. The presence of pulmonary tuberculosis would be a valuable point in the diagnosis.

Prognosis.—The disease continues for several years, but can sometimes be checked by appropriate treatment, though even when the ulceration has healed there is great tendency to recurrence, especially if the cicatrices remain red and indurated. With advancing age there is sometimes spontaneous recovery. In some instances it extends to the pharynx and larynx; in these, recovery is not likely to take place.

Treatment.—Arsenious acid and other tonics, with cod-liver oil, sometimes prove beneficial. The local treatment consists in removing or destroying the diseased tissues by the knife, curette, caustic, or the galvano-cautery. The treatment generally recommended consists of scraping the ulcers thoroughly with the curette, and then applying lactic acid, which should be repeatedly used until the process of repair is thoroughly established; other powerful caustics such as nitric acid, caustic potash, and zinc chloride have been recommended, but they are more severe and seem no more effective than lactic acid. The galvano-cautery has also been efficiently used for the same purpose. Koch's tuberculin has a wonderful effect on the disease, and has proven curative in some cases. Complete removal by the knife is sometimes practised.

RHINOSCLEROMA.

Rhinoscleroma is a rare affection, most cases of which have been observed in Austria, Hungary, and Italy, but a few have been seen in Germany. As described it is characterized by the formation about the nostrils or upper lip of smooth, flat, slightly raised, and extremely hard patches. The integument over these is either natural or of a reddish hue, and the spots are tender on pressure, but not otherwise painful. No constitutional symptoms are developed. The disease may appear in two or more places simultaneously; it progresses slowly, and may involve the alæ of the nose and septum, and may pass backward to the throat,

larynx, and even the trachea, causing extensive swelling of the mucous membrane and symptoms due to mechanical interference with the functions of the parts.

Etiology.—Rhinoscleroma is probably due to local infection, but the specific cause has not yet been identified, though micro-organisms are always to be found in the cells and lymphatic spaces of the affected part, and some of these have been specially studied.

Diagnosis.—Rhinoscleroma is to be distinguished from syphilis, epithelioma, and keloid. It is differentiated from *syphilis* by its chronic course, the absence of softening and ulceration, and the fruitlessness of specific medication. *Epithelioma* is softer, it soon ulcerates and bleeds, which does not occur in the affection under consideration and it is much shorter in duration. Rhinoscleroma must be distinguished from *keloid* by the location and progress of the case. Keloid usually occurs on the front of the chest as an irregular, corrugated, cicatrix-like excrescence, of slow growth.

Prognosis.—There is no tendency to spontaneous recovery, and if extirpated or destroyed it is sure to recur, but it does not shorten life.

Treatment.—Treatment is of no avail except as a palliative measure; obstructing masses should be removed from the air passages, and in case the larynx becomes involved, tracheotomy should be performed to prevent suffocation. Injection of Koch's tuberculin produces no reaction in these cases.

GLANDERS.

Glanders is a contagious disease derived directly by inoculation usually from a horse suffering from the affection. It is characterized by the formation of nodules, which soon become pustular and ulcerated, with symptoms of septicæmia and thick, muco-purulent, or sanious, offensive discharge. The affection is rare and is hardly observed except among veterinary surgeons, grooms, coachmen, and others whose occupation brings them in contact with horses. The disease may extend to the skin and various parts of the body, causing inflammation of the lymphatics, and it is then termed farcy. It may be either acute or chronic; the chronic form frequently precedes the acute.

Anatomical and Pathological Characteristics.—There is usually but little swelling and redness of the mucous membrane, which is covered by scabs, beneath which ulcers will be found in several places; it extends in less degree to the mouth, throat, and larynx.

Etiology.—Glanders in the human subject is always caused by direct inoculation from a horse suffering from the disease, and is due to the bacillus malei.

Symptomatology.—The acute form is marked at its outset by chills, high fever, and erysipelatous rash on the nose and face, soon followed by vesicles which burst and discharge a thin, serous fluid. These pustules

appear on the face associated with blebs. The secretion soon dries and forms a crust, under which a deep and rapidly spreading ulcer is found. Obstruction in the nose and throat is caused by the pustules. The chronic affection is characterized by similar symptoms, coming on more slowly, but it is likely to be merged suddenly into the acute form. When the disease becomes fairly developed, the muscles and tendons are often tender and the seat of rheumatic pain. The voice becomes husky or even lost, and some dyspnœa may develop; frequently there is slight cough. The discharge from the nose and throat is always extremely offensive, and usually profuse and thin at first, but later thick and glutinous, and sometimes streaked with blood. Nausea, diarrhœa, and abdominal pains are sometimes experienced. As the disease progresses, the patient passes into a typhoid condition, which, in the acute form soon terminates in coma and death. In the chronic form the patient may remain ill for several years, and he seldom fully regains his health.

Diagnosis.—Glanders is liable to be mistaken for rheumatism, pyæmia, typhoid fever, syphilis, and scrofulous eruptions. The essential points in the diagnosis are: the history of infection, the marked constitutional symptoms, nasal obstruction and offensive discharge, pains in the limbs, and abscesses in various parts of the body. It will be distinguished from *rheumatism* by the history, the presence of pustules and ulceration, and the occurrence of pain in the muscles and tendons, instead of in the articulations. It will be distinguished from *pyæmia* by less pronounced rigors, and by the pustules, ulceration, and offensive nasal discharge. It will be differentiated from *typhoid fever* by the history, the pustules, ulceration, and discharge. There should be no difficulty in distinguishing glanders from *syphilis*, if the history, marked constitutional symptoms, and failure of specific medicines to give relief are considered. It is readily distinguished from *scrofulous eruptions* by the marked constitutional symptoms.

Prognosis.—The chronic disease usually runs from four to eight months or even longer. Bollinger (Ziemssen's Cyclopædia of Medicine) mentions a case in which the symptoms lasted for eleven years.

The acute affection usually lasts for about three weeks when coming on independently; but when following the chronic disease, it generally terminates fatally within a week. The acute disease is almost always fatal, probably always if the nose is attacked. The symptoms preceding a fatal termination are protracted fever, night sweats, diarrhœa, delirium, and great exhaustion.

Treatment.—No form of treatment seems to be of any avail, but the case should be managed on general principles, and an attempt made to relieve suffering and sustain the vital powers.

NASAL AFFECTIONS IN ACUTE DISEASES.

Acute coryza is one of the earliest symptoms of *measles* and it is occasionally followed by severe inflammation, with epistaxis and muco-purulent secretions. Atrophic rhinitis and ulceration of the septum sometimes result.

Slight or severe acute rhinitis, with profuse serous or muco-purulent discharge and sometimes epistaxis, may attend *scarlet fever*.

An eruption in the nares, with obstruction of the passages, and subsequently epistaxis, is sometimes caused by *small-pox*, and cases are not very uncommon where the nostrils have become occluded by healing of the ulcerated surfaces.

Very distressing catarrhal symptoms, due to collection of secretions and formation of large crusts, sometimes attend *typhoid fever*. Under the crusts, ulceration may possibly take place, and sometimes the septum is partially destroyed.

Severe rhinitis sometimes attends *rheumatism*, but more frequently will be observed rheumatic or neuralgic pains, associated with but little if any evidence of inflammation. In all of these cases the diagnosis is comparatively easy, and the local treatment is that suitable for acute catarrhal rhinitis.

PERVERTED SENSE OF SMELL.

Parosmia.

Parosmia indicates a perversion of the sense of smell by which the patient experiences sensations of odors, usually disagreeable, which are not really present. It is said to be comparatively common in epileptics and among the insane, but is also observed in those who are otherwise perfectly healthy. The condition is analogous to neuralgia of a nerve of common sensation. In some it is constantly present, in others intermittent. In some patients the sensation occurs without an exciting cause, whereas in others agreeable odors smell offensive.

Diagnosis.—The diagnosis is made from the subjective features of the disease.

Treatment.—No rules for treatment can be formulated.

Anosmia.

Anosmia or loss of the sense of smell is dependent upon obstructions in the nares or disease of the olfactory nerves or lobes, or of their cerebral centres.

Etiology.—Anosmia is caused by obstruction of the nares from an acute cold, polypi, hypertrophy of the mucous membrane, or presence

of foreign bodies; also by disease of the olfactory nerves, either distal, or along the trunk, or at the centres. The most frequent cause is obstruction from mucous polypi, or swelling of the middle turbinated body, or of the mucous membrane covering the septum directly opposite. In these cases it is usually intermittent. It not infrequently results from injury to the head, as from blows or falls, and cases are on record in which it has been caused by prolonged exposure of the olfactory nerve to some pungent or extremely disagreeable odor. It has been caused by inhalation of irritating vapors, snuff-taking and local use of solutions of alum, or other nasal washes. It sometimes follows prolonged rhinitis especially of the dry variety, frontal neuralgia, or long-continued paralysis of the fifth or seventh nerve, and it is occasionally congenital.

Symptomatology.—In addition to the loss of smell, the patient is usually deprived of the sense of taste for all substances with a distinct flavor, but bitter, sweet, sour, salt, and acids are usually recognized. The loss of the sense of smell may be unilateral or bilateral, and is often intermittent, returning for a few minutes or even days, after exertion or without evident cause; but disappearing again without the slightest known provocation.

Diagnosis.—The diagnosis is made from the subjective symptoms and the exclusion by inspection of conditions causing obstruction of the nares.

Prognosis.—When due to mechanical obstruction, most cases are relieved when the obstruction has been removed. Cases dependent upon catarrhal inflammation of the Schneiderian membrane usually recover unless they have already existed for two or three years, in which case a favorable termination cannot be expected. When due to cerebral disease, the sense of smell is seldom restored.

Treatment.—The condition causing the affection should be sought and, if possible, removed. When this cannot be found, Mackenzie recommends the insufflation of a powder containing one twenty-fourth of a grain of strychnine with two grains of starch twice a day, and if it does not succeed he increases the strychnine to one-sixteenth or even one-twelfth of a grain (Diseases of the Throat and Nose).

CHAPTER XXXV.

DISEASES OF THE NASAL CAVITIES.—*Continued.*

CONGENITAL DEFORMITY OF THE NOSE.

THE principal nasal deformities which have been observed are: absence of the septum, double septum, narrowness of one naris as compared with the other, and occlusion of the posterior nares by membranous or bony tissues. Cases have also been recorded of complete absence of the nose, and of double nose. Closure of the posterior nares seriously interferes with respiration, especially in infants, and in them may be a serious menace to life.

TREATMENT.—Various plastic operations have been performed to correct these deformities. Congenital closure of the posterior nares, which principally concerns us, demands prompt attention, for infants will not thrive unless they can breathe through the nose. A passage must be forced through the obstruction by a strong probe, blunt forceps, or other instrument, and the opening thus made must be dilated and kept open until healing occurs.

FRACTURES OF THE NOSE.

Fractures of the nose are usually caused by falls upon the sharp edge of a step or the corner of a table, blows from the fist, a baseball bat, or flying missile, or the kick of a horse.

SYMPTOMATOLOGY.—The injuries vary from a slight fracture to complete crushing of the nose with great displacement and more or less injury to the surface. There is usually much swelling and ecchymosis of the parts and frequently subcutaneous emphysema. Profuse bleeding is likely to occur at the time of the accident, and to recur from time to time on sneezing or blowing of the nose. The sense of smell is often lost at first, and sometimes it is permanently destroyed.

DIAGNOSIS.—In order to make an accurate examination, it is sometimes only necessary to inspect the part with the aid of the speculum and rhinoscope; but if much contusion has occurred, complete anæsthesia should be induced, to allow of careful manipulation, but even then crepitus is not often detected.

PROGNOSIS.—Great deformity may result if the injury be not properly attended to at the time, and it must not be forgotten that a blow

may have also caused fracture of the base of the skull and serious injury to the brain.

Treatment.—With the patient under an anæsthetic, the fragments should be replaced, as nearly as possible in their normal position, by the finger and forceps; and if there has been much displacement, the part should be retained by plugging the nares lightly with antiseptic wool or by the introduction of plugs or tubes of gutta-percha or other substances, or by a spring, as practised by Roe (*New York Medical Record*, July, 1891). At the same time a plaster of Paris dressing may be applied with benefit externally. Sometimes it will be necessary first to reduce the swelling by cold applications, and wait from twenty-four to forty-eight hours before an attempt is made to replace the fragments; but it must be remembered that healing in this location takes place very rapidly, and it is desirable, therefore, to correct the deformity before union has occurred.

DISLOCATION OF THE NASAL BONES.

Dislocation of the nasal bones is a rare accident, which in the few reported cases has resulted from a blow on the side of the nose by which the bones at the upper third of the organ have been laterally displaced. Reduction is accomplished by means of combined internal and external manipulation while the patient is fully anæsthetized.

DEFLECTION OF THE NASAL SEPTUM.

Uncomplicated deflection of the septum does not often exist, but, associated with thickening of the cartilage and bone or enchondroma and exostosis, it is one of the most common deformities of the nose. Indeed, Mackenzie found a deflection of from half a millimetre to nine millimetres in over seventy-six per cent of 2,152 crania examined in the museum of the Royal College of Surgeons (Diseases of the Throat and Nose). Delavan has found among European races well marked deflection in fifty per cent of several thousand crania examined (Transactions of the American Laryngological Association, 1887).

Anatomical and Pathological Characteristics.—The cartilaginous or the bony septum, or both portions, are simply bent to one side, the cartilaginous portion usually being most involved. The deformity cause enlargement of one nasal chamber, at the expense of its fellow. Simple bending of the septum is uncommon, for in most instances of deflection there is also thickening, especially at the lower part of the convex surface.

Etiology.—The causes of the affection are obscure. It was at one time thought to be often congenital, but Zuckerkandl, as reported by Mackenzie and Delavan, states that it is never found before the seventh

year; this, however, is a mistake, for I have operated upon several cases in children under four years of age, and I observed it in a child less than eighteen months old. Delavan believes that it is generally due to injury, especially when situated anteriorly, and that otherwise it is due to hypernutrition, particularly when located posteriorly (*op. cit.*). Chassaignac attributes it to hypernutrition (*Bulletin de la Société de chirurgie*, 1851 to 1852, Tome II). My own observation is in accord with that of Delavan, excepting that I have found comparatively few cases that could be clearly traced to an injury; and the evidence in support of some of the older views, as suggested by Mackenzie, is, to say the least, insufficient. It is probable that not infrequently trauma is the starting-point, but undoubtedly chronic catarrhal congestion, by determining an increased flow of blood to the part, gives rise to hyperplasia.

SYMPTOMATOLOGY.—When the deflection is great, the most prominent symptom is twisting of the nose to one side, usually opposite the convexity of the septum. This deformity is sometimes very marked from bending to the side of the anterior edge of the cartilage, even though there is but little deflection farther back. More or less difficulty in nasal respiration is experienced according to the amount of obstruction. Interference with the free passage of air through the obstructed side causes the secretion to collect behind the convex portion and in the naso-pharynx, giving rise to post-nasal catarrh. Pressure upon the external wall, especially when this is associated with exostosis, often induces atrophy of the turbinated body of that side, whereas the inferior turbinated body of the other side is usually hypertrophied; and thus it frequently happens that patients find respiration easier through the cavity which upon inspection seems most obstructed. As further consequences of the obstruction, the voice acquires a nasal twang, and mouth-breathing becomes necessary, with all its attendant evils.

DIAGNOSIS.—There is no disease with which deflection of the septum is liable to be confounded if a careful rhinoscopic examination is made.

PROGNOSIS.—Most of the evil results of the obstruction can be remedied by a suitable operation, and the external deformity may be largely removed if the nasal bones have not been crushed so as to cause depression of the bridge of the nose.

TREATMENT.—The simplest treatment that has been recommended is for the patient to push the nose or the septum firmly over to the opposite side several times daily; but unfortunately this is seldom capable of accomplishing any good.

In 1851 Chassaignac recommended a form of treatment especially applicable to deviations with thickening of the cartilaginous septum. This consisted in dissecting up the mucous membrane and paring off the superfluous tissue. It is not always easy of accomplishment, but in certain cases no better operation could be devised. Blanden first advocated punching out a portion of the septum and establishing free con-

nection between the two nares (Compendium de Chirurgie Pratique, Tome III), but this does not afford the desired relief and cannot be recommended. Walsham proposes forcible replacement of the bent septum (Nélaton: Pathologie Chirurgicale, seconde édition, Tome III), its resiliency having first been overcome by stellate incisions. This practice has been effectual in moderate deviations of the septum without thickening. Where the deviation is marked, the redundant tissue must be removed in order to obtain perfect results. In slight

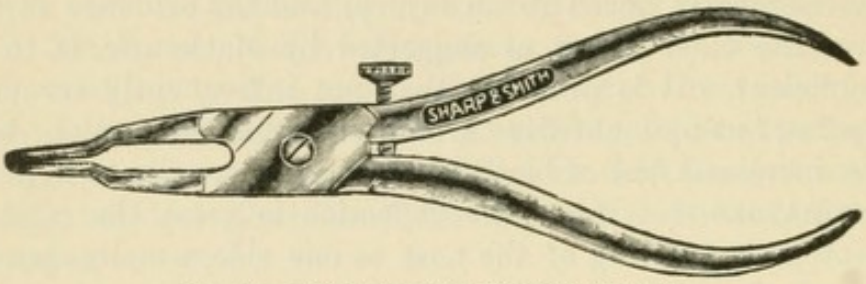

Fig. 218.—Ingals' Septum Forceps (½ size).

deviations most excellent results may be attained by making a crucial incision through the cartilage, the cut being made obliquely so that the bevelled edges will easily slide past each other. The septum is then forced into its normal position by forceps (Fig. 218), the vomer being fractured if necessary, and a gutta-percha plug of sufficient size is kept in the obstructed nostril until union has taken place. Where the stellate incisions are made either by knife or punch, the plug, or Adam's clamp, must be worn in a similar manner; the plug is simpler and quite as effective. In most instances it will be found necessary to remove the redundant tissue before a good result can be obtained. In cases where the cartilage is bent, almost at right angles, across the nostrils, I have found it most satisfactory (as I stated in Transactions American Laryngological Association, 1880) to dissect up the mucous membrane, remove a triangular piece from the cartilage of sufficient size, incise the cartilage farther

Fig. 219.—Ingals' Septum Knife (2·5 size).

back to destroy its resiliency, and then place a plug in the obstructed nostril to maintain the septum in position until union has taken place. When the obstruction is less complete, and there is simple deviation of the septum, I have frequently operated by making three or four horizontal incisions through the cartilage from the front backward, the cut being made obliquely from above downward and outward; sometimes across these near the middle is made an oblique vertical incision; the whole is then pushed over and retained by a plug or tube of gutta-percha until union has occurred. The main objection to this, and to other operations in which no tissue is removed, is that certain parts remain thickened and

the resiliency of the cartilage is seldom perfectly destroyed; the plug then has to be worn for several weeks, and when removed, in many instances, the cartilage will again return so far toward its old position as to prevent a satisfactory result. During the past two years I have frequently operated on these cases by cutting through from the front backward, in three or four places, and as much as possible beneath the mucous membrane, with a small trephine about two and one-half millimetres in diameter (Fig. 202). The removal of these cores destroys the resiliency of the cartilage so that it may be readily carried back and retained in its proper position. Whatever operation is adopted it is undesirable to perforate the cartilaginous septum because of the subsequent tendency of the secretions to dry about the edges of the opening and form obstructive crusts which are a constant annoyance to the patient. Perforations of the bony septum give rise to little or no inconvenience, provided they are as far as an inch back of the nostril, in which position the edges are kept moistened by the secretions, and scabs do not collect.

When deformity of the nose and obstruction to respiration result from protrusion to one side of the anterior edge of the triangular carti-

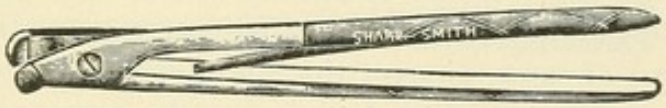

FIG. 220.—INGALS' RIGHT-ANGLE CUTTING-FORCEPS (⅓ size).

lage, the most satisfactory operation consists of incising the mucous membrane, over the edge of the cartilage, dissecting it back upon both surfaces, and then cutting off with a right-angle cutting-forceps (Fig. 220) all of the cartilage that projects beyond the normal plane of the septum into the obstructed nostril. This operation not only relieves obstructed respiration, but largely remedies the external deformity or twisting of the nose.

In order to secure sufficient anæsthesia for this operation with cocaine, it will be necessary to inject a few drops of a weak solution (Form. 140) under the integument on the outer surface of the cartilage; the mucous membrane on its posterior surface being anæsthetized in the usual manner.

ECCHONDROMA AND EXOSTOSIS OF THE NASAL SEPTUM.

Ecchondroma and exostosis of the nasal septum consist of thickening of the cartilaginous and bony parts of the septum with a more or less prominent outgrowth or spur in most cases, and usually some deflection. They are present in nearly all cases of deflected septum, and the etiology and symptomatology are practically the same in both. The projecting spur is usually directed from below upward and backward along

the line of articulation between the vomer and the perpendicular plate of the ethmoid. This may be small, or so large as to impinge against the outer wall of the nasal cavity. The spur is covered by mucous membrane, its anterior portion is cartilaginous, the posterior bony, and the inferior part immediately back of the cartilaginous septum is made up of bone of extreme hardness. These formations, because larger and exerting more pressure against the outer wall, are more liable than simple deviations of the septum to excite neuralgic pain and various other nervous symptoms. They are frequently found in cases of hypertrophic rhinitis, hay fever, asthma, and persistent supra-orbital or occipital neuralgia.

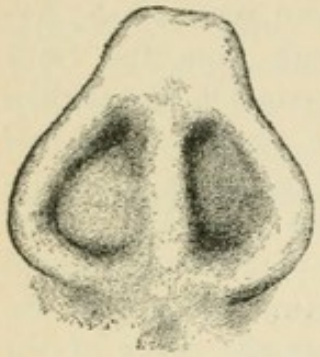

FIG. 221. — ECCHONDROMA AND EXOSTOSIS OF RIGHT SIDE OF SEPTUM. Hypertrophy of inferior turbinated body of left side.

DIAGNOSIS.—The diagnosis is easily made by inspection of the nares and the application of a probe, which detects the difference in the density of simple thickening of the soft tissue, and that of bony or cartilaginous tissue.

PROGNOSIS.—The obstruction may be completely removed by suitable operation, and many of the symptoms will be relieved accordingly; but the surgeon should not be too confident of the result, for in a considerable number of cases, some of the symptoms will remain.

TREATMENT.—The excessive tissue must be removed by operation, during which an effort should be made to save as much of the mucous membrane as possible. Before commencing the operation, the septum, both upon the affected side and upon the opposite side, and all other portions of the walls of the cavity liable to be touched during the operation should be thoroughly anæsthetized by cocaine. It will be found impossible to produce complete anæsthesia by applying cocaine to the surface near the nostrils, therefore when the operation is to extend far forward a few drops of the solution (Form. 140) should be injected beneath the mucous membrane where it joins the integument. Ecchondroma near the nostril may be removed by dissecting up the mucous membrane and paring away the cartilage with a knife, or cutting it with saws, trephines, or drills. Jarvis has devised a drill for cutting cartilage beneath the mucous membrane, but I have not seen its work. C. H. Wright, a dentist of Chicago, had made for me a burr which cuts cartilage very well in adults, but it will not cut mucous membrane except under firm pressure, and unfortunately does not accomplish much on cartilage in children. This, or other drills or trephines (Fig. 202) I use with an electric motor. The burr may be made to penetrate the mucous membrane by firm pressure while it is in motion; and then, by moving it slowly about, the excess of cartilaginous or bony tissue may be cut away without injuring the mucous covering. Any of the *débris* which is not extruded during the drilling process is washed away with a two per cent solution of car-

bolic acid, applied by a small syringe. Ordinary dental burrs will not cut cartilage. Trephines may be run directly through from the front

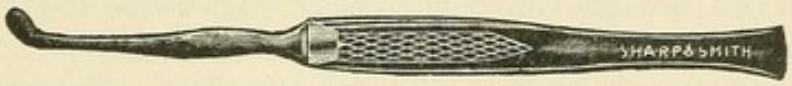

FIG. 222.—SAJOUS' KNIFE (½ size).

backward, and with care most of the mucous membrane may be preserved, but more of it is destroyed than when a burr is employed. For re-

FIG. 223.—NASAL SPUD (½ size).

moval of ecchondroma or exostosis situated farther back, I cut the mucous membrane along the lower edge of the spur with Sajous' knife

FIG. 224.—INGALS' NASAL SAW (½ size).

(Fig. 222), and bring the incision, in a curved line, forward and upward to the anterior and upper portion of the mass to be removed. The

FIG. 225.—INGALS' FLAT NASAL SAW (2-5 size).

mucous membrane is then lifted from the subjacent tissues by the back of the same instrument or a spud (Fig. 223); a saw is passed beneath

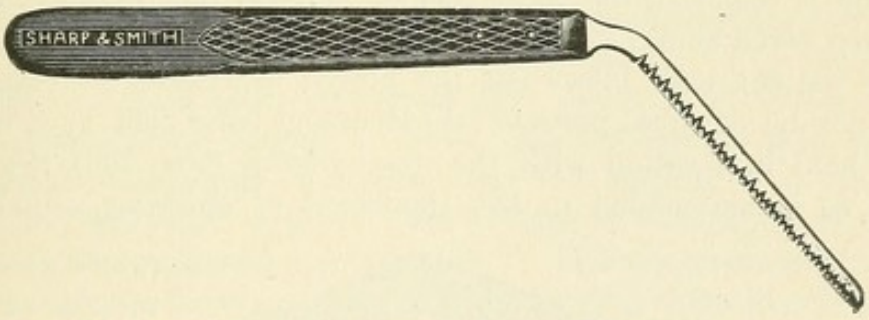

FIG. 226.—SAJOUS' NASAL SAW (½ size). Form used for downward cutting.

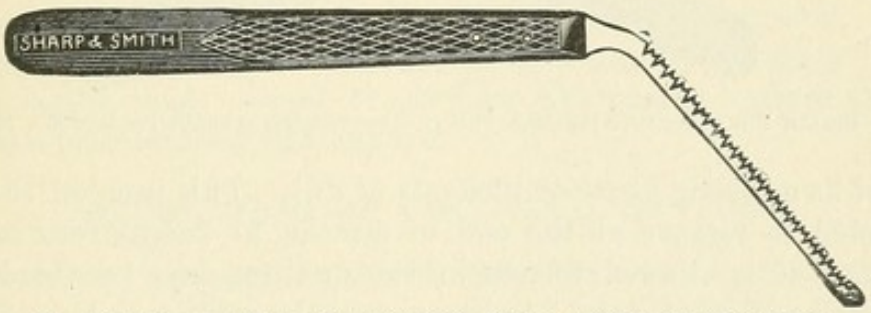

FIG. 227.—SAJOUS' NASAL SAW (½ size). Form used for upward cutting.

the loosened flap at the upper part of the spur, and a cut made downward on the normal plane of the septum until it reaches nearly to the

lower part of the nasal fossa; a narrow saw is then passed beneath the spur, and a cut made directly upward to meet the one from above. After the bone is cut through, it may be held by soft tissues, and these are cut by scissors (Fig. 200), to allow removal of the fragment. Sometimes stronger scissors, as shown (Fig. 228), will be needed. Subsequently with

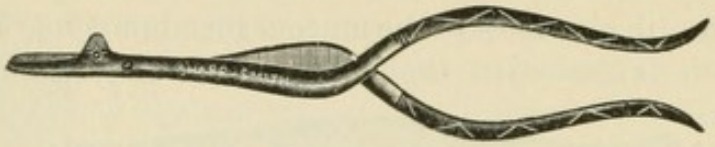

FIG. 228.—INGALS' HEAVY-BONE SCISSORS (½ size).

bone forceps (Fig. 229) any sharp spiculæ are cut off. In some instances I find it preferable to cut through the lower portion of the spur with a good-sized trephine. In others where the spur is not large, I use the trephine, only removing one or more cores as seems desirable. This latter operation is usually made without first having removed the mucous membrane, and the cut is made as much as possible beneath it. After

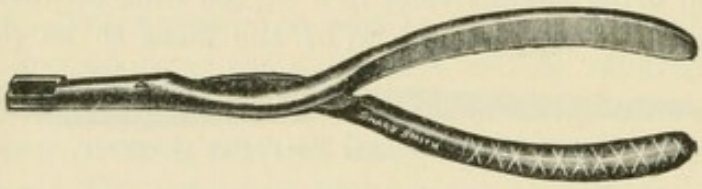

FIG. 229.—INGALS' NASAL-BONE FORCEPS (½ size).

the bone is removed, the loose flap of mucous membrane, which may have been saved above, is pressed down smoothly against the septum.

The patient then blows out the blood; the cavity is freely dusted with a powder of equal parts of iodoform and boric acid, and, while the flap is held in position with the nasal spatula (Fig. 230), the naris is packed, as recommended in the treatment of epistaxis, either with a

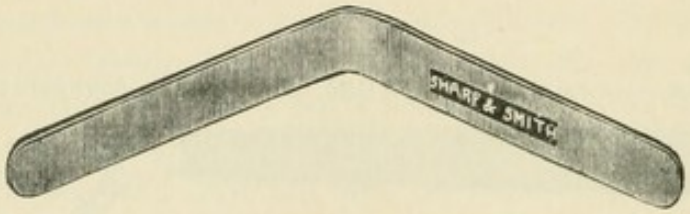

FIG. 230.—INGALS' NASAL SPATULA (½ size). Sets of three varying in width, angle of 45°. Made of steel.

strip of hæmostatic gauze or pledgets of lint. This tampon the patient is directed to remove at the end of sixteen to twenty-four hours, but sometimes it is allowed to remain two or three days provided there is no offensive odor or pain. Subsequently the wound is kept clean and as nearly antiseptic as possible, and the patient is directed to use two or three times a day a powder containing from twenty to fifty per cent of iodol.

Healing usually takes place in from one to six weeks, according to the size of the wound produced, and it is often remarkable that after a few months, even when large spurs have been removed, the membrane over the wound appears normal with no cicatrix that can be seen. H. Holbrook Curtis prefers to remove these spurs with the trephine alone; Bosworth usually employs saws; others are in favor of dental burrs. By using a trephine to cut the lower portion, where the bone is very hard, and a saw for the upper part of the incision when the spur is large, I am enabled to make the most complete and expeditious operation. The main objection to operating with the trephine alone is that after making two or three cuts it will be found that sufficient tissue has not been removed, and the parts are so obscured by bleeding that it is difficult to complete the operation accurately; it therefore requires much more time than with the saw; in the mean time the effects of the cocaine are liable to pass away, and much pain will be caused. Perforation of the cartilaginous septum should always be avoided, and an opening should not be made in the bony septum if sufficient room can be obtained without it; but often when there is a sharp deflection, together with the exostosis, it is impossible to free the nostril without opening through to the other side. There is, however, no serious objection to this, providing it is more than an inch back from the nostril, and the opening in such cases is certainly preferable to a cavity only one-third or one-half its normal size. Cartilage may be removed by electrolysis, preferably performed with both needles introduced into the tissue near each other.

A current is used of from 5 to 15 M.A., continued when the patient can bear it, for ten or fifteen minutes at each sitting. The operation is not repeated until the eschar is thrown off. James E. Newcomb, of New York (*Medical Record*, August 5, 1893), who has recently gone over this entire subject thoroughly, concludes that the method is worthy of a further trial, but that "whatever can be done by electrolysis can be, by other means, accomplished more quickly." In most instances cauterization of the inferior turbinated body of the opposite side will subsequently be found necessary, and sometimes it is desirable to remove during the operation a part of the inferior turbinated body of the same side. When the operation is finished, the cavity should be perfectly free and about one-third larger than normal, to allow for the partial closure which is sure to take place during cicatrization.

PERFORATION OF THE NASAL SEPTUM.

Perforation of the septum is often found as a result of syphilis, but it also not infrequently occurs, in persons of low vitality, as a result of constant picking at the nose; or it may happen during an exhausting disease, as typhoid fever, pneumonia, and phthisis. I have known quite

a large piece of the cartilaginous septum to be expelled, without warning, in a person apparently in perfect health; and I have even seen such openings independent of any of the causes already mentioned, which have occurred without the patient's knowledge.

TREATMENT.—The treatment consists in making suitable applications to heal any ulceration which may be present. It is not worth while to try to close the opening, an attempt which even at best could give little benefit, and which would usually result in failure.

HÆMATOMA OF THE NASAL SEPTUM.

Hæmatoma is a collection of blood in the septum indicated by the formation of a tumor usually at the lower anterior part, and projecting alike upon both sides; it results from an effusion of blood between the deep layer of the mucous membrane and the underlying cartilage.

ETIOLOGY.—Rare cases of spontaneous hæmatoma have been observed, but it is usually due to fracture of the bony or cartilaginous septum by violent blows on the nose.

SYMPTOMATOLOGY.—The blood collects immediately or within a few hours after the causative accident, and causes a smooth, uniform tumor of purple color, which hue sometimes extends to a considerable portion of the mucous membrane of the nose. These tumors are situated just within the nostril, are soft and fluctuating, usually symmetrical upon both sides, and may be so large as to protrude from the nostrils. More commonly they cause simply an extremely thickened appearance of the cartilaginous septum.

DIAGNOSIS.—The tumors are liable to be mistaken for mucous polypi, hypertrophy of the turbinated body, ecchondroma, or abscess of the septum. The essential points in the diagnosis are the symmetrical character of the swelling, the color, and the fluctuation.

These tumors are distinguished from *cartilaginous tumors* by their softness and symmetrical appearance; from *mucous polypi* by their uniform character, broad base, and color; from *extreme hypertrophy* of the anterior end of the inferior turbinated body, by their location in the septum, as demonstrated by the probe; from *abscess* by their color and by the result of exploratory puncture.

PROGNOSIS.—The enlargements sometimes exist for a long time, but usually, within a few days, they eventuate in abscess, the patient rarely recovering without a permanent aperture in the septum.

TREATMENT.—Cold applications to reduce the swelling and inflammation should be made at first; if the blood does not become absorbed, as sometimes happens, within three or four days, it is apt to become purulent, and the swelling must then be opened upon one side at its most dependent part. Usually a single opening will drain both sides, but an incision on each side may be necessary.

ABSCESSES OF THE NASAL SEPTUM.

Abscesses of the nasal septum may be acute or chronic. They are found in the same position as the hæmatoma just described. They may result from the latter, or follow from simple inflammation of the parts. The symptoms, diagnosis, prognosis, and treatment are essentially the same as those of hæmatoma of the septum.

FOREIGN BODIES IN THE NOSE.

Foreign bodies of great variety have been found in the nose where they are most commonly placed by children in play. Beans, peas, buttons, or pebbles, are most common. Insane people frequently insert things into the nares. Occasionally some of the contents of the stomach are lodged in the nose during the act of vomiting. I have seen one instance where a child, during the act of deglutition, choked and coughed, thus lodging in the posterior naris a cervical vertebra of a chicken, which remained there several months.

SYMPTOMATOLOGY.—Foreign bodies sometimes remain in the nose for a long time without exciting any symptoms. Substances which absorb moisture soon swell and obstruct the nostril, and beans, peas, and other seeds may germinate. Irregular bodies may excite acute and severe inflammation. Headache, often assuming a neuralgic form, is occasionally present at an early period. The most characteristic symptom is a more or less profuse discharge from one nostril, which becomes exceedingly offensive when the body is one which will take up moisture and decompose. Upon inspection, the nasal fossa usually appears filled with secretion, but when this is wiped away the foreign body may be seen, or felt with the probe.

DIAGNOSIS.—The presence of a foreign body is to be distinguished from exostosis, rhinoliths, other causes of nasal obstruction, and from simple catarrh, by the history, which may oftentimes be obtained from the child or its playmates; by the occurrence of the discharge from one side only, which does not occur in simple catarrhal inflammation of the nasal mucous membrane; by the offensive nature of the discharge in many instances; and by careful inspection or palpation with the probe. As an illustration of the difficulty which sometimes attends the diagnosis, I recall an instance in which a long match had been inserted into the nose and had been sought unsuccessfully by a physician. The mucous membrane was so swollen and the naris so filled with secretion that the object was found only after carefully wiping this away, and feeling backward with the probe along the floor of the nasal fossa. Since the discovery of the properties of cocaine, it is much easier to make a diagnosis in these cases, for by the injection of a small

quantity of this drug the swelling is removed and the mucous membrane is benumbed so that a careful exploration can be made. A good light is always essential to a satisfactory examination.

Foreign bodies are distinguished from *polypi* by their color, consistence, and mobility; from *exostosis* in the same way.

PROGNOSIS.—Small bodies may remain for a long time, even many years, without attracting attention. By the accretion of chalky deposits they may become the nuclei of rhinoliths. They are not dangerous, but in most instances sooner or later provoke an extremely offensive discharge.

TREATMENT.—The nasal cavity should be anæsthetized with cocaine, and the substance removed with forceps, catheter, probe, hooks, screws, posterior nasal douche, or the snare; the latter I have found more use-

FIG. 231.—GROSS' INSTRUMENTS FOR REMOVING FOREIGN BODIES FROM THE NASAL CAVITIES AND EARS.

ful than other instruments. The loop is easily passed by the sides of the foreign body, and when tightened upon it the object is firmly held so that it can be withdrawn. In one instance I extracted by this means a wild tooth from the floor of the naris which had caused a catarrhal discharge for several years.

RHINOLITHS.

Rhinoliths are cretaceous masses of comparatively rare occurrence which usually owe their origin to the lodgment in the naris of some foreign substance upon which phosphate of lime is gradually deposited from the secretions. They are generally hard on the surface, but softer toward the centre.

SYMPTOMATOLOGY.—The symptoms are similar to those described as due to the presence of foreign bodies, the most characteristic being obstruction and a fetid discharge from one nostril. When situated in the upper and anterior portion of the nasal fossa, they sometimes cause swelling of the face. The symptoms come on more slowly than those resulting from a foreign body; but as the calculus continually enlarges, the obstruction finally becomes greater. The calculus is usually single, but more than one may occasionally be found. It is generally of a grayish or blackish color, with a rough, and more or less uneven though sometimes smooth surface. Sometimes it becomes partially imbedded in the mucous membrane, which then is apt to ulcerate and bleed. The size of the calculus varies greatly. W. N. Browne records a case (*Edinburgh Medical Journal*, 1859) in which the stone measured one inch and three-quarters in length, one inch in breadth, and nearly half an inch in thickness.

DIAGNOSIS.—A rhinolith may be confounded with osteoma or cancer. It is distinguished from *osteoma* in that it is movable and can be penetrated by a sharp probe or needle. Owing to the fungoid, bleeding granulations which sometimes spring up from the edges of the mucous membrane, where ulceration has occurred, and also to the offensive discharge, it may be mistaken for *cancer*, from which it is distinguished by its slow growth, the comparative absence of pain, and by inspection and palpation with the probe.

PROGNOSIS.—Rhinoliths may remain many years, causing much annoyance, but they are not dangerous to life.

TREATMENT.—Rhinoliths may usually be removed with polypus forceps or the snare, or they may sometimes be crowded back into the naso-pharynx, when they will be expelled by the patient. If too large to be readily removed, they should be broken down with the nasal bone forceps (Fig. 229).

MYASIS NARIUM.

Synonym.—Maggots in the nose.

Myasis narium is a condition very rare excepting in the tropics. It is characterized by destruction of the soft tissues and occasionally of the bone, with offensive discharge, formication, severe pain, insomnia, and sometimes convulsions. It has been frequently observed in British India, South America, and Mexico, but in those countries it is said not to be found in the cooler atmosphere of high altitudes. Very few cases have been recorded either in Europe or the United States. A case is recorded by D. N. Rankin (Transactions of the American Laryngological Association, 1888).

ETIOLOGY.—Usually the worms owe their presence to the hatching of eggs deposited in or near the nostril by flies, which are attracted by the odor of an already existing discharge or foul breath.

SYMPTOMATOLOGY.—Soon after deposit of the eggs, the mucous membrane becomes irritable, tickling sensations, with attacks of sneezing, soon follow, and subsequently troublesome crawling sensations are experienced. There is a sanious or bloody discharge from the nostrils, and œdema of the face and eyelids may also appear; severe and sometimes excessive, unceasing, pain is felt at the root of the nose and over the frontal region. In this affection the mucous membrane, and even the cartilages and bones, may be destroyed, and the resulting inflammation may extend to the brain, causing convulsions and death. As many as two or three hundred maggots have been ejected from the nose in a single case. Upon inspection, the horrible condition may be readily detected.

DIAGNOSIS.—All the symptoms may be caused by other affections, therefore the diagnosis must depend upon finding maggots in the nasal cavity.

PROGNOSIS.—If neglected, a considerable proportion of cases will eventually prove fatal.

TREATMENT.—Chloroform has been found most efficient for destruction of the parasites. In some instances inhalation only, of chloroform is sufficient to effect a cure. When this does not succeed, the patient should be fully anæsthetized, and the nasal cavities thoroughly syringed with pure chloroform. This does not seem to affect the mucous membrane deleteriously, but it would cause extreme pain if the patient were conscious.

CHAPTER XXXVI.

DISEASES OF THE NASO-PHARYNX.

RHINO-PHARYNGITIS.

Synonyms.—Post-nasal catarrh, retro-nasal catarrh, follicular disease of the naso-pharynx.

Rhino-pharyngitis consists of chronic inflammation of the mucous membrane of the naso-pharynx, characterized by collection of viscid or drying secretion, and a tendency to hawk frequently and clear the throat, especially in the early morning or after eating. It is a very common and widespread affection, but seems especially prevalent in America, where it is found in all regions and among patients of differing age, sex, and condition; it is less frequent in warm and equable climates.

Etiology.—Beverley Robinson justly attributes it largely to cold and damp atmosphere subject to sudden and great changes of temperature, but believes that it is also due to a special diathesis which he terms *catarrhal* (Nasal Catarrh, 1880). Mackenzie believes it is mainly due to dust, and frequently to dyspepsia. I am satisfied that a cold, damp climate, and an excessive amount of irritating dust in the atmosphere, are the chief of its predisposing causes, and that disturbance of the digestive organs is a pronounced etiological factor in many instances; but I am equally satisfied that obstruction of the nares, as in hypertrophic rhinitis, is the exciting cause in a large proportion of cases; while in certain others the affection is due to extension of inflammation from the nares or oro-pharynx. Hypertrophy of Luschka's tonsil or even of the faucial tonsils undoubtedly causes the disease in some cases; but the catarrhal symptoms caused by hypertrophy of Luschka's tonsil, or excessive adenoid growths in the naso-pharynx, should not be confounded with the result of simple inflammation. Tornwaldt contends that it is often due to catarrhal inflammation of the pharyngeal bursa (Ueber die Bedeutung der Bursa pharyngea, u. s. w., Wiesbaden, 1885); this is undoubtedly true of some cases, but not of a large percentage. Many cases are apparently caused by submucous thickening at the sides of the posterior part of the vomer. I am unable to explain the direct relation of this thickening to the discharge and chronic inflammation, but I am satisfied of its etiological relation from the fact that its reduction will often greatly benefit, if not

completely cure, the post-nasal catarrh. Tobacco-smoking is a comparatively frequent cause, and the excessive use of alcoholic stimulants may produce congestion and inflammation of the mucous membrane here as in other localities.

Symptomatology.—In slight cases the patient is merely troubled with a sensation as of something sticking in the naso-pharynx, but usually the secretion is tenacious or dry, and difficult to dislodge, and gives the patient great discomfort, causing him to hawk and make frequent efforts at its removal. Distinct articulation is frequently prevented, partially from obstruction of the naso-pharynx and partially from a mild form of chronic laryngitis which often coexists. These conditions are most annoying early in the morning or after eating, when the patient's efforts to dislodge the secretion may produce nausea or even vomiting. The symptoms are especially troublesome in damp or chilly weather, or after catching cold. Dull aching in the upper part of the throat, and sometimes weight and pain in the occipital region, are experienced by some of these patients, but the latter is apparently due to the rhinitis rather than to the pharyngitis. The sense of hearing is often obtunded, in consequence of extension of the inflammation through the Eustachian tube.

Upon examining the pharynx, tenacious secretion will usually be observed coming down from the naso-pharynx, upon the vault of which similar secretion or adherent crusts may be found. The muous membrane is more or less congested and usually has a relaxed appearance, often exhibiting one or more enlarged follicles, especially just back of the posterior pillars of the fauces; indeed, in many instances this affection appears to be simply a chronic *follicular* inflammation of the upper part of the pharynx associated with a similar condition in the oro-pharynx. The diseased follicles referred to appear as small, oval or round, reddish granulations, usually raised about two millimetres, and from four to eight millimetres in diameter. Small erosions or ecchymotic spots are sometimes seen, and in young subjects adenoid growths in the vault are frequently present. The Eustachian orifices are often congested and swollen and sometimes blocked with secretion. Varicose veins are often observed in the pharynx, and the pillars of the fauces are usually congested and thickened. In advanced cases, atrophy occurs, with accompanying dryness and irritation of the parts. Whatever the condition, there is apt to be a similar affection of the oro-pharynx.

Diagnosis.—The disease may be confounded with adenoid growths or other tumors, or syphilitic disease of the parts. We can distinguish *adenoid* and *other growths* by inspection and palpation, and *syphilitic disease* by a consideration of the history, and by inspection, which is liable to reveal mucous patches, condylomata, ulcers, or cicatrices.

Prognosis.—The disease may extend over a period of many years,

but is not dangerous to life, and, contrary to the popular belief, which is fostered among the laity by designing charlatans, there appears to be no tendency for it to extend downward and eventuate in pulmonary tuberculosis. When the affection has lasted for many years it is doubtful whether it is often cured, but in the majority of cases removal of the nasal obstruction will greatly relieve, if not cure, the disease in the naso-pharynx.

TREATMENT.—As a means of prophylaxis the patient should be protected so far as possible from sudden changes of weather; he should avoid dampness and chills; summer and winter constantly wear woollen underclothes; keep the skin and digestive organs vigorous by the observance of proper hygienic rules, and when exposed to an excessive amount of dust in the atmosphere, protect the nares and pharynx by wearing loose pledgets of wool in the nostrils, or by some form of respirator.

The treatment of this disease resolves itself in the main into curing the nasal disease which has caused it. Constitutional treatment is indi-

FIG. 232.—POST-NASAL SYRINGE (2-5 size).

cated for debility, and faulty digestion must be corrected by appropriate treatment, as has been so judiciously insisted upon by Beverley Robinson (Transactions of the American Laryngological Association, Vol. X). In the direct treatment of the naso-pharynx, cleanliness is of first importance. This may be accomplished by means of the nasal douche, nasal insufflation, the post-nasal syringe (Fig. 232), or the free use of nasal or post-nasal atomizers. The salicylate wash (Form. 187) is an excellent detergent application; but any alkaline wash, as, for example, sodium bicarbonate or equal parts of sodium bicarbonate with sodium chloride ʒ i. ad O i. of water, or Dobell's solution may be used instead. It should always be borne in mind that with the nasal douche, and to a less extent even with the other methods of cleansing just recommended, there is some danger that fluid may pass through the Eustachian tube to the middle ear. This may generally be avoided by causing the patient to keep the mouth open, not to use too much force, and to be careful not to swallow while the application is being made. The solution should always be used lukewarm.

The parts having been cleansed, Mackenzie specially recommends the insufflation of astringent powders. The old-time application of a solution of silver nitrate, varying in strength from ten to sixty grains to the ounce, will be found beneficial in many cases; and astringent or stimulating sprays, either aqueous or oleaginous, are often desirable.

When there are enlarged follicles without great congestion, and where the parts remain moist, I have seen great benefit from the insufflation, two or three times per week, of two or three grains of a powder consisting of berberine muriate one part and sugar of milk or acacia two parts. For excessive secretion, either here or in the nares, I have found terebene beneficial in the proportion of about ten minims to the ounce of liquid albolene, combined or not with other substances as seems desirable. If the parts have a tendency to dryness, after they have been thoroughly cleansed the application of an oily spray containing from two to six grains of carbolic acid to the ounce may be made by the patient twice daily back of the palate, or in case he cannot do this a weaker spray may be thrown through the nose while the head is held backward so that it will run gradually down over the pharyngeal wall. Indeed, the same remedies are applicable here as to the nasal cavities, it being remembered that the naso-pharynx will tolerate advantageously applications from fifty per cent to one hundred per cent stronger than the nasal cavities.

THROAT DEAFNESS.

Morbid changes in the naso-pharynx, particularly when near the orifice of the Eustachian tube, frequently involve the latter and extend to the middle ear, affecting more or less the sense of hearing. Probably most cases of deafness are of this nature.

ETIOLOGY.—The disease may depend upon a paretic condition of the Eustachian tube, or chronic inflammatory thickening of its lining membrane, or any morbid state of the naso-pharynx which gives rise to obstruction of the Eustachian orifice. Edward Woakes considers this, or motor paralysis, the fundamental cause (Diseases of the Nose). He also attributes the deafness to exaggerated folds of mucous membrane at the orifice of the Eustachian tube, and to folds projecting from the sides of the pharynx, and to partial obstruction of the nasal cavity by exostosis, or hypertrophy of the turbinated bodies; whereby during inspiration, but especially deglutition, the air is rarefied in the tympanic cavity, producing depression of the drumhead. Persistence of this condition eventuates in permanent collapse of the membrane and resulting deafness. One of the most frequent causes of throat deafness is enlargement of Luschka's tonsil. Atrophic rhinitis is also a cause; the affection has also been attributed to syphilis, diphtheria, rheumatism, progressive muscular atrophy, chlorosis, and extreme anæmia.

SYMPTOMATOLOGY.—According to Weber-Liel, the chief feature of the complaint is paralysis of the tensor palati muscle (Mackenzie: Diseases of the Throat and Nose, Vol. II). In severe cases there is collapse of the Eustachian tube, the air in the tympanic cavity becomes rarefied and the tympanic membrane yielding to the pressure of the

denser air on its external surface becomes abnormally concave (drawn in) and as this movement of the drumhead is necessarily transmitted to the chain of ossicles, the foot-plate of the stapes is abnormally pressed into the oval fenestra. Secondary changes soon follow, passive congestion of the tympanic cavity leads to trophic changes of a more or less cirrhotic character, consisting at first in the growth of a low form of connective tissue, with subsequent atrophy. Adhesion takes place, the stapes becomes fixed in the fenestra ovalis, and the labyrinth becomes the seat of disease. The patient often complains of tickling or scratching sensations in the throat; of snapping sounds heard during mastication or deglutition; of fatigue in listening, and difficulty in hearing during general conversation, though he may readily understand one person talking alone; and often of noises in the head and giddiness.

Diagnosis.—In the mildest form, according to F. C. Hotz, professor of ophthalmology, Chicago Polyclinic, the tympanic membrane is of normal color and brightness, but abnormally concave (personal letter from F. C. Hotz, July, 1891). In the medium variety, attended by acute inflammation of the middle ear, the membrane is congested according to the degree of inflammation, and the injection may be limited to a small streak along the malleus or may occupy the upper flaccid portion only, or it may spread over the whole membrane. The Eustachian tube is obstructed, and the tympanic cavity contains more or less secretion, the presence of which is indicated by characteristic râles heard through the auscultating tube while insufflation is made through the Eustachian catheter. In the most serious variety, the drum membrane may be bright and clear or dull and opaque, its movements may be impeded indicating sclerosis or anchylosis, or they may be excessive, indicating atrophy, and the Eustachian tubes may be either closed or unusually patent. The drum cavity may be either dry and empty or it may contain inspissated mucus, and we must distinguish by the tuning-fork test whether the deafness is due to changes in the middle ear or to lesions of the internal ear. If the patient hears the sounding-fork better when placed near the external ear than when touched to his forehead or held between the teeth, we must assume that the internal ear is involved; but if the fork is heard better against the forehead or between the teeth, we conclude that the chief cause of deafness is located in the middle ear.

Prognosis.—In the mild variety the prognosis is favorable provided the congestion and swelling of the pharynx and Eustachian tube can be removed by occasional insufflation. In the second variety, also, the prognosis is good if proper treatment is adopted early; but if neglected, permanent damage to the structure and sense of hearing is likely to ensue. In the most severe or chronic form, the chances for cure or even relief are poor, especially when the tuning-fork test shows that the internal ear is affected; but even in these cases the prognosis is somewhat more

favorable if there are râles, indicating the presence of mucus in the tympanic cavity, or if, as sometimes happens, there is marked and frequent variation in the hearing power. In the majority of cases no improvement can be expected, and we are fortunate if by treatment we can check the onward progress of the disease and save the patient from absolute deafness.

TREATMENT.—Our first effort should be directed to removing the cause of the disease. Obstruction of the naso-pharynx, or of the nares by the various forms of inflammation or exostosis or tumors, should be removed and the inflammation subdued by the methods already suggested. For the chronic thickening and congestion of the rhino-pharynx, with extension to the Eustachian tubes, the frequent application of strong solutions of silver nitrate, varying in strength from forty to one hundred and twenty grains to the ounce, have been most highly recommended, and the various alteratives, astringents, and stimulants already recommended may be tried. In a considerable number of cases

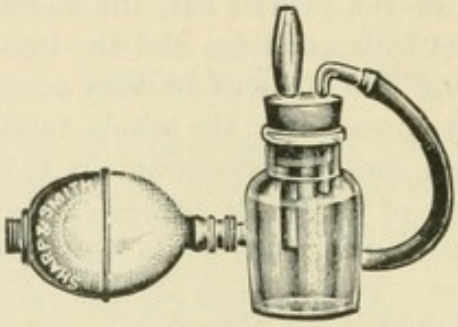

FIG. 233.—CURTIS' VAPORIZER. For inflation of the Eustachian tubes and middle ear. The bottle should be held in the hand with the thumb alongside the glass bulb. When applied to the nostrils, the thumb completely covers one and the glass bulb snugly fits the other orifice. The right hand grasps the rubber ball, and simultaneously with the rapid enunciation of the letter K or the guttural G, a number of pressures upon the bulb will inflate the middle ear without the trouble of taking a swallow of water. This method of treatment of the Eustachian tubes by the vapor of iodine, ether, chloroform, etc., dropped upon the sponge of the vaporizer, is reported to be very efficacious by H. Holbrook Curtis. By removing the sponge, the instrument may be used as a powder blower.

I have obtained much benefit from spraying into the naso-pharynx and Eustachian tubes, while the nostrils are held, a solution of two to five grains of menthol to the ounce of liquid albolene. This may be readily done by the Davidson atomizer No. 66 with the long tip (Fig. 106), and there is no danger in using fifteen to twenty pounds pressure, for the palate will yield before injury will be done to the drum membrane. As stated by Hotz, in addition to the treatment of the pharynx, in mild cases, when the chief trouble is the insufficient ventilation of the tympanic cavity on account of the catarrhal swelling in the Eustachian tube, it is only necessary every two or three days to supply the drum cavity with fresh air by means of Politzer's method. But when the tympanic cavity itself is the seat of catarrhal changes the use of the Eustachian catheter is indispensable for the efficient introduction of suitable reme-

dies. When the auscultating tube reveals the presence of mucus in the Eustachian tube and tympanic chamber, warm solutions of boric acid (gr. x. ad ℥ i.) are very serviceable. Two or three drops of this are put into the catheter and blown into the cavity by means of the air-bag. In the atrophic forms of otitis media, stimulating vapors are recommended, as of ammonium muriate, eucalyptol, or benzol

In cases of severer grade with acute inflammation, he specially recommends hot solutions of cocaine four per cent, frequently dropped into the external meatus, and warm compresses covering the ear and mastoid region, together with careful insufflations through the Eustachian catheter to ventilate the drum chamber and clear it of accumulated mucous secretions, and at the same time spraying this cavity through the catheter with solutions of boric acid, eucalyptus, or other suitable remedies. In this variety, rapid and copious secretion into the cavity is liable to take place, indicated by intense pain and bulging of the membrane, for which paracentesis should be done at once. In the severer forms of the disease the local applications recommended may be tried, but not much can be accomplished. Mackenzie recommends constitutional treatment by the use of iron, strychnine, and phosphorus, and suggests that in the later stages nothing remains but the doubtful operation of paracentesis of the tympanum or tenotomy of the tensor tympani (Diseases of the Throat, Vol. II).

These cases are most unpromising, and it is only by carefully adapting the treatment to the requirements and the peculiarities of each individual patient that we can hope to prevent even absolute deafness. The details of treatment are more properly set forth in works on diseases of the ear, and the treatment itself should be carried out by an experienced aurist.

HYPERTROPHY OF THE PHARYNGEAL TONSIL.

Synonyms.—Hypertrophy of Luschka's tonsil, adenoid growths in the vault of the pharynx.

An abnormal enlargement of the glandular tissue normally found in the vault and walls of the pharynx, is characterized by obstruction of nasal respiration, alterations in the voice, and in many cases partial deafness, with catarrhal symptoms and more or less deterioration of the general health. It is particularly observed in damp climates. It commonly occurs in children, but is not infrequently observed in young adults.

Anatomical and Pathological Characteristics.—The changes in the glandular tissue closely resemble those which are frequently witnessed in the faucial tonsil. The gland is of a grayish or pinkish color, though sometimes even of a bright red hue, and the surface often has a lobulated appearance. Enlarged blood vessels are not present upon the surface, as in many other forms of abnormal growth. The tissue

may be soft and friable (Fig. 234) or exceedingly firm. It consists of lymphoid structure and increased connective tissue similar to that found in hypertrophy of the faucial tonsil. The effect upon respiration and the general health depends upon the size and the amount of obstruction.

ETIOLOGY.—Heredity evidently bears some part in the etiology of the affection, although statistics have not yet proven the point; frequently several children in the same family will be found affected. It appears to be due in most cases to the same causes as enlargement of the faucial tonsil. The exanthematous diseases and diphtheria are common causes, and frequent colds, as well as the strumous and rheumatic diatheses, appear to be predisposing factors. McDonald (Diseases of the Nose, 1890) attributes the majority of cases to obstruction of the nasal passages, and consequent rarefaction of the air in the naso-pharynx during respiration. This theory, however, would seem to be opposed to the fact that nearly all cases of cleft palate are also affected by the disease; it certainly does not correspond with my own observations, although it is true that in many cases anterior nasal stenosis does exist.

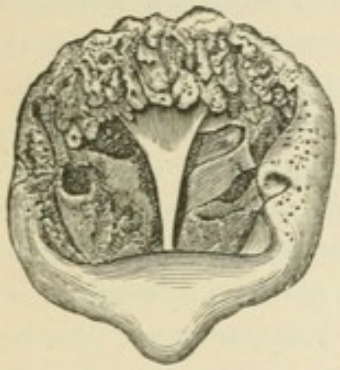

FIG. 234.—RHINOSCOPIC VIEW OF VEGETATIONS IN THE VAULT OF THE PHARYNX (COHEN).

SYMPTOMATOLOGY.—There is usually a history of mouth-breathing, which has lasted for several months or years, with all its attendant symptoms. During this time the parents have been continually disturbed at night by the loud snoring of the patient. The child is usually very restless, and often wakens from troubled dreams during the early part of the night, but later sinks into a heavy sleep, from which it wakens in the morning with headache or a feeling of malaise that does not wear off for several hours. Spasmodic croup is sometimes apparently caused by this condition. Nasal or post-nasal catarrh and partial deafness are not infrequently present, and it is common to find that these have come on after diphtheria or one of the exanthematous diseases. The deafness appears to be due to obstruction of the Eustachian tube by the hypertrophied gland, and in some cases to gradual extension of inflammation to the middle ear. Acute earaches are frequently caused by this affection. The deafness is sometimes outgrown as the gland atrophies during advancing life, and it may often be cured by removal of the abnormal tissue, but if allowed to persist for a few years it is likely to become permanent.

The voice is thick and indistinct in proportion to the interference with nasal resonance, and it becomes impossible for the patient to sound the letters *m* or *n*, especially when occurring before a vowel, *b* and *d* being sounded instead. In such cases the voice sounds as though the patient had a cold in the head. Wròblewski of Warsaw (*Internationale Klinische Rundschau*, Vienna, *Annual of the Universal Medical Sciences*,

1892) found adenoid growths in over fifty-seven per cent of one hundred and sixty deaf and dumb patients. Shortness of breath upon exertion is often noticed, and where children are trained to keep the mouth closed we may frequently observe catching or sighing respiration at intervals, an effort to compensate for the constant deficiency of air; and it is often necessary for these patients to clear out the mucus from the naso-pharynx by the act of hawking. A barking, reflex cough is sometimes present, and occasionally a spasmodic affection simulating whooping-cough. Often a peculiarly disagreeable nasal screatus becomes a fixed habit. Occasionally, though not in the majority of cases, rhinorrhœa is present.

The mucous membrane of the nostrils and anterior nasal cavities is found abnormally swollen in some cases, and in the majority the faucial tonsils are also enlarged. The uvula, pillars of the fauces, and edge of the palate are generally slightly congested, and frothy or muco-purulent secretion is found upon the pharyngeal wall dropping down from the naso-pharynx. In many cases the pharynx is relaxed and the follicles are swollen, as in advanced cases of follicular pharyngitis. The follicles, which are liable to be paler than the surrounding mucous membrane, usually increase in size toward the upper part of the pharynx, until just above the edge of the palate they become continuous with the glandular enlargement. In posterior rhinoscopic examination we should observe especially the posterior pharyngeal wall, the vault of the pharynx, and the choanæ. Irregularity of the upper outlines of the latter are among the most easily recognized signs of the disease.

Upon the pharynx the growth has a cushion-like appearance, more or less nodular upon its surface, but in rare instances it hangs from the vault in soft, pendulous masses resembling condylomatous warts. In color it is usually pale pink or grayish, though it may have any shade from this to a deep red. Its surface is not traversed by blood vessels. In adults, where atrophy has taken place, the remains of the gland may sometimes be seen as small excrescences. Palpation is often desirable in adults to determine the consistency of the growth, and it is frequently essential in children because of the difficulty of rhinoscopic examination. In performing it, a gag having been placed between the teeth, the forefinger of the right hand should be carried back to the pharyngeal wall and then turned upward behind the palate, where it at once detects the abnormal growth. Those unfamiliar with the normal feeling of the part should at first search for the septum and carry the examination from this backward and upward along both sides. Slight bleeding usually follows, though the examination is not specially painful to the patient. Chronic pharyngitis, rhinitis, or laryngitis will be found present in some cases, and occasionally deformity of the thorax will have resulted, as shown in the pyriform chest or pigeon-breast already referred to in speaking of hypertrophy of the tonsils.

DIAGNOSIS.—The affection is to be distinguished from nasal mucous polypi and fibroid tumors by inspection and palpation.

We seldom find *mucous polypi* at so early an age as hypertrophy of the pharyngeal tonsil; they are of a lighter color, semi-translucent, and usually have coursing across their surface blood vessels, which are not seen in this disease. They usually spring from the nasal cavities and may be readily detected by anterior rhinoscopy.

We find *fibroid tumors* much harder than the hypertrophied glandular tissue; they are frequently attended by severe epistaxis, and, upon being touched, bleed easily and profusely. They are usually of a bright red color with blood vessels apparent upon the surface. When large, they cause distortion of the neighboring parts. None of these signs are observed in hypertrophy of the pharyngeal tonsil.

PROGNOSIS.—Probably in seventy-five per cent of the cases the gland, if left to itself, would atrophy at about the twelfth or fourteenth year of the patient's age; but in the mean time irreparable mischief to the ear, the voice, or the general health may result. In the remaining cases the gland gradually diminishes in size, and disappears before middle life. When the affection has existed for a long time, the hearing may be permanently impaired, but usually removal of the gland greatly benefits this condition. The voice is not always perfectly restored, because a person having learned to talk with an obstruction in the naso-pharynx may require a considerable time to overcome the muscular habit, and in adults it may never be entirely remedied. The results of operative procedure, if not too long delayed, are most satisfactory.

TREATMENT.—Internally, particularly for anæmic children, I have occasionally found the syrup of iodide of iron of value. Sometimes other preparations of the iodides will prove beneficial and probably calcium chloride might cause some reduction of the gland in some instances. As a rule, however, medicinal treatment is of little value. Locally, astringents have been recommended, and seem to be useful in a few cases.

The most satisfactory results follow removal of the gland by surgical measures, and there are no contra-indications to operating even on young children. In a few patients where friends have objected to an operation I have employed chromic acid successfully. In using this caustic I fuse a few crystals on the end of a flat aluminium probe and pass this through the nostril to the enlarged pharyngeal tonsil, where it is held for two or three seconds. Previously the nares may be oiled to prevent the possible contact of any of the acid with its mucous membrane, and a small amount of cocaine may have been applied to the nares and naso-pharynx by means of powder or spray. The acid applied in this way usually causes a moderate amount of pain at the time, and some soreness for several hours afterward, but it is not severe. The applications may be repeated

once in from three to five days, being made through the opposite nostrils alternately.

The galvano-cautery may be used to destroy the growth, a bent electrode being passed up behind the palate, but the method is painful, tedious, and altogether not very satisfactory. Scraping off the gland by means of a long finger-nail or various forms of curettes is in favor with some operators and may in certain cases answer an excellent purpose; but usually the operation is less complete than when performed by Loewenberg's forceps, and therefore recurrence is more likely to take place. Écrasement by means of a bent snare is practised satisfactorily in some cases where the growth is very soft. Some operators prefer scissors or punch-like forceps, but they are both open to some objections. The scissors-like instruments which I have seen may be satisfactory for cutting out a portion of the mass, when it is soft, but they are not well adapted to a complete extirpation of the growth, so that other instruments must generally be used to make a complete operation. The punch-like forceps are not open to the same objection, but it is asserted that unnecessary bleeding results from their use.

By far the most satisfactory instrument to me for extirpation of the gland is Loewenberg's forceps, or some one of its modifications, especially that suggested by John N. Mackenzie. I have had a similar instrument made with shorter blades, for operating upon young children.

In performing the operation upon adults, it is often sufficient to anæsthetize the parts by cocaine, which may be applied by spray, syringe, or swab, or by the hypodermic syringe with a bent needle, by which it may be injected directly into the gland. My own custom has been to apply a ten per cent solution by spray behind the palate, and a similar solution by means of a syringe with a long blunt nozzle, to the upper part of the gland through the nares. The application should be repeated about once a minute until the part is fairly anæsthetized, which

Fig. 235.—Mackenzie's Modification of Loewenberg's Forceps.

will take about ten minutes. A self-retaining palate retractor should then be adjusted and the patient may hold the tongue with a depressor. The forceps are then inserted with the aid of the rhinoscopic mirror, and thus one or two bites may be made accurately, but subsequently the blood obstructs the view and the remainder of the operation may be postponed to another sitting or completed by the sense of touch if the patient will permit. Usually, even with cocaine, after two or three

bites have been made, patients prefer to have the remainder of the operation done at another time. Two or three sittings, however, will be sufficient in the majority of these cases. When an anæsthetic is objected to, or if for any reason a complete operation will not be permitted, a single, large excision may be recommended when the gland is soft. This, in the case of either children or adults, will generally give much relief. In children chloroform or ether should be administered, chloroform being preferable. When anæsthesia is complete, the child should be turned upon its abdomen and face, the mouth coming over the side of the table. A gag should then be inserted to hold the teeth apart.

Henrotin's gag is the simplest one that I have seen for this purpose, but sometimes Allingham's will be found preferable, especially for large children (Fig. 113).

The surgeon standing at the right side of the table, facing the patient's head, passes the index finger of his left hand behind the palate into the naso-pharynx, where it is retained as a guide for the forceps. The forceps may then be passed along the dorsal aspect of the finger and applied accurately to the growth. Thus piece by piece the gland is extracted, the forceps being guided each time by the finger until every part has been extirpated. Care should be taken to avoid seizing the posterior edge of the vomer or the projecting end of the Eustachian tubes. The latter often feel to the uneducated finger like abnormal growths. If care is taken not to turn the forceps sideways, there is but little danger of doing damage, providing the operator is familiar with the normal condition of the parts. Sometimes masses, located just back of the Eustachian orifice, are liable to be overlooked, but the most common difficulty arises from small pendent masses which hang just back of the choanæ and are liable to be crowded forward by the finger into the posterior nares. It is sometimes quite difficult to get the finger in front of this mass and push it back where it may be caught with the forceps. Some operators attempt to scrape this portion of the growth away with the finger-nail, but this effort can only be partially successful. When I find difficulty in removing this part with the post-nasal forceps, I employ a straight nasal forceps with cutting edge (Fig. 229), which I pass through the nostril, and guide to the proper point in the vault of the pharynx with my finger still retained behind the palate. In this manner a piece which might otherwise be difficult to catch is very readily removed. This procedure also enables us to determine whether the nasal fossæ are free, or if they are not to break down any adhesions or slight bony obstruction. With the patient in the position just recommended, there is no necessity for care in swabbing out the throat, as the blood cannot run *up* the trachea. With the patient on his back and the head thrown far backward, as recommended by some English surgeons, it is necessary to swab out the throat and

naso-pharynx frequently to prevent blood from getting into the air passages. There is usually considerable bleeding, but this stops as soon as the operation is completed. If undue hemorrhage should occur, the vault of the pharynx may be packed in the usual way or, as I prefer, with a long strip of gauze which is passed through the nares. This strip is saturated with a thick solution of tannic and gallic acids, as recommended for checking hemorrhage from the nares. This should be pushed back through the nares, and packed up behind the palate with the finger, which is inserted through the mouth. The nares should also be packed, and the gauze brought forward to the nostril to prevent the packing from falling into the throat. This packing should be removed within from twelve to twenty-four hours, to avoid the danger of exciting inflammation of the middle ear.

When the operation is completed, the mouth should be wiped out and the nostrils squeezed to press out what blood is possible, but it is neither necessary nor desirable to wash out the parts. The patient should then be placed in bed, and it is well for the nurse to keep him as much as possible upon the face till he has thoroughly recovered from the chloroform. This latter suggestion, however, is not very important, and it is seldom followed. The patient should be kept in bed for a few hours, and in the house for from two days to a week according to the weather. During this time I usually have insufflations made through the nostrils two or three times during the day, of a powder of two per cent of cocaine, fifty per cent of iodol, and sufficient sugar of milk to make one hundred parts. A simple detergent alkaline spray is not objectionable, but washes should be avoided for fear of injury to the middle ear; even sprays will sometimes find their way up the Eustachian tube, and therefore, unless by the odor there seems to be a special indication for them, I prefer to use the powder in connection with an antiseptic oily spray containing thymol gr. $\frac{1}{3}$, oleum caryophylli ♏iij., toliquid albolene ʒ i.

As a result of the operation there is usually a little soreness of the parts for a day or two, but not sufficient to interfere with swallowing. There is sometimes slight elevation of temperature; the improvement in breathing is marked and immediate in many cases; very often the friends become alarmed during the first night because the child breathes so quietly. Where partial deafness exists, considerable improvement may be expected within a few days or weeks, but recovery from alterations of the voice is sometimes less rapid. Some danger of otitis media exists from the liability of blood or other fluids passing into the Eustachian tube, but thus far no permanently bad results have been observed from it. In case it should occur, the continuous use of hot water in the ear, or hot water with glycerin and opium and dry heat externally, are the best remedies that can be employed.

In some cases nasal obstruction will be found to exist after the opera-

tion, and it must receive appropriate treatment subsequently. The final results of removing the hypertrophied pharyngeal tonsil are the most satisfactory of any with which I am acquainted in the domain of special surgery. The operation should not be recommended unless the diseased gland is large enough to interfere with nasal respiration, at least when the patient has a cold, or unless it affects the sense of hearing by pressure on the orifice of the Eustachian tube. In cases suitable for the operation, the patient's general condition undergoes a revolution for the better, which often astonishes even the physician, and gives the friends most unbounded satisfaction. In a child of from three to six years of age it is not unusual for a gain in weight of from twenty to twenty-five per cent to occur within five or six months after the gland has been removed. I have never seen any ill results follow the operation, and I think it safe to tell the friends that when properly done it is no more dangerous than the removal of a finger.

RETRO-NASAL FIBROUS TUMORS.

Fibrous tumors of the naso-pharynx are characterized by obstruction of the nose and dyspnœa, frequent epistaxis, and, when large, by great disfigurement known as frog face. They usually occur in young adults, sometimes in infants, but seldom after the twenty-fifth year of age, and they are much more common in men than in women. The affection is so rare that in over five thousand records of private patients suffering from disease of the throat and nose I find but six cases.

Anatomical and Pathological Characteristics.—The growths are generally smooth, hard, and unyielding, red or purplish in color, and sometimes ulcerated or bathed in a sanious secretion. They may spring from the periosteum of any portion of the roof or lateral walls of the naso-pharyngeal cavity, but they usually originate from the basilar process of the occipital bone and the body of the sphenoid, or from the upper cervical vertebræ. In character they are like fibromata in other localities, but occasionally are composed quite largely of erectile tissue. They are exceedingly dense, destitute of elastic fibres, and the blood vessels in their interior are small, while those in the investing membrane are larger, and have brittle walls which render them peculiarly liable to bleed. The tumor is usually single and attached by a broad pedicle.

Etiology.—The etiology is unknown.

Symptomatology.—The patient first experiences a sense of obstruction in the naso-pharynx, and finally one or both nasal passages become occluded. Many complain much of fatigue and drowsiness, probably due to imperfect aëration of the blood. Later, the symptoms depend upon the direction which the tumor may take in its development. If it extends toward the throat, it interferes with deglutition; by pressure

upon the Eustachian tube, it may excite inflammation of the middle ear, with more or less pain and deafness. When it projects forward, the nasal bones may be separated, the eyes pushed apart, and the bridge of the nose flattened, giving the characteristic deformity already mentioned as frog face. Pressure upon the lachrymal ducts causes epiphora. Sometimes the tumor extends into the antrum of Highmore and gives rise to swelling of the cheek. It may perforate and fill the sphenoid cells, and sometimes, as in one instance I have seen, it may cause absorption of the base of the skull, pressure upon the brain, and fatal meningitis. The filling up of the naso-pharynx interferes with articulation, giving a nasal twang to the voice, and, if the tumor is large and extends downward, great dyspnœa may occur. There is usually profuse purulent or muco-purulent secretion, sometimes offensive in character; and epistaxis, frequent and sometimes dangerous, is a common symptom. Dysphagia may be present. By inspection of the anterior and posterior nares, and palpation with the finger, the characteristics already pointed out may be readily detected.

Diagnosis.—The growths are liable to be mistaken for mucous or fibro-mucous polypi and sarcomata. From the latter they can only be distinguished by a microscopic examination. The essential points in the diagnosis are the age, sex, smoothness and density of the growth, and frequent epistaxis. They are distinguished from *mucous polypi* by their color, density, and tendency to bleed. Fibromata are distinguished from *fibro-mucous polypi* or tumors, the latter being less dense, having less tendency to bleed, and by microscopic examination. We might possibly mistake *hypertrophy of Luschka's tonsil* for fibromata, from which it will be differentiated by the age of the patient, its slower growth, lack of tendency to bleed, and by its having a lighter color, more irregular surface, and less density. Adenoid vegetations in the vault of the pharynx bleed easily, are soft, irregular, and occur at an earlier age than fibrous tumors.

Prognosis.—The growths tend steadily to increase in size, and, unless recognized and removed, will prove fatal in most cases, in the course of four or five years. Even when removed, there yet remains a strong tendency to recurrence, but fortunately, if they can be kept in check until the patient has attained the age of twenty-five, there is a tendency to spontaneous arrest of development.

Treatment.—If possible, the tumor should be removed through the natural passages by the écraseur, galvano-cautery, or by electrolysis. When large, it may be necessary to adopt the more severe measures recommended by Dupuytren, Rouge, Langenbeck, Chassaignac, Ollier, Lawrence, Palasciano, or Rampolla, which consist of various operations for exposure and removal of the tumor through the face that are fully described in the textbooks on surgery. I have never seen cases in which these methods were necessary, and the experience of Lincoln

(Transactions of the American Laryngological Association, 1883), as well as my own experience in two cases, show that even large tumors may be extirpated through the nares and naso-pharynx with even better results than are obtained by external operations. The simplest operation, and one which is sometimes attended by success, consists of electrolysis, which is performed by passing one or more needles connected with the negative pole into the tumor from behind the palate or through the nares, a single needle connected with the positive pole being introduced in a similar manner. A continuous current as strong as the patient can tolerate should be used, and the operation continued ten or fifteen minutes, and repeated about once a week or less frequently according to circumstances, until the growth has been dissipated.

Ligatures have been employed for the removal of these growths, but they are less satisfactory than the écraseur or galvano-cautery. In all cases when ligation is practised, a thread should be passed through the neoplasm and brought out at the mouth so that upon separation the mass may be removed before it falls deep into the throat and causes strangulation.

When a strong écraseur of sufficient power can be passed about the tumor, it may be readily and safely removed by this instrument, but the chances of recurrence are greater than if the galvano-cautery snare is used. Evulsion by strong forceps has been practised in some cases, but this method is not generally applicable. The tumor may be cut away with a curved, blunt-pointed bistoury, curved scissors, or strong cutting-forceps; or it may be removed by the gouge. Any of these methods are applicable in some instances, but they are apt to be attended by profuse hemorrhage, and if much force is used the resulting inflammation may prove fatal by extension to the brain, as in two of Ollier's cases (Spillmann: Dictionnaire Encyclopédie des Sciences médicales, fig., seconde série, Tome XIII).

When the tumor is pedunculated, it may sometimes be secured in the loop of an écraseur, but more easily in a loop of steel wire used with the ordinary snare; usually the tissue is so firm that it cannot be cut with the cold-wire snare in common use. The No. 5 piano wire used for mucous polypi is liable to break, and wire of larger size cuts the tissue much less easily, so that it cannot be drawn through the pedicle excepting with a stronger and much more powerful instrument. The galvano-cautery snare (Fig. 207) is the best instrument for the removal of these tumors whenever they are sufficiently pedunculated to allow of its employment. In performing the operation, I pass two soft catheters through the naris, endeavoring to carry one on either side of the growth, and bring them out of the mouth. Into the ends that are brought out of the mouth the ends of a piece of platinum wire about three feet in length are introduced and pushed on until they

come out of the nostril. I attach a thread to the wire loop to enable me to draw it backward in case of failure on the first attempt to place it about the tumor. The catheters with the wires protruding from the nostril are now drawn upon and the loop, passing back into the mouth, is carried with the finger or with the aid of a post-nasal snare-applicator (Fig. 236) up about the tumor, where it is drawn firmly into place. The catheters are then withdrawn, and the wires intrusted to an assistant, who holds them carefully, to prevent their becoming crossed in the naris. The ends of the wire are then slipped through the tubes of the galvano-cautery écraseur and fastened to the ratchet on the handle. It is desirable to have the distal ends of this electrode separated about a quarter of an inch or even more, so that it may be the more readily passed upon either side of the tumor. As the instrument is pushed into the nose, the ratchet is turned to tighten the loop, which is drawn

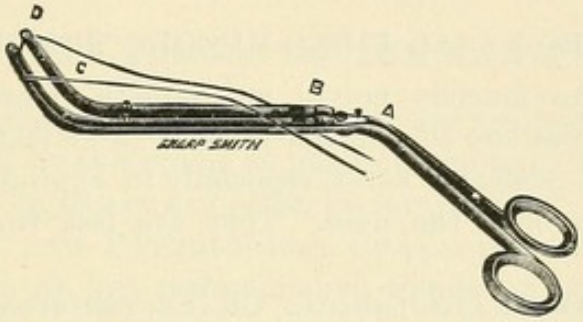

FIG. 236.—INGALS' POST-NASAL SNARE APPLICATOR (½ size). For tumors in naso-pharynx. The wire loop is held in notches at D by the slides B, C, which are held firmly by the cam A. As the loop is carried behind the palate, the blades are opened so that the wire incloses the tumor; it is then tightened, the cam is loosened, the slides B, C are drawn slightly backward, and the wire is released and left in position while the applicator is withdrawn.

tight upon the pedicle of the tumor before the electric current is turned on.

As it is very difficult to adjust the platinum loop properly with the patient under ether or chloroform, I have in recent cases relied upon the anæsthetic effects of cocaine; but its benumbing effect in this locality is not sufficient to prevent considerable pain during the burning off of the growth; therefore, when everything is in readiness, I tell the patient to bear the burning as long as possible, and that I will stop the current as soon as he requests it. The current is then turned on and the ratchet tightened at the same time. The patient will endure the pain two or three seconds, then the circuit is broken and he is allowed to wait two or three minutes; as soon as he is again ready, the circuit is again closed and thus the process is continued until the pedicle is burned through. The tumor is then seized with a pair of post-nasal forceps and withdrawn through the mouth. There is little or no hemorrhage from this operation.

Whenever as the result of an operation hemorrhage ensues, it may be necessary to plug the posterior nares. For this purpose I have

found most satisfaction in passing through the naris a long strip of gauze, rendered styptic by saturation with tannic and gallic acids, as recommended in the treatment of epistaxis. The gauze is pushed back with the probe through the naris to the naso-pharynx, and there it is packed into the vault, with the finger carried up behind the palate. Finally, the naris itself is completely filled to prevent the plug from falling into the throat if it should become loosened. The tampon should be removed within from twelve to twenty-four hours, by traction upon the end protruding from the nostril, by which the strip is gradually unfolded. In case clotting of blood has rendered the tampon hard, and bound its folds together, it should be softened by gently injecting into the nostril a warm solution of sodium bicarbonate. Should recurrence of the tumor take place, it should be treated while it is yet small by the galvano-cautery or by electrolysis.

RETRO-NASAL FIBRO-MUCOUS TUMORS.

Retro-nasal fibro-mucous polypi are smooth, more or less ovoid tumors, varying from two to ten centimetres in diameter. They cause obstruction of the posterior nares, especially in expiration, with consequent inability to blow the nose. They are less frequent than the fibrous tumors.

Anatomical and Pathological Characteristics.—The growths originate near the posterior opening of the nasal fossæ and are more or less fibrous or mucous according to their position. Those growing

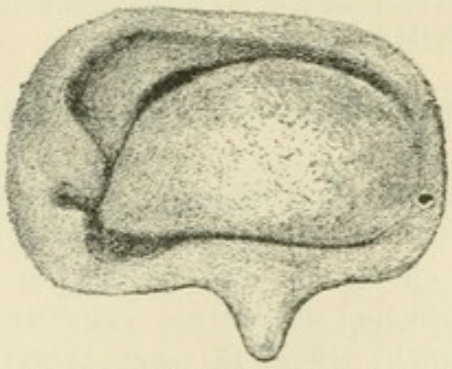

Fig. 237.—Retro-Nasal Fibro-Mucous Tumor.

largely from the retro-nasal space are mostly fibrous, those from the nares, as a rule, are chiefly mucous, in character. They do not cause so much pressure as fibrous tumors, and do not displace the bony structures like the latter.

Etiology.—The etiology is unknown.

Symptomatology.—The growths develop slowly, and are attended by the well known symptoms of nasal obstruction.

Diagnosis.—The retro-nasal fibro-mucous polypi are to be distinguished from fibrous and mucous polypi and malignant growths. They differ from *fibrous tumors* in that they are less dense, they do not de-

stroy the bony structures, and they are not attended by frequent epistaxis. They are distinguished from *mucous polypi* by their greater density, their darker color, and by their size and position. They are distinguished from *malignant growths* by the history, absence of pain and hemorrhage, smooth surface, and less degree of density.

PROGNOSIS.—The tumors grow slowly, and when removed have little tendency to recur.

TREATMENT.—If not too firm, the tumors may be safely torn away with post-nasal forceps, but they are best removed with the steel wire écraseur or galvano-cautery applied as recommended in speaking of fibromata.

RETRO-NASAL CARTILAGINOUS TUMORS.

True cartilaginous tumors of the retro-nasal locality are so rare as to barely need mention. Only three or four cases are on record.

MALIGNANT TUMORS OF THE NASO-PHARYNX.

Malignant tumors of the naso-pharynx are comparatively rare ; they are characterized by symptoms of nasal obstruction, with abundant discharge, frequent epistaxis, and often by severe pain.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—The growths are usually more or less pedunculated, somewhat pyriform in shape, and they have a nodular or lobulated surface covered by mucous membrane. They appear to be mostly of a sarcomatous nature, and often contain mucous or fibrous elements to a considerable extent. Microscopically they are found to contain the usual round or spindle-shaped cells and sometimes cartilaginous cells. In common with malignant tumors elsewhere, they are characterized by rapid growth, speedy recurrence after removal, and tendency to form new deposits in other organs.

ETIOLOGY.—The etiology is unknown.

SYMPTOMATOLOGY.—The tumors cause the common symptoms of nasal obstruction, with more or less discharge and bleeding, and often, but not invariably, severe lancinating pain shooting toward the ear and most troublesome at night. As the tumor increases in size, dyspnœa and dysphagia may become pronounced. It may be readily seen upon rhinoscopic inspection.

DIAGNOSIS.—The malignant tumor is to be distinguished from other retro-nasal growths by the features mentioned in speaking of fibrous and fibro-mucous polypi, and by microscopic examination.

PROGNOSIS.—The tumors grow rapidly and terminate fatally, usually within from four to six months. Recurrence is the almost constant rule.

TREATMENT.—When seen in the early stage, if possible, the growths should be thoroughly removed by the steel wire or galvano-cautery snare; but more serious operations cannot be advised.

CYSTIC TUMORS OF THE NASO-PHARYNX.

Cystic tumors of the naso-pharynx are of rare formation; only a few cases have been reported in this country, by Lefferts, Clinton Wagner, and myself. They are characterized by the usual signs and symptoms of nasal obstruction. They are most readily removed by evulsion with strong post-nasal forceps, and show little or no tendency to recurrence.

Diseases of the Thyroid Gland and Œsophagus.

CHAPTER XXXVII.

DISEASES OF THE THYROID GLAND.

GOITRE.

Synonyms.—Bronchocele, Derbyshire neck, struma.

Goitre consists of an enlargement of the thyroid gland, which may be vascular, parenchymatous, or cystic.

Anatomical and Pathological Characteristics.—In the vascular variety in some cases the veins, in others the arteries, and in still others all the blood vessels are enlarged, elongated, and tortuous, and the walls may be greatly thickened, so that the vessels themselves make up a large part of the increased size of the gland. In the parenchymatous variety the glandular structure itself is increased, sometimes the alveoli are much enlarged, and the tumor is made up in great part of colloid material, while in other cases the alveoli are smaller and the tumor is composed largely of the solid stroma. In many instances the goitre consists mainly of true adenoid growth. In cystic goitre there may be one or more large or small cysts, usually combined with hypertrophy of the parenchyma to a greater or less extent. As a rule, these cysts contain tenacious, ropy, albuminous fluid, often more or less tinged with blood from rupture of varicose veins into them, and of various shades of color in consequence of the amount or condition of the blood which has been thrown out. Sometimes their contents are entirely serous and in other cases entirely hemorrhagic in character. These growths sometimes attain enormous size. They are more frequent in women than in men, and are most apt to occur at about the age of puberty. The disease is most common in the Italian and Swiss Alps, the Pyrenees in France, in the Himalayas, in Derbyshire and Nottinghamshire, England, and in certain limited but not well defined areas in the United States.

Etiology.—The cause cannot be definitely determined; but the commencement can frequently be traced to repeated congestion of the thyroid body occurring at the time of menstruation, or due to violent efforts. Goitre is sometimes hereditary. It is often attributed to the drinking of snow and glacial water, water impregnated with chalk, or to bad air and bad surroundings and deficient sunlight; but the prevalence of the disease in places differing from each other widely in atmo-

sphere, temperature, and surroundings, and in some of which the drinking-water cannot possibly account for it, shows that we are still in the dark regarding the etiology.

Symptomatology.—The symptoms depend upon the amount of pressure exerted upon surrounding structures. The extent of pressure is not necessarily commensurate with the size of the tumor, which, though small, may send prolongations downward and backward that press upon the trachea or the pneumogastric or recurrent laryngeal nerves and cause alteration of the voice, and dyspnœa, which may be slight or severe. When dyspnœa is severe, it often comes on in paroxysms due to acute congestion and swelling of the already narrowed tube. These attacks are sometimes speedily fatal, and though the patient may recover from one attack he is liable to others during which the danger is great. Pressure upon the brachial plexus may cause pain, numbness, or even paralysis of the arm; but there is seldom any pain referred to the enlarged thyroid gland.

Diagnosis.—There is usually no difficulty in the diagnosis excepting in rare cases, where small goitres press posteriorly, causing difficulty in respiration, while the external growth may be hardly perceptible. Pressure upon the veins causes turgescence and lividity of the face, with prominence of the superficial veins over the tumor, and passive hyperæmia of the brain. There is occasionally, though not often, pressure upon the œsophagus, which then causes difficult deglutition. The gland, which is connected with the trachea, rises and falls during deglutition unless too large; the skin over it is freely movable, and the tumor is not attached to the jaw and does not involve the surrounding parts. The size varies from slight fulness of the neck to an enormous growth. The surface is sometimes even, but often nodular, and in extreme cases lobulated. The fibro-cystic variety, which is most common, has an irregular surface, firm to the touch, with here and there soft spots over the cysts.

It is distinguished from tumors of other portions of the neck by its position and movements during the act of swallowing. It is distinguished from *exophthalmic goitre* by absence of the ophthalmic and cardiac signs; and from *malignant tumors* by comparative absence of pain, and by not being adherent to other tissues and consequently moving beneath the skin and with the deglutitory movements of the larynx and trachea.

Prognosis.—The tumor usually slowly increases for many years, but is always a source of danger, as, from sudden swelling or steady pressure, with acute inflammation of the lining membrane of the air passages, it is liable to cause strangulation.

Treatment.—It is necessary to remember that endemic causes play a prominent part in the etiology of goitre, and therefore removal to some other locality may be the most important measure in effecting a cure. If the tumor is small or of medium size, it may often be dissipated by iodine in some form. The tincture of iodine may be applied

locally to the neck, and the remedy given internally in the form of potassium iodide in doses of from gr. v. to gr. xx., or the tincture of iodine in doses of ♏ v. to xx. may be administered in capsules, which are taken with a large draught of water, three hours after each meal. The internal use of the remedy often fails, and then injections have been practised in some cases with excellent results. Here again iodine may be used, but it is important that the solution should be thoroughly aseptic; for this purpose I would recommend the aqueous solution prepared by J. E. Clark of Detroit for the treatment of tuberculosis. Hypodermic injections into the tumor, of carbolic acid in doses of ♏ xv. to lx. of a three to five per cent solution, are sometimes followed by excellent results. These should be given once or twice a week according to the irritation they produce. Injections of iodoform according to the Mosetig-Moorhof plan are said to be safe and efficacious. This method consists in injecting into the gland, with antiseptic precautions, about once a week, from one to four grains of iodoform dissolved in ether and olive oil seven parts each. Five to ten injections are said to be necessary for a cure.

In the cystic variety, Mackenzie recommends puncturing the cyst, drawing off its contents and injecting the sac with a solution of perchloride of iron, ʒ ij. ad ℥ i., which is to be left in for three days; the canula being corked and held in place by a strip of tape passed about the neck. The cork is then removed and, if suppuration has occurred, the cyst should be thoroughly washed several times with an antiseptic solution (*London Lancet*, May 11th, 1872). Obliteration of the sac accompanies the healing process. If the first operation is not successful, it should be repeated until a sufficiently high grade of inflammation has been induced.

Electrolysis is sometimes a very efficient means of curing these cystic growths. It may be practised by inserting into the tumor suitable needles at a distance of an inch or more from each other and passing through them a galvanic current as strong as can be borne by the patient for ten or fifteen minutes at each sitting; to be repeated at intervals of five or ten days until the cyst disappears. If the tumor presses upon the trachea so as to interfere seriously with respiration, tracheotomy should be done and a long, flexible canula introduced and worn while the danger remains. Owing to the success obtained during the last decade, partial extirpation of the gland is an operation which meets with considerable favor among general surgeons. Total extirpation is a dangerous operation, very liable, in those who survive the immediate effects, to be followed by cachexia, strumipriva or myxœdema, therefore it cannot be recommended. The operation itself is fully described in recent works on surgery.

EXOPHTHALMIC GOITRE.

Synonyms.—Graves' disease, Basedow's disease.

Exophthalmic goitre is a disease of the sympathetic nervous system characterized by enlargement of the thyroid gland, prominence of the eyes, disturbance of the action of the heart, and deficient chest expansion, though one or two of these symptoms may be absent. It is fully described in textbooks on practice, and, as stated in the previous edition of this work, it belongs to the domain of the neurologist rather than to the specialist on diseases of the throat and chest. It is mentioned here because the laryngologist is sometimes consulted about it and to call attention to the remarkable effects sometimes exerted upon it by the administration of the tincture of strophanthus, which has proven curative in several reported cases. Daniel R. Brower, of Chicago, has treated three cases by this agent successfully. I have cured two cases by the administration of ten-minim doses of tincture of strophanthus three times daily for a period of several months, combined with repeated injections into the gland of thirty minims of a three to five per cent solution of carbolic acid. In some cases it seems to be of no value.

DISEASES OF THE ŒSOPHAGUS.

ŒSOPHAGITIS.

ACUTE ŒSOPHAGITIS.

Acute œsophagitis is a comparatively rare affection of the mucous membrane lining the œsophagus, characterized by painful deglutition. The inflammation may be either circumscribed or diffused.

Etiology.—Œsophagitis sometimes results from simple exposure to cold, in which case it is generally rheumatic; it may be induced by the use of extremely hot or irritating foods, or by iced drinks, particularly when the subject is warm; it may be caused by irritating medicines, foreign bodies, or the passage of surgical instruments; but most frequently it results from swallowing very hot or corrosive substances. It is sometimes associated with diphtheria, pneumonia, scarlet fever, small-pox, dysentery, cholera, tuberculosis, pyæmia, or cancer.

Symptomatology.—In mild cases there may be simply a sense of constriction in the œsophagus; but in those more severe, pain, which in the acute disease may be increased by pressure, is felt deep beneath the sternum or in the back, between the scapulæ. This pain is experienced upon deglutition even of saliva, and is much aggravated by swallowing solids. Dysphagia or aphagia results from swelling or spasm of the œsophagus during attempted deglutition which may cause

regurgitation of food and vomiting. The vomited matter consists of glairy, sometimes blood-stained mucus, together with the food that has been swallowed. There is fever, with intense thirst, commonly accompanied in children by convulsions. Sometimes involvement of the larynx causes hoarseness, and cough may be produced by the act of swallowing. By auscultation while the patient is swallowing fluid, a peculiar gurgling sound may be heard at the seat of inflammation provided it has caused narrowing of the tube.

Diagnosis.—The diagnosis will depend upon the history, the seat of the pain, the time of its occurrence and the presence of dysphagia.

Prognosis.—In mild cases the disease usually subsides within three or four days; in those more severe it may terminate favorably within a week or ten days, but where there is extensive inflammation the prognosis is grave. When associated with diphtheria or small-pox, it is generally fatal. Phlegmonous inflammation of the œsophagus may cause death within two or three days. Where recovery occurs, the walls of the tube usually remain more or less thickened, and if the inflammation has been severe a stricture results.

Treatment.—In mild cases, demulcents should be employed, and frequent comparatively large doses of bismuth subnitrate are valuable, given in powder and with as little fluid as possible. The food should be liquid. When swallowing is impracticable, food should be given per rectum. In the early stage, the sucking of ice, and the application of cold compresses externally, are useful. In cases resulting from an impacted foreign body, the cause should be removed. In those resulting from the swallowing of acids or alkalies, weak chemical antidotes should be administered in the beginning.

CHRONIC ŒSOPHAGITIS.

A chronic inflammation of the mucous membrane of the œsophagus, with more or less thickening of the walls, is characterized chiefly by difficulty in deglutition.

Etiology.—Chronic œsophagitis usually results from the acute disease, from the excessive use of alcohol, from syphilis, or from impaction of foreign bodies; but it may be due to extension of inflammation from neighboring parts, to pressure of aneurismal or other tumors, or to prolonged congestion occasioned by chronic pulmonary or cardiac affections.

Symptomatology.—The symptoms resemble those of the acute disease, though they are less pronounced.

Diagnosis.—The diagnosis depends upon the history and symptoms. The sounds obtained upon auscultation while the patient is swallowing are apt to be more pronounced than in the acute affection.

Prognosis.—The affection usually extends over a considerable time, and is liable to eventuate in stricture.

Treatment.—The cause should be removed if possible, and any

associated disease should receive appropriate treatment. Locally the use of astringents or stimulants, applied by means of a soft sponge attached to a whalebone, has been found beneficial. For this purpose, solutions of alum, zinc sulphate, or tannin, varying in strength from gr. x. to xxx. ad ℥ i., or silver nitrate gr. v. to x. ad ℥ i., may be employed. Solutions of iodine are also recommended. Any of these in small quantity, not more than ♏ xv. to xx. at a dose, and in weak solution, may be brought in contact with the parts by the act of deglutition. As the inflammation subsides, bougies should be passed at intervals of one or two weeks to prevent the formation of stricture, and in some cases this procedure will be found beneficial for overcoming a persistent low grade of inflammation.

STRICTURE OF THE ŒSOPHAGUS.

Stricture of the œsophagus consists in a narrowing of the tube, occasionally congenital, but generally as the result of injury. It occurs most frequently in children or young adults.

Anatomical and Pathological Characteristics.—The thickening usually involves the mucous membrane and connective tissue, and sometimes the muscular walls also. It occurs oftenest at the upper, narrowest portion of the tube, and next in frequency near the cardiac orifice of the stomach. It varies in degree from slight obstruction to almost complete closure, and rarely involves more than a few inches of the tube; it may be single or multiple, symmetrical or tortuous; the thickening may be uniform about the tube, leaving the opening in its centre, or it may involve only a portion of the walls, leaving the opening at one side. Atrophy of the wall is usually found below the seat of stricture if it is narrow. Collection of food above the stricture causes hypertrophy first, with subsequent fatty degeneration and dilatation. As a result of this weakening and dilatation of the wall and collection of food, not infrequently a large cul-de-sac may be formed above the obstruction.

Etiology.—Stricture is sometimes congenital, but usually it results from acute or chronic inflammation most commonly excited by swallowing of hot water or lye, or the result of rheumatism, syphilis, or cancer.

Symptomatology.—Except in traumatic cases, the symptoms usually come on gradually, the patient at first experiencing some difficulty in swallowing large boluses of solid food. As the obstruction increases and deglutition becomes more and more difficult, solids have to be taken in small boluses and washed down with liquid. Subsequently the diet is necessarily restricted to fluids; and eventually, in extreme cases, even these cannot be swallowed. Sometimes the bolus is regurgitated immediately after it has been taken, perhaps covered with mucus, pus, or blood. When dilatation of the œsophagus has occurred above the stricture the food may be retained for some hours, finally to be regurgitated

more or less decomposed and softened. The patient is usually much depressed and very nervous, and this adds to the tendency to spasm of the œsophagus, which not infrequently takes place during deglutition. Pain at the seat of the stricture is sometimes experienced, and occasionally dyspnœa is complained of; this is especially likely to occur in cancerous strictures involving the recurrent laryngeal nerve. Usually nothing can be discovered by laryngoscopic examination, but by carefully passing œsophageal bougies the location and degree of stricture may be determined.

DIAGNOSIS.—Stricture of the œsophagus is to be distinguished from tubercular laryngitis, from tumors of the pharynx, larynx, or œsophagus, from spasms of the œsophagus, from paralysis of the pharynx and œsophagus, and from the presence of foreign bodies. The diagnosis is not usually difficult; the essential points are the history, and presence of dysphagia, and regurgitation of food. By auscultation the seat of the stricture may frequently be located when the patient is swallowing, owing to the sound caused by the ascent of bubbles of air just above the narrowest portion; but the degree of stricture can only be accurately determined by the passage of the œsophageal bougie. For this purpose, graduated dilators made of the same material as flexible catheters are the safest instruments; but surgeons usually employ an olivary bougie firmly attached to a long whalebone rod. These olivary bougies should be of several sizes, about one and a half inches in length, and conical at both ends; and when the instrument has once passed the stricture, it should be carried down to the stomach to determine whether other strictures exist. Great care should always be used in its passage, for the walls of the œsophagus are often thin and friable or ulcerated, and there is liability of perforation with fatal results. Upon laryngoscopic examination, stricture is readily distinguished from *tubercular laryngitis* and *tumors in the pharynx*. By passage of the bougie, it is distinguished from *tumors* of the *œsophagus*, *spasm*, *paralysis* or *foreign bodies*. It is sometimes difficult to determine whether the stricture is the result of simple chronic catarrhal inflammation, or whether it is of malignant origin, but in advanced life cancerous disease may always be suspected, and a differential diagnosis may usually be made by examination of the regurgitated matter.

PROGNOSIS.—Non-malignant strictures may continue for many years, but those of cancerous origin are always fatal, usually within from eight to eighteen months. Strictures due to simple inflammation, if not too narrow, may often be cured by persistent dilatation; if not relieved, they tend to interfere more and more with nutrition, and finally, sometimes after many years, they may cause death by inanition. Occasionally death is the result of abscess caused by the pressure of food in the dilatation above the stricture, or of tubercular degeneration, or gangrene resulting from the reduced condition of the system. Pressure upon the

recurrent nerve sometimes causes paralysis of the abductor muscles of the vocal cords, with dangerous or even fatal dyspnœa unless tracheotomy is promptly performed. Ulceration may occur into the trachea, the bronchial tubes, or into one of the adjacent large vessels.

TREATMENT.—When resulting from chronic catarrhal inflammation, rheumatism, or syphilis, the administration of the iodides is occasionally followed by relief. In malignant cases, opiates must be given to relieve pain. When food in sufficient quantity cannot be taken, nutritive enemata must be employed. Dilatation is indicated in all suitable cases. In those of malignant nature it must be practised, if at all, with the greatest care, but as a rule it is inadvisable.

Charters J. Symonds, of London, in seventeen cases of malignant stricture of the œsophagus, has successfully used, for keeping the stricture pervious, a gum elastic tube four to six inches long (*London Lancet*, March, April, 1889). This is funnel-shaped above and closed at its lower end, but has an opening just above the closed extremity like an ordinary catheter. This tube is introduced through the stricture, upon a whalebone staff, and has attached to its upper end a strong silk thread which is fastened to the ear. It may be left *in situ* for weeks or months, allowing the passage of liquid food, without hast-

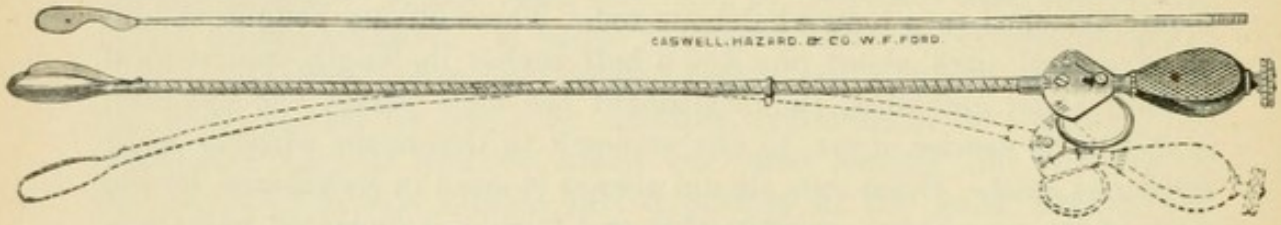

FIG. 238.—SANDS' ŒSOPHAGOTOME.

ening the inevitable progress of the disease. In other cases dilatation should be attempted by the graduated bougies already described, and the operation should be repeated every two, three, or four days according to the amount of irritation produced, time always being allowed for this to subside before the next operation. When an instrument has been passed, it should be allowed to remain for a few seconds, as long as the patient can tolerate it, and then withdrawn and followed by one of a size larger. Thus the largest instrument that can be passed without great force should be used at each sitting; at the next an instrument a size smaller than the one previously introduced should be first used followed by one or two larger sizes. If the dilatation proves successful, bougies should be introduced from time to time with diminishing frequency, and the patient should be taught to perform the operation himself, which must be repeated at intervals for several months or possibly years, the cure usually requiring a treatment for at least six to eighteen months. When the stricture is very narrow, an œsophagotome (Fig. 238) may be employed for incising the mucous

membrane to allow of more rapid and permanent dilatation. The bulb is to be introduced beyond the stricture, the knife slightly protruded, and the instrument withdrawn. The operation is attended by great danger, and is liable to be a direct cause of death in about thirty-five per cent of the cases operated upon. If this operation is adopted, two or three slight incisions should be made at different parts of the stricture, gradual dilatation being practised subsequently. External œsophagotomy and gastrotomy are recommended in special cases, but they come more properly within the domain of general surgery. Electrolysis has also been recommended in the treatment of stricture, but the close proximity of the œsophagus to the vagus nerve renders it hazardous. A. Fort, of Paris, has practised it successfully in several instances, and appears to have obtained considerable benefit even in malignant cases.

COMPRESSION OF THE ŒSOPHAGUS.

Compression of the œsophagus results from the pressure of mediastinal tumors, which may be carcinomatous, aneurismal, or purulent. It is sometimes caused by enlargement of the bronchial or thyroid glands, and may be occasioned by pressure of the fluid in pericarditis. It is to be distinguished from true stricture, by the process of exclusion. The prognosis and treatment will depend upon the etiology.

SPASM OF THE ŒSOPHAGUS.

Synonyms.—Cramp of the œsophagus, œsophagismus, spasmodic stricture.

Spasmodic contraction of the œsophagus is sometimes associated with a similar condition of the pharynx. It is characterized by paroxysmal inability to swallow, which may come on suddenly and as speedily disappear; or it may continue for several hours or at irregular intervals for days or weeks. It is most frequently seen in nervous women, but is said to occur at all ages, and judging from my own experience it is not infrequent in men past middle life. It may be associated with disease of the œsophagus, but is usually independent of it.

Etiology.—The attacks are sometimes caused by attempts to swallow certain kinds of food, but they are often brought on by solid food of any kind, and not infrequently even by fluids. The affection is attributed by Cohen to rheumatism, to acute disease of the stomach, heart, lungs, uterus, brain, or spinal cord, and to hysteria and hydrophobia (Diseases of the Throat).

Symptomatology.—In many instances the spasm comes on suddenly and may as speedily disappear, but in others the constriction remains, or at least the patient supposes it to remain, for many hours or even days, so that he is afraid to swallow food. When sudden, it is usually

followed by prompt regurgitation of any food that the patient attempts to swallow, and sometimes by spasm of the air passages, palpitation of the heart or syncope. The difficulty is usually intermittent, but occasionally, as before mentioned, the constriction remains for many hours; indeed, when occurring in a low position, it sometimes continues so long that food may be regurgitated in a softened and decomposing condition some hours after it has been swallowed, owing to the occurrence of dilatation in the œsophagus above the constriction. The seat of the difficulty may be referred by the patient to any portion of the œsophagus.

DIAGNOSIS.—The diagnosis is based upon the intermittent character of the dysphagia, and exploration with œsophageal bougies, the passage of which is not often greatly hindered by the spasmodic contraction. It is most likely to be confounded with organic stricture or paralysis of the œsophagus. It is distinguished from *organic stricture* by the history and the ready passage of the bougie. It is distinguished from *paralysis* by the history, paralysis usually following diphtheria; by the sudden regurgitation of food, which often takes place in spasm but is not common in paralysis; by its intermittent character; and by the introduction of the bougie, which passes readily in paralysis, and is more or less obstructed in spasmodic stricture.

PROGNOSIS.—The spasm is usually transient, and the liability to recurrence may disappear after a few days or weeks; but in some instances it continues for a long time, and I have seen patients who have been unable to swallow satisfactorily for three or four years.

TREATMENT.—Anti-spasmodics, as bromides, camphor, valerian, and asafœtida, are frequently of benefit, and in most instances such tonics as iron, quinine, strychnine, and arsenious acid are necessary; but the repeated passage of an œsophageal bougie will give more relief than any other measure. Usually it is necessary to repeat the operation only three or four times.

Borgiotti reports a case of œsophageal spasm in a woman thirty-one years old, which continued uninterruptedly for five hundred and thirty days, rarely permitting the passage of the sound or liquid food. Cure was effected within a few days by the use of Verneuil's œsophageal dilator (*Centralblatt für klinische Medicin*, 1888).

PARALYSIS OF THE ŒSOPHAGUS.

Paralysis of the œsophagus consists of loss of muscular power, characterized by difficulty in deglutition. It is said to be very common in the insane, and it is comparatively frequent in old age or in those broken down by poor health, and also as a sequel of diphtheria.

ANATOMICAL AND PATHOLOGICAL CHARACTERISTICS.—The lesions may consist of changes at the nerve centres, such as hemorrhage into the pons or the medulla, or tumors of these organs, bulbar pa-

ralysis, multiple sclerosis, cerebral atrophy, and progressive locomotor ataxia; or of pressure upon the nerve as in tubercular enlargement of the pharyngeal lymphatic glands, or syphilitic enlargement of the cervical vertebræ; or there may be simple muscular weakness without nervous lesions, as observed in the feeble or aged.

ETIOLOGY.—The most common causes are diphtheria, and simple muscular weakness from old age or ill health. The affection is occasionally caused by syphilis, tuberculosis, lead poisoning, acute fever, and hysteria. Inability to swallow is usually observed in approaching dissolution some time before failure of respiration and circulation.

SYMPTOMATOLOGY.—The essential symptom is difficulty in swallowing, which may develop quickly or slowly according to the cause. It is probable that complete aphagia is never present unless the pharynx is paralyzed at the same time. When due to hemorrhage into the nerve centres, it comes on suddenly, and is at once complete; but if resulting from tumors, it develops gradually. Following diphtheria, it usually appears within three or four weeks after the beginning of the attack, and may reach its full intensity in three or four days. As the result of nervous diseases it is a rare affection, and in any case seldom appears until late in their course. When of central origin, it is sometimes associated with more or less paralysis of the sensory or motor nerves of the larynx. In local paralysis, the affection comes on gradually; Mackenzie states that he has seen several instances in which the disease has lasted from ten to twenty years, that it apparently leads after a time to some stenosis of the gullet, and that in long-standing cases the isthmus faucium, and even the mouth, is often much contracted (Diseases of the Throat and Nose, Vol. II).

Patients are commonly very weak, but emaciation is not usually a marked symptom excepting in cases of long duration. There is seldom any regurgitation of food, though in mild cases patients complain of its lodging in the œsophagus. The sound, which may be heard during deglutition over the normal œsophagus is greatly altered or may be suppressed by paralysis, so that, instead of being distinct as in health, only a trickling or dropping can be heard. A bougie may be passed easily and is less likely to cause nausea than in health, but occasionally, in cases of long standing, contraction of the gullet is said to occur, causing much difficulty in passing the instrument.

DIAGNOSIS.—Paralysis is to be distinguished from spasm and from malignant diseases.

Paralysis is distinguished from spasm of the œsophagus as follows:

PARALYSIS OF THE ŒSOPHAGUS.	SPASM OF THE ŒSOPHAGUS.
Most common in advanced life and in feeble patients.	Most frequent in the young and hysterical subjects.
Dysphagia continuous.	Dysphagia intermittent.

Paralysis of the œsophagus.	Spasm of the œsophagus.
Seldom any regurgitation of food.	Regurgitation of food common
Bougie passed easily, except in rare cases of long standing.	At times impossible to pass bougie.
No distinct sound produced by swallowing.	Sharp sound heard over œsophagus during deglutition.

We find that *malignant disease* of the œsophagus causes difficulty in deglutition, and, like paralysis, generally occurs in advanced life, but it is attended by pain, regurgitation of food, and constant obstruction to the passage of the bougie.

Prognosis.—When depending upon muscular weakness, diphtheria, or lead poisoning, the prognosis is very favorable, but if due to lesions of the nervous system it is grave.

Treatment.—In the severe forms, little can be accomplished in the way of treatment. In any case where the cause can be found it should be removed if possible. Usually iron, quinine, and strychnine, especially the latter, are important agents, together with a stimulating diet. Mackenzie recommends faradization of the œsophagus once or twice daily, preferably before meals. The positive pole should be placed by means of the necklet in contact with the spinous processes of the upper cervical vertebræ, the negative attached to the œsophageal electrode, which should be introduced three or four times at each sitting, and retained for a few seconds. It is sometimes desirable to feed the patient through an œsophageal tube; especially is this necessary if the pharynx and larynx are also paralyzed.

FOREIGN BODIES IN THE ŒSOPHAGUS.

Foreign bodies, of great variety, may become impacted in the œsophagus, where they interfere with respiration and deglutition. They generally lodge either in the lower portion of the pharynx or just below it or at the upper portion of the œsophagus directly behind the cricoid cartilage, but sometimes they pass lower and occlude the passage opposite the bifurcation of the trachea or just above the cardiac orifice of the stomach. The most common of these foreign bodies are large boluses of food, coins, pins, fragments of bone, and plates with false teeth.

Symptomatology.—When foreign bodies are large and lodged in the lower part of the pharynx, they may depress the epiglottis so as to cause immediate suffocation. Large bodies may provoke retching or vomiting, and prevent swallowing either of solids or fluids. Smaller bodies usually cause actual pain or pricking sensations, sometimes slight bleeding, and frequently interfere with the swallowing of solids, but not with swallowing of liquid. Sharp, irregular bodies cause pain and inflam-

mation. Large or irregular bodies may cause cough, spasm of the glottis, aphonia, or asphyxia. The respiration may be impeded by involution of the trachea or by spasm.

DIAGNOSIS.—The presence of foreign bodies is to be distinguished from globus hystericus and from paræsthesia of the œsophagus. The essential features in the diagnosis are the history, laryngoscopic examination, and exploration with the bougie. By inspection, *affections of the pharynx and larynx* may be excluded; and sometimes, in the case of irregular bodies, blood or pus may be detected at the œsophageal entrance. Exploration with the finger will sometimes detect a foreign substance, and passage of the œsophageal bougie will usually locate the object unless small; but in some cases spasm of the œsophagus above or below the foreign substance seriously interferes with this examination. Care must be taken not to be misled by the dense pharyngo-epiglottic ligament and normal narrowing at the entrance of the œsophagus. Foreign bodies will be distinguished from *globus hystericus* by the history, by the presence of other symptoms of hysteria, by frequent change in location of the sensations in the nervous affection, and by exploration with the bougie. From *paræsthesia of the œsophagus*, where the patient's sensations indicate the presence of a foreign body, and where the history generally points to an accident of this kind, the diagnosis can only be made by careful exploration with the bougie and extractor.

PROGNOSIS.—The lodgement of a foreign body often proves immediately fatal from suffocation. Sometimes comparatively smooth objects have remained in the œsophagus for months or years and then been removed or spontaneously discharged, but as a rule there is danger so long as a foreign body remains impacted in the œsophagus, since it is apt to set up inflammation which may be followed by abscess; or the pressure may cause ulceration and opening into the mediastinum, the trachea, or the aorta. Impacted bodies sometimes work their way to

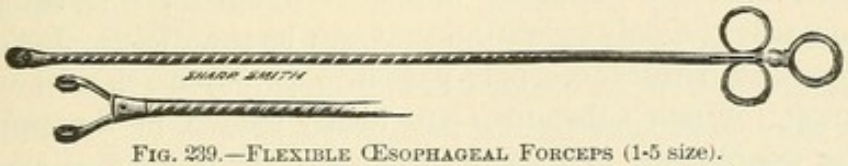

FIG. 239.—FLEXIBLE ŒSOPHAGEAL FORCEPS (1-5 size).

the surface and may be discharged without immediate danger, but in this way they may give rise to a fistula. Sometimes they cause inflammation and caries of the vertebræ, or secondary disease of the lungs, pericardium, or other organs. Perforation of the œsophagus usually leads to emphysema of the neck, and commonly proves fatal. Great injury is sometimes unavoidably inflicted in withdrawing these substances.

Repetition of the accident is observed in some people in consequence of spasm of the constrictor muscles of the œsophagus or of partial paral-

ysis; but in such cases the obstructing bolus may generally be carried on by the swallowing of another bit of food or a drink of water.

TREATMENT.—Prompt removal of the body is desirable in all instances. If not too large, it may be speedily removed by an emetic, for which purpose apomorphine, gr. $\frac{1}{10}$, injected subcutaneously, may be effectually employed. If the foreign body can be seen or felt, it may sometimes be removed by the finger, blunt hook, or forceps. Even when lower, it may often be caught with flexible œsophageal forceps (Fig. 239) or with the bristle extractor (Fig. 240) or the coin-catcher.

In several instances Créquy has succeeded in removing foreign bodies by having the patient swallow a well lubricated tangled skein of thread with a long stout thread tied to its centre; traction is made upon the thread when the bundle has had time to pass the obstruction (*Gazette des Hôpitaux*, 1870, No. 56).

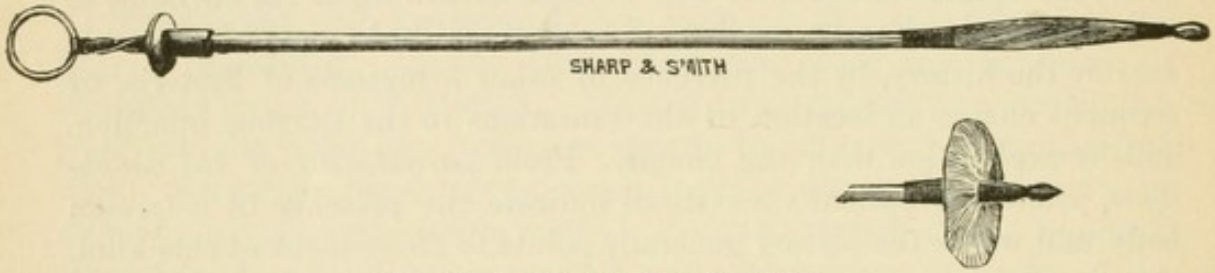

FIG. 240.—BRISTLE EXTRACTOR (½ size).

B. Polikier, of Warsaw (*Revue mensuelle des maladies de l'enfance*, Paris, 1892), reports two cases in which he succeeded in removing foreign bodies from the œsophagus in children, by a sort of massage upward and backward with the finger pressed down between the trachea and sterno-cleido-mastoid muscle; while with the other hand he tickled the child's throat until it vomited and brought up the foreign body.

When susceptible of digestion, there is no objection to pushing the foreign body into the stomach, care being used to avoid injuring the œsophagus; and if the offending object be lodged low in the passage, this is frequently the only operation that can be practiced. Fortunately many indigestible substances may pass into the stomach without harm to the patient. When substances are firmly lodged in the upper portion of the œsophagus, and cause distressing or dangerous symptoms, laryngotomy or œsophagotomy must be performed. These operations, which are fully described in textbooks on general surgery, not infrequently give good results.

PARÆSTHESIA OF THE ŒSOPHAGUS.

Paræsthesia is a nervous affection in which the patient fancies some foreign body lodged in the pharynx or œsophagus. It usually occurs in women of enfeebled health, with nervous temperament, or in hysteri-

cal subjects. There are no anatomical changes in the parts, but the patient fancies she is unable to swallow solids, or she is unwilling to attempt it perhaps from a vague fear of choking.

ETIOLOGY.—Some of the cases are neuralgic in character, others hysterical; some depend upon derangements of the digestive system or genito-urinary tract; others upon a small ulcer or fissure in the pharynx or œsophagus; but most frequently the condition is due to something which has lodged for a time in the œsophagus, or, having inflicted injury, has subsequently passed on through the alimentary canal. Pins, tacks, fishbones, and other small, sharp objects are most likely to leave this sensation.

SYMPTOMATOLOGY.—There is usually a history of something swallowed, which has apparently lodged in some part of the throat or œsophagus, giving rise to pricking sensations, or soreness, fulness, pressure, or weight, which seems to the patient clearly to indicate the presence of a foreign body. The seat of the fancied object frequently changes by deglutition or efforts made by the patient or physician to remove it; and although in many instances the patient readily swallows large, solid morsels, she cannot be convinced that these would necessarily carry the object with them. Inspection of the pharynx and mouth of the œsophagus will sometimes disclose a small fissure or ulcer which gives rise to the sensation, but usually it only reveals to the physician a normal condition of the parts.

DIAGNOSIS.—One of the most valuable points in the diagnosis is a changeableness of the fancied position of the object. The patient is often found to be anæmic, debilitated, and nervous, frequently able to swallow without much difficulty; but the diagnosis must finally be decided by passage of the œsophageal bougie, or an extractor, by which foreign bodies can be felt or removed.

PROGNOSIS.—The sensations often continue weeks or months, and in some cases it is impossible to convince the patient that the sensations are altogether nervous.

TREATMENT.—Cases depending upon ulceration or fissure are usually best relieved by the application of solutions of silver nitrate or the mineral acids. Those resulting from having swallowed some substance are often cured by the passage of the bougie or of the bristle extractor, thus demonstrating to the patient that nothing can be lodged in the œsophagus. Those of purely nervous origin are best relieved by the same means, together with the internal administration of iron, quinine, strychnine, arsenious acid, and the bromides.

APPENDIX.

FORMULÆ FOR PRESCRIPTIONS.

SEVERAL of the formulæ relating to diseases of the throat and nasal passages are taken from the Pharmacopœia of the Hospital for Diseases of the Throat, London. The various mixtures, excepting Formula 3, which would not be prescribed in quantities of less than four ounces, have been reduced to the standard of one ounce; prescriptions for drugs to be given in pill form contain quantities sufficient for one pill.

1. ℞ Morphinæ sulphatis . . . . . gr. i.
   Antimonii et potass. tart. . . . . gr. i.
   Ammonii chloridi . . . . . ʒ i.
   Ext. grindeliæ robustæ fluidi . . . . . fl. ʒ iv.
   Syrupi pruni virginianæ et
   Misturæ glycyrrhizæ comp. . . . . āā fl. ʒ ij.
   M. S. Teaspoonful, for cough. *Especially useful in acute bronchitis.*

2. ℞ Morphinæ sulphatis . . . . . gr. i.
   Chloralis . . . . . ʒ i.
   Syrupi zingiberis . . . . . ʒ iv.
   Misturæ glycyrrhizæ . . . . . ad fl. ℥ i.
   M. S. Teaspoonful every half-hour until relieved. *For spasmodic asthma.*

3. *Emulsion of Cod-Liver Oil.*
   ℞ Olei morrhuæ . . . . . ℥ ij.
   Sacchari . . . . . ʒ vi.
   Acaciæ . . . . . ʒ iv.
   Olei gaultheriæ . . . . . ♏ xv.
   Aquæ . . . . . q.s. ad fl. ℥ iv.

Triturate the sugar and acacia thoroughly with one-half the amount of water until a uniform mucilage is formed; then add the oil slowly, with constant trituration, and subsequently add the remainder of the water. It requires about an hour to make the perfect emulsion, to which may be added lacto-phosphate of calcium or phosphoric acid, which will give it an agreeable acidulous taste. Chloride of calcium may be added when desired, but the lactophosphate of calcium is much more agreeable to the taste and answers a similar remedial purpose.

4. ℞ Potassii bromidi . . . . . gr. xl.
   Syrupi lactucarii (Aubergier's)
   Syrupi acidi hydriodici . . . . . āā ʒ iv.
   M. S. Teaspoonful every four to six hours. *A most useful cough medicine for protracted bronchitis in children.*

5. ℞ Morphinæ sulphatis . . . . . gr. i.
Ammonii carbonatis . . . . . gr. xxx.-xl.
Syrupi pruni virginianæ
Misturæ glycyrrhizæ comp. . . . . āā fl. ʒ iv.
M. S. Teaspoonful in water, for cough. *A most useful cough syrup when opiates are not contra-indicated.*

6. *Pil. Can. Ind., Hyoscyam., et Quininæ Comp.* (No. 1).
℞ Ext. can. Ind. (Allen's) . . . . . gr. $\frac{1}{8}$
Ext. nucis vom. . . . . gr. $\frac{1}{8}$*
Ext. hyoscyam. (alcoholic) . . . . . gr. $\frac{1}{2}$
Camphoræ. . . . . gr. i.
Quininæ muriate . . . . . gr. iss.
M. S. Before meals and at bed-time.

7. *Pil. Can. Ind., Hyoscyam., et Quininæ Comp.* (No. 2).
℞ Ext. can. Ind. (Allen's) . . . . . gr. $\frac{1}{8}$
Ext. nucis vom. . . . . gr. $\frac{1}{8}$
Ext. hyoscyam. (alcoholic) . . . . . gr. i.
Creasoti . . . . . ♏ i.
Dextro-quininæ . . . . . gr. ij.
M. S. Before meals and at bed-time.

8. *Pil. Capsicum, Hydrastine, Papain Comp.*
℞ Oleoresinæ capsici . . . . . ♏ $\frac{1}{24}$
Ext. nucis vom. . . . . gr. $\frac{1}{4}$
Hydrastine muriate . . . . . gr. $\frac{1}{8}$
Papain (Carica papaya) . . . . . gr. iij.
Acidi salicylici . . . . . gr. i.
M. S. After meals.

9. *Iodol Ointment.*
℞ Acidi carbolici . . . . . ♏ vi.
Olei rosæ . . . . . ♏ v.
Iodol . . . . . gr. xxv.
Lanolini . . . . . ʒ ss.
M. S. *A valuable ointment for healing abrasions of the nostril and upper lip and for healing erosions of the septum.*

10. ℞ Antimonii et potassii tartratis . . . . . gr. xx.
Cantharidis et
Olei tiglii . . . . . āā gr. xl.
Camphoræ et
Ext. stramonii (aqueous) . . . . . āā gr. lxxx.
Adipis . . . . . ʒ iiss.
Cerati simplicis . . . . . ad ʒ i.
M. S. Counter-irritant ointment.

11. ℞ Tincturæ iodi . . . . . ʒ ss.-ʒ i.
Potassii iodidi . . . . . gr. x.-xx.
Aquæ . . . . . ad fl. ʒ i.
M. S. Use as an injection, which should be withdrawn in about five minutes. *For chronic pleurisy.*

GARGLES.

Gargles are only useful in diseases of the fauces. They cannot affect the nasal passages, lower pharynx, or larynx. The preparations may be sedative, astringent, stimulant, or antiseptic.

SEDATIVES.

12. ℞ Potassii bromidi . . . . . gr. xxx. ad fl. ℥ i.

13. ℞ Potassii nitratis . . . . . gr. xx.
Potassii chloratis . . . . . gr. xx.
Aquæ ferventis . . . . . ad fl. ℥ i.
M. S. Use as hot as it can be borne.

ASTRINGENTS.

14. ℞ Acidi tannici . . . . . gr. xij.–℥ ij. ad fl. ℥ i.

15. ℞ Aluminis . . . . . gr. viij. ad fl. ℥ i.

16. ℞ Ferri et ammonii sulphatis . . . gr. viij. ad fl. ℥ i.

17. ℞ Sodii boratis . . . . . gr. xxv.
Glycerinæ . . . . . ♏ xxv.
Tincturæ myrrhæ . . . . . ♏ xxv.
Aquæ . . . . . ad fl. ℥ i.
M.

STIMULANTS.

18. ℞ Acidi acetici dil. . . . . ♏ xv.
Glycerinæ . . . . . ♏ xviij.
Aquæ . . . . . ad fl. ℥ i.
M.

19. ℞ Acidi carbolici . . . . . gr. ij.–x. ad fl. ℥ i.

20. ℞ Potassii chloratis . . . . . gr. x.–xxv. ad fl. ℥ i.

ANTISEPTICS.

21. ℞ Acidi carbolici vel.
Potassii chloratis (see Stimulants 19 and 20).

22. ℞ Potassii permanganatis . . . . . gr. ij.–iv. ad fl. ℥ i.

23. ℞ Hydrargyri chloridi corrosivi . . . gr. ¼–gr. ss. ad fl. ℥ i.

24. ℞ Aquæ cinnamomi . . . . . q.s.

TROCHISCI OR LOZENGES.

Each lozenge contains seventy to eighty per cent of red-currant fruit paste, one to two per cent of powdered tragacanth, four per cent of sugar, and a varying quantity of the medicament according to the following formulæ:

SEDATIVES.

25. Troch. morphinæ sulphatis . . . . gr. $\frac{1}{30}$ ad troch.

26. Troch. ext. opii . . . . . gr. $\frac{1}{10}$ " "

27. Troch. sodii boratis . . . . . gr. iij. " "

28. Troch. ammonii chloridi . . . . . gr. ij. " "

29. Troch. lactucarii (Aubergier's)

S. One every half-hour or hour as needed. *These are very pleasant to take and efficient in mild cases.*

30. Troch. chlorodyne . . . . . ♏ v. ad troch.

31. *Troch. Lobeliæ Compound.*
℞ Ammonii chloridi . . . . . gr. i.
Ext. lobeliæ . . . . . gr. $\frac{1}{10}$
Ext. glycyrrhizæ . . . . . gr. i.
Codeinæ . . . . . gr. $\frac{1}{20}$ ad troch.

32. *Troch. Morphia, Antimony et Ipecac Compound.*
℞ Morphinæ hydrochloratis . . . . . gr. $\frac{1}{33}$
Antimonii sulph. . . . . gr. $\frac{1}{40}$
Pulv. ipecac. . . . . gr. $\frac{1}{20}$
Olei sassafras
Balsam tolu
Ext. glycyr., acaciæ et sacch. alb. . . . āā q.s. ad troch.

33. *Troch. Terpin Hydrate and Cannabis Compound.*
℞ Terpin hydrate . . . . . gr. ij.
Ext. can. ind. . . . . gr. $\frac{1}{20}$
Codeinæ . . . . . gr. $\frac{1}{8}$
Ol. menth. pip. . . . . ♏ $\frac{1}{5}$
Sacch. . . . . gr. iij.

34. ℞ *Troch. Mist. Glycyrrhizæ Compound.*
Same as mist. glycyrrhizæ comp., U. S. P.

35. *Troch. Opii et Anisi Compound.*
℞ Pulv. opii . . . . . gr. $\frac{1}{10}$
Olei anisi, ext. glycyrrhi. æ, acaciæ, et sacch. alb. q.s. ad troch.

DEMULCENTS.

36. *Troch. Altheæ.*
℞ Altheæ, acaciæ, et sacch. alb.

37. *Troch. Ulmi.*
℞ Mucil. ulmi cort., albumen ovi, acaciæ
Sacch. alb., ... āā q.s. ad troch.

ASTRINGENTS.

38. ℞ Krameriæ . . . . . gr. iij. ad troch.

39. ℞ Kino . . . . . gr. ij. " "

40. ℞ Acidi tannici . . . . . gr. iss. " "

41. *Troch. Krameriæ Compound.*
℞ Pulv. cubebæ . . . . . gr. $\frac{1}{4}$
Ext. krameriæ . . . . . gr. i.
Potassii chloratis . . . . . gr. ij. ad troch.

STIMULANTS.

42. ℞ Acidi benzoici . . . . . gr. iij. ad troch.

43. ℞ Cubebæ . . . . . gr. ss. " "

44. ℞ Guaiaci . . . . . gr. ij.-iij. " "

45. ℞ Pyrethri . . . . . gr. i. " "

46. *Troch. Acid Benzoic Compound.*

℞ Pulv. cubebæ . . . . . gr. ¼
Acidi benzoici . . . . . gr. ½
Potassii chloratis . . . . . gr. ij. ad troch.

47. *Troch. Cubeb and Potassium Chlorate.*

℞ Cubebæ . . . . . gr. ⅛
Potassii chloratis . . . . . gr. iij. ad troch.

48. *Troch. Ammonium Compound.*

℞ Ext. glycyrrhizæ . . . . . gr. ½
Cubebæ . . . . . gr. ½
Pulv. ulmi cort. . . . . gr. i.
Ammonii chloridi . . . . . gr. iij.
Acaciæ et sacch. alb. . . . . q.s. ad troch.

49. *Guaiac and Ammonium Compound.*

℞ Ammonii chloridi . . . . . gr. i.
Guaiaci resinæ . . . . . gr. i.
Potassii chloratis . . . . . gr. ij. ad. troch.

Potassium chlorate is more pleasant and more efficacious in compressed pills than in troches.

ANTISEPTICS.

50. ℞ Acidi carbolici . . . . . gr. i. ad troch.

51. ℞ Potassii chloratis (see Stimulants 19, 20).

VAPOR INHALATIONS.

Mackenzie's eclectic inhaler is the most complete, but some of the cheaper instruments will answer the same purpose. An inhaler which is in common use consists of a glass flask holding about a quart. This has a perforated cork, through which two glass tubes are passed, one to the bottom of the flask to admit the air, and the other, through which the patient inhales the vapor, into its upper part. In the absence of an inhaler an earthen teapot may be employed. I sometimes place the medicine in a pint of water in a small tin pan which is then covered by a cone of paper from the top of which the patient inhales. The inhalations are prepared by adding a teaspoonful of the medicated solution to a pint of water, at a temperature of about 150° F. or as indicated by the formula. They should be used morning and evening for about five minutes each time, six respirations being taken per minute.

The oleaginous or balsamic remedies should be rubbed up with light carbonate of magnesium, in order to maintain their suspension in the water, as shown in the following formula:

52. ℞ Olei cajuputi . . . . . ♏ viij.
Mag. carb. lev. . . . . gr. v.
Aquæ . . . . . ad fl. ℥ i.
M. S. A teaspoonful in a pint of water at 150° F., for each inhalation.

The vapors may be sedative, antispasmodic, antiseptic, or gently or strongly stimulant.

SEDATIVES.

53. ℞ Ætheris et alcoholis, ... āā
54. ℞ Chloroformi et alcoholis . . . . . āā
55. ℞ Lupulinæ . . . . . gr. xxx.
56. ℞ Ext. belladonnæ vel
Ext. stramonii . . . . . gr. v. ad fl. ℥ i.
57. ℞ Ext. opii . . . . . gr. v. ad fl. ℥ i.
58. ℞ Tinct. benzoini comp. . . . . fl. ℥ i.
59. ℞ Tinct. opii camph. . . . . fl. ℥ i.

ANTISPASMODICS.

60. ℞ Ætheris vel chloroformi (as in 53, 54).
61. ℞ Amyl nitritis . . . . . ♏ viij. ad fl. ℥ i.

MILD STIMULANTS.

62. ℞ Olei pini sylvestris . . . . . ♏ xl. ad fl. ℥ i.
63. ℞ Olei cubebæ . . . . . ℥ ss. ad fl. ℥ i.
64. ℞ Olei cassiæ . . . . . ♏ vi.
Olei limonis . . . . . ♏ x. ad fl. ℥ i.
M.

65. ℞ Olei anisi . . . . . ♏ vi. ad fl. ℥ i.

66. ℞ Olei myrti . . . . . ♏ vi.
Camphoræ . . . . . gr. v. ad fl. ℥ i.
M.

67. ℞ Terebene . . . . . ℥ i.
Alcoholis . . . . . ℥ i.
M.

68. *More stimulating than the above, and antiseptic.*
℞ Acidi carbolici . . . . . gr. xx. ad fl. ℥ i.

69. ℞ Creasoti . . . . . ♏ xl. ad fl. ℥ i.
70. ℞ Olei cari . . . . . ♏ vi. ad fl. ℥ i.
71. ℞ Olei juniperi . . . . . ♏ xx. ad fl. ℥ i.

72. ℞ Acidi carbolici . . . . . gr. xxx.
Ammonii chloridi . . . . . gr. xxx.
Glycerinæ . . . . . ℥ i.
Aquæ dest . . . . . ℥ i.
M.

73. ℞ Tinct. iodi comp. . . . . ♏ v.
Glycerinæ . . . . . ℥ i.
Aquæ dest. . . . . ℥ vij.
M.

74. ℞ Creasoti . . . . . ℥ ss.
Glycerinæ . . . . . ℥ ij.
Aquæ dest. . . . . q.s. ad ℥ i.
M.

75. ℞ Hydrargyri chloridi corrosiv. . . . . $\frac{1}{1000}$
Glycerinæ . . . . . ʒ ij.
Aquæ dest. . . . . ℥ i.
M.

STRONG STIMULANTS.

76. ℞ Olei calami arom. . . . . ♏ v. ad fl. ℥ i.

77. ℞ Olei caryophylli . . . . . ♏ x. ad fl. ℥ i.

78. ℞ Tinct. iodi comp. . . . . ♏ x.
S. Repeat two or three times at each inhalation.

79. ℞ Aquæ ammoniæ et aquæ . . . . . āā fl. ʒ iv.

SPRAY INHALATIONS.

Spray inhalations are to be used by the physician or patient in full strength, with the compressed-air atomizer; the aqueous solutions may be used in about double strength by the steam atomizer. These applications are useful principally in treating diseases of the fauces and of the nasal cavities. It is almost impossible for the patient to draw them into the larynx. The inhalations may be classified as sedatives, astringents and stimulants, hæmostatics, and antiseptics.

SEDATIVES.

80. ℞ Potassii bromidi . . . . . gr. xx. ad fl. ℥ i.

81. ℞ Cocainæ hydrochloratis . . . . gr. xl. to lx. ad fl. ℥ i.
M.

82. ℞ Ext. pinus canadensis dest. . . . . ʒ ss.
Olei geranii . . . . . ♏ iv.
Olei petrolinæ vel liquid albolene . . q.s. ad fl. ℥ i.
M.

83. ℞ Antipyrini . . . . . gr. x.
Zinci sulph. . . . . gr. ij.
Ext. hamamelidis . . . . . ʒ i.
Aquæ dest. . . . . q.s. ad ℥ i.
M.

84. ℞ Acidi carbolici . . . . . gr. iiss.
Mentholis . . . . . gr. v.
Liquid albolene . . . . . ℥ i.
M.

85. ℞ Acidi hydrocyanici dil. . . . . ʒ ss. ad fl. ℥ i.
To be used only as a cold spray.

86. ℞ Acidi carbolici . . . . . gr. i.
Sodii boratis
Sodii bicarb. . . . . āā gr. ij.
Glycerinæ . . . . . ʒ i.
Aquæ dest. . . . . q.s. ad ℥ i.
M.

87. ℞ Olei petrolinæ vel liquid albolene.

ASTRINGENTS AND STIMULANTS.

88. ℞ Acidi tannici . . . . . gr. iij. ad fl. ℥ i.

89. ℞ Zinci sulphatis . . . . . gr. ij.–x. ad fl. ℥ i.

90. ℞ Zinci chloridi . . . . . gr. ij.–x. ad fl. ℥ i.

91. ℞ Aluminis . . . . . gr. x. ad fl. ℥ i.

92. ℞ Ferri perchloridi . . . . . gr. iij. ad fl. ℥ i.

93. ℞ Morph. sulph. . . . . gr. iv.
Acidi tannici
Acidi carbolici . . . . . āā gr. xxx.
Glycerinæ
Aquæ dest. . . . . āā fl. ℥ ss.
M.

94. ℞ Acidi tartarici . . . . . gr. i.
Acidi carbolici
Zinci sulph. . . . . āā gr. ij.
Aquæ dest. . . . . fl. ℥ i.
M.

95. ℞ Acidi tartarici . . . . . gr. ij.
Zinci sulph. . . . . gr. xv.
Aquæ dest. . . . . fl. ℥ i.
M.

96. ℞ Acidi tartarici . . . . . gr. iij.
Zinci sulph. . . . . gr. xxx.
Aquæ dest. . . . . fl. ℥ i.
M.

97. ℞ Acidi tartarici . . . . . gr. ij.
Zinci chloridi . . . . . gr. xv.
Aquæ dest. . . . . fl. ℥ i.
M.

98. ℞ Acidi tartarici . . . . . gr. iij.
Zinci chloridi . . . . . gr. xxx.
Glycerinæ . . . . . ℥ iij.
Aquæ dest. . . . . fl. ℥ i.
M.

99. ℞ Ext. hamamelidis dest.

100. ℞ Acidi carbolici . . . . . gr. xl.
Glycerinæ . . . . . ℥ i.
Aquæ dest. . . . . fl. ℥ i.
M.

101. ℞ Cupri sulphatis . . . . . gr. x.
Aquæ dest. . . . . fl. ℥ i.
M.

102. ℞ Cupri sulphatis . . . . . gr. xx.
Aquæ dest. . . . . fl. ℥ i.
M.

103. ℞ Acidi carbolici . . . . . gr. xxx.
Ext. pinus canadensis dest. . . . . ♏ xx.
Liquid albolene . . . . q. s. ad fl. ℥ i.
M.

104. ℞ Acidi carbolici . . . . . gr. ijss.
Mentholis . . . . . gr. v.
Liquid albolene . . . . . fl. ℥ i.
M.

105. ℞ Acidi carbolici . . . . . ♏ i.
Mentholis . . . . . gr. i.
Olei gaultheriæ . . . . . ♏ i.
Liquid albolene . . . . . fl. ℥ i.
M.

106. ℞ Olei caryophyl. . . . . ♏ v.
Liquid albolene . . . . . fl. ℥ i.
M.

107. ℞ Olei caryophyl. . . . . ♏ viij.
Terebene . . . . . ♏ xx.
Liquid albolene . . . . . q.s. ad fl. ℥ i.
M.

108. ℞ Fl. ext. thuja occidentalis.

109. ℞ Aluminis pulv. . . . . gr. xxx.
Glycerini . . . . . ℥ iv.
Aquæ dest. . . . . q.s. ad fl. ℥ i.
M.

HÆMOSTATICS.

110. ℞ Ferri chloridi . . . . . gr. v. ad fl. ℥ i.

111. ℞ Acidi tannici . . . . . gr. x. ad fl. ℥ i.

112. ℞ Liquor. ferri chloridi . . . . . ℥ ij.
Aquæ dest. . . . . q.s. ad fl. ℥ i.
M.

ANTISEPTICS.

113. ℞ Sodii benzoatis . . . . . ℥ i. ad fl. ℥ i.

114. ℞ Aquæ calcis . . . . . fl. ℥ i.

115. ℞ Bromini . . . . . gr. ss. ad fl. ℥ i.

116. ℞ Acidi lactici . . . . . ♏. xx. ad fl. ℥ i.

117. ℞ Potassii permanganatis . . . . . gr. v. ad fl. ℥ i.

118. ℞ Potassii chloratis . . . . . gr. xx. ad fl. ℥ i.

119. ℞ Acidi borici . . . . . gr. x. ad fl. ℥ i.

120. ℞ Listerine . . . . . ℥ i.–ij. ad fl. ℥ i.

121. ℞ Hydrogen perioxidum.

This is used in full strength as purchased at the drug store, or diluted with one or two parts of water, according to the amount of smarting produced.

122. ℞ Acidi tartarici . . . . . gr. iss.
Hydrarg. chlorid. corrosiv. . . . . gr. ss.
Aquæ dest. . . . . fl. ℥ i.
M.

DRY INHALATIONS.

Dry inhalations are composed of substances which volatilize at ordinary temperatures, or simply by the heat of the hand. They may be used with any of the instruments which are ordinarily used for vapor inhalations, or they may be easily inhaled from a small wide-mouthed bottle in the bottom of which the medicine has been placed on a sponge.

One of the simplest and most efficacious inhalers for dry preparations consists of a glass tube about four or five inches in length, open at both ends, and holding a small sponge at its middle. The remedy is dropped on the sponge, and air is inspired through the tube.

When the substances are used with the small glass-tube inhaler, the amount given for each inhalation should be divided into three or four parts which are to be used successively.

If the effect is only needed in the throat and nose, the solution may be concentrated so that the same amount of medicine will be obtained without repeatedly charging the inhaler. In this case, the patient should not inspire deeply, and only two or three inhalations should be taken per minute. These inhalations may be sedative or stimulant.

SEDATIVES.

123. ℞ Acidi hydrocyanici diluti . . . . . fl. ʒ i. ad fl. ℥ i.
S. A teaspoonful at each inhalation.

124. ℞ Ætheris. S. A half-teaspoonful at each inhalation.

125. ℞ Amyl nitriti . . . . . ♏ i.
Alcoholis . . . . . ♏ xxx.
M. S. Use at each inhalation. *This is useful, especially in spasmodic affections.*

126. ℞ Olei santali albi . . . . . ♏ i.
Alcoholis . . . . . ♏ xxx.
M. S. To be used at each inhalation in divided doses.

127. ℞ Chloroformi . . . . . fl. ʒ ss.
S. To be used at each inhalation; to be breathed slowly.

STIMULANTS.

128. ℞ Tinct. iodi . . . . . ♏ x.–xxx.

In this same category may be included the carbonate of ammonium and camphor, used as smelling-salts; and nascent chloride of ammonium, used by any of the inhalers constructed especially for that purpose.

FUMING INHALATIONS.

Fuming inhalations are prepared by saturating bibulous paper with a solution of the remedy of a given strength, drying the paper, and then cutting it into twenty equal parts, each of which will contain one twentieth of the amount of medicine used. These strips may be rolled into cigarettes, or they may be burned under a funnel which will conduct the smoke to the mouth. They are employed in asthma and spasm of the larynx. The principal medicines employed in this manner are:

129. ℞ Potassii arseniatis . . . . . gr. xv.

130. ℞ Sodii arseniatis . . . . . gr. xx.–xl.

131. ℞ Potassii nitratis . . . . . gr. xxx.–lx.
Aquæ . . . . . ad fl. ℥ i.

The three latter may be modified, as recommended in the Throat Hospital Pharmacopœia, by the addition of various volatile principles. These volatile substances are added by moistening the nitre paper in a tincture, or, in the case of volatile oils, in a solution, of one part of the oil to nine parts of alcohol, and then exposing the paper to the air a few minutes to allow the alcohol to evaporate. The papers must be freshly prepared and kept in tinfoil. The following are the preparations most useful:

SEDATIVES.

132. Nitrated papers with tinct. benzoini comp.

133. Nitrated papers with tinct. hyoscyami vel stramonii.

134. Nitrated papers with oleum santali.

135. Nitrated papers with oleum sumbuli.

STIMULANTS.

136. Nitrated papers with spts. camphoræ.

137. Nitrated papers with oleum cinnamomi.

138. Nitrated papers with oleum cassiæ.

PIGMENTS.

The name pigments is given to the various mixtures which are designed for topical application by means of a brush, a probang wound with cotton, or by the compressed-air atomizer; the latter is now almost invariably employed in preference to the brush or probang. They may be prepared with water or with glycerin, but it should be remembered that the latter is irritating to some throats. The pigments may be anæsthetic, astringent, stimulant, or antiseptic in their effects.

LOCAL ANÆSTHETICS.

139. ℞ Morphinæ sulphatis . . . . . gr. iv.
Acidi carbolici . . . . . gr. xxx.
Glycerini . . . . . fl. ℥ i.
M.

Thirty grains of tannin may be added, when a slightly astringent effect is desired.

140. ℞ Atropinæ . . . . . gr. $\frac{1}{10}$
Strophanthin. . . . . gr. $\frac{1}{8}$
Olei caryophylli . . . . . ♏ iij.
Acidi carbolici . . . . . gr. x.
Cocainæ hydrochloratis . . . . . gr. xx.
Aquæ dest. . . . . fl. ℥ i.
M.

141. ℞ Chloral . . . . . ℥ i.
Aquæ . . . . . ad fl. ℥ i.
M.

142. ℞ Morphinæ sulphatis . . . . . gr. xx.
Chloroformi . . . . . ad fl. ℥ i.
M.

143. ℞ Sol. cocainæ . . . . . 10% to 25%

This solution is rarely used for any other purpose than that of producing anæsthesia of the faucial surfaces—where the throat is hyper-sensitive—to facilitate an examination of the pharyngo-larynx.

ASTRINGENTS.

144. ℞ Zinci chloridi . . . . . gr. x. ad fl. ℥ i.
145. ℞ Zinci sulphatis . . . . . gr. x.–xxx. ad fl. ℥ i.
146. ℞ Ferri et ammonii sulphatis . . . . gr. xxx. ad fl. ℥ i.
147. ℞ Liquor ferri chloridi . . . . . ♏ xl. ad fl. ℥ i.
148. ℞ Acidi tannici . . . . . ʒ ij.
Glycerini . . . . . ad fl. ℥ i.
M.

STIMULANTS AND CAUSTICS.

149. ℞ Zinci chloridi . . . . . gr. xxx. ad fl. ℥ i.
150. ℞ Cupri sulphatis . . . . . gr. xx. ad fl. ℥ i.
151. ℞ Liquor ferri chloridi . . . . . fl. ʒ ij. ad fl. ℥ i.
152. ℞ Argenti nitratis . . . . . ʒ ss. to ʒ i. ad fl. ℥ i.
153. ℞ Liquor hydrargyri nitratis . . . . ♏ xl. to ʒ ij. ad fl. ℥ i.
154. ℞ Tinct. iodi . . . . . ℥ i.
155. ℞ Iodi . . . . . gr. xxx.
Glycerini . . . . . ad fl. ℥ i.
M.
156. ℞ Argenti nitratis . . . . . gr. lx. ad fl. ℥ i.
157. ℞ Argenti nitratis . . . . . gr. xl. ad fl. ℥ i.
158. ℞ Argenti nitratis . . . . . gr. x. ad fl. ℥ i.
159. ℞ Tinct. iodi.
160. ℞ Liquor iodi comp.

ANTISEPTICS.

161. ℞ Acidi carbolici . . . . . gr. xxx. ad fl. ℥ i.

INSUFFLATIONS.

Powders have been extensively used in the treatment of nasal and laryngeal affections. I am accustomed to dilute most of the drugs which I employ in powdered form with from one to four parts of sugar of milk, acacia, or starch. Of the following powders, two or three grains are used at each insufflation.

SEDATIVES.

162. ℞ Bismuthi carbonatis.

163. ℞ Morphinæ sulphatis . . . . . gr. $\frac{1}{8}$–gr. $\frac{1}{4}$
Bismuthi carbonatis . . . . . gr. ij.
M.
Tannin or iodoform may be added.

164. ℞ Morph. sulph. . . . . gr. iv.
Bismuthi subnit. . . . . ʒ iv.
Amyli . . . . . ℥ i.
M.

165. ℞ Morphinæ . . . . . gr. v.
Iodol
Bismuthi subnit.
Sacch. lact. . . . . āā gr. xxx.
M.

166. ℞ Sodii bicarbonatis
Sodii boratis . . . . . āā gr. iss.
Amyli . . . . . gr. i
Cocainæ hydrochloratis . . . . . gr. iv.
Sacch. lact. . . . . q. s. ad gr. C.
M.

167. ℞ Cocainæ hydrochloratis . . . . . gr. x.
Atropinæ . . . . . gr. ½
Mag. carb. levis . . . . . gr. xv.
Sacch. lact. . . . . q. s. ad gr. D.
M.

168. ℞ Cocainæ hydrochloratis . . . . . gr. x.
Atropinæ . . . . . gr. ½
Morph. sulph.
Mag. carb. levis . . . . . āā gr. xv.
Sacch. lact. . . . . q. s. ad gr. D.
M.

ANTISEPTICS AND STIMULANTS.

169. ℞ Acidi borici

170. ℞ Iodol

171. ℞ Iodoformi
Acidi borici . . . . . āā gr. l.
M.

172. ℞ Iodoformi . . . . . gr. l.
Bismuthi subnit.
Benzoini res. . . . . āā gr. xxv.
M.

173. ℞ Iodoformi.

ASTRINGENTS AND STIMULANTS.

174. ℞ Hydrastum muriatis . . . . . gr. xxv.
Acaciæ . . . . . q. s. ad gr. C.

175. ℞ Pulv. res. myrrhæ.

176. ℞ Morph. sulph. . . . . gr. v.
Acidi tannici . . . . . gr. xxv.
Pulv. Andersonii . . . . . ℥ i.
M.

177. ℞ Benzoini res.
Bismuthi subnit. . . . . āā gr. l.
M.

178. ℞ Bismuthi subnit.

179. ℞ Hyd. chlor. mitis.

180. ℞ Aluminis
Sacch. albi . . . . . āā gr. l.
M.

181. ℞ Antipyrin.
Cocainæ hydrochloratis . . . . . āā gr. x.
Mag. carb. levis . . . . . gr. xv.
Sacch. lact. . . . . q. s. ad gr. D.
M.

NASAL DOUCHES.

The following preparations may be used as insufflations or by the anterior or posterior nasal douche or syringe, for detergent or antiseptic purposes. They should always be used warm, and may be followed by more potent remedies. The amount given below should be added to a pint of water at blood heat, and part or all of it used at each application.

182. ℞ Sodii chloridi . . . . . ʒ i.

183. ℞ Sodii bicarbonatis . . . . . ʒ i.

184. ℞ Potassii permanganatis . . . . . gr. iii.

185. ℞ Acidi carbolici . . . . . gr. xxv.

186. ℞ Zinci sulpho-carbolatis . . . . . gr. xxv.

187. *Salicylate Wash.*
℞ Sodii salicylatis
Sodii biboratis . . . . . āā ʒ vi.
Sodii bicarbonatis
Sodii chloridi . . . . . āā ʒ x.
M. S. ʒ i. ad aquæ tepidæ O i.

INDEX.

It has been deemed best to give a synopsis of the articles on each disease and its differentiations, using abbreviations that will need no explanation to the profession.

ABDOMINAL breathing, 11
Abscess, infraglottic, due to syphilis, 430
of the larynx, illus., 429, 430
symp., 429; diag., prog., treat., 430; diff. fr. croup; fr. retro-pharyngeal abscess, fr. acute catarrhal inflammation, fr. œdema, 430
of the lung, 129–131
symp., 129; diag., 130; prog., treat., 131; diff. fr. bronchitis, fr. pneumonia, fr. pleurisy, 130
of the nasal septum, 603
diff. fr. cancer, 573; fr. hæmatoma, 602
of the tonsils, syn. of phlegmonous tonsillitis, 368
retropharyngeal, 383–386
Abscission of the uvula, 359
Accentuation of the heart-sounds, 192
Acute and subacute bronchitis, 89, 90
anat., path., etiol., symp., 89
catarrhal laryngitis, syn. of acute laryngitis, 394
cold in the head, syn. of acute rhinitis, 522
coryza, syn. of simple acute rhinitis, 522
endocarditis, 219–222
syn., anat., path., 219; etiol., symp., diag., 220; prog., treat., 221
diff. fr. pericarditis, 220
follicular glossitis, symp., diag., prog. treat., 347
follicular pharyngitis, 339, 340
anat., path., etiol., symp., diag., 339; prog., treat., 340
diff. fr. simple acute sore throat, 339
follicular tonsillitis, diff. fr. mycosis, 376, 377
inflammation and œdema of the uvula, treat., 358
laryngitis, 394–397
syn., anat., path., etiol., symp., 394; diag., 395; prog., treat., 396
diff. fr. spasm of the glottis, fr. croup, fr. paralysis of the vocal cords, fr. foreign bodies, 395, 396; fr. croup, 414; fr. retropharyngeal abscess, 430
miliary tuberculosis, 165–167 [166
anat., path., etiol., 165; symp., diag., diff fr. other forms, 166, 167
myocarditis, 231
nasal catarrh, syn. of simple acute rhinitis, 522
Acute œsophagitis, 632, 633
etiol., symp., 632; diag., prog., treat., 633
pericarditis, 212
pleurisy, 61–72
etiol., symp., 62; diag., 68; prog., 71; treat., 72
diff. fr. pleurodynia, fr. pericarditis, fr. pneumonia, fr. phthisis, fr. collapse of the lung, fr. cancer, fr. hypertrophy of the liver and spleen, 68–71; fr. abscess of the lung, 130; fr. angina pectoris, 251
pneumonia, syn. of lobar pneumonia, 113
rheumatic sore throat, 316, 317
anat., path., etiol., symp., 316; diag., prog., treat., 317
diff. fr. acute sore throat, 312, 323
rhinorrhea, syn. of simple rhinitis, 522
sore throat, 311–314
syn., anat., path., etiol., symp., 311; diag., 312; prog., treat., 313
diff. fr. scarlatina, fr. acute tonsillitis, 312; fr. acute rheumatic sore throat, 312, 324; fr. sore throat of scarlet fever, 324; fr. acute follicular pharyngitis, 339
sthenic pneumonia, syn. of lobar pneumonia, 113
tonsillitis, 362–367
syn., 362; anat., path., etiol., symp., 363; diag., 364; prog., treat., 366
diff. fr. acute sore throat, 312; fr. scarlatina, fr. diphtheria, fr. suppurative tonsillitis, fr. syphilitic sore throat, 364–366
tubercular phthisis, diff. fr. lobular pneumonia, 127
tubercular sore throat, 350–353
anat., path., etiol., symp., 350; diag., 351; prog., treat., 352
diff. fr. rheumatic sore throat, 320; fr. chronic follicular pharyngitis, 344; fr. syphilitic sore throat, 351, 352, 355; fr. scrofulous sore throat, 350, 351, 352
tuberculosis diff. fr. emphysema, 111
Adams' clamp, 596
Adenoid growths in the vault of the pharynx, syn. of hypertrophy of the pharyngeal tonsil, 613
Adenomata, 467

Adhesion in syphilitic sore throat, 355
of the inner surfaces of the arytenoid cartilages, diff. fr. bilateral paralysis, 513
Adirondacks for phthisis, the, 175
Adventitious sounds, 48–54
Ægophony, 55, 57
Aerial goitre, syn. of tracheocele, 486
Age modifies percussion sounds, 27
Aitken, membranous croup, 411
Albolene in inhalations or sprays for throat and nose, 95, 441, 530, 535, 538, 557, 568, 587
Alcoholic stimulation in bronchitis, 98; in pulmonary phthisis, 171; in acute endocarditis, 221; in chronic endocarditis, 224; in angina pectoris, 252; in erysipelatous sore throat, 316; in diphtheria, 335; in syphilitic laryngitis, 448
Algiers for phthisis, 176
Allen, Harrison, inequality of the choanæ, 309; galvano-cautery, 544
Allingham, mouth gag, illus., 419, 617
Allison, Scott, stethogoniometer, illus., 18; differential stethoscope, illus., 37
Alps, goitre in the, 629
Ambidexterity in examination of the larynx, 285
American Journal of Medical Sciences, contagious pneumonia, Wagner, 116; diphtheria, Prudden, 329; congenital syphilis, John N. Mackenzie, 449; lupus, G. M. Lefferts, 451; laryngectomy, George B. Fowler, 483
Laryngological Association, Transactions, Registers of male and female voices, Thomas R. French, 298; choanæ unequal, Allen, 309; acute tubercular sore throat, Delavan, 352; leucoplakia buccalis, Ingals, 362; chromic acid in trachoma, Charles E. Sajous, 408; tubercular laryngitis, Jarvis, 441; feeding in laryngitis, Beverly Robinson, 443; snare forceps, Jarvis, 473; thyrotomy, Joseph Leidy, 475; laryngotomy, Cohen, 482; chorea laryngis, George M. Lefferts, E. Holden, 501; same, F. I. Knight, 501, 502; falsetto voice, J. C. Mulhall, 503; laryngeal vertigo, F. I. Knight, 504; relation of hay fever and conditions in the nasal passages, William H. Daly, 553; nasal cancerous tumors, R. P. Lincoln, 573; deflection of the nasal septum, D. Bryson Delavan, 594; same, D. N. Rankin, 605; rhinolaryngitis, Beverly Robinson, 609; extirpation of nasal tumors, Lincoln, 622
Amphoric cough, 59; resonance defined, 30; respiration, 41, 46, 47; sound, 41; voice, 55, 57; whisper, 58
Amygdalitis, syn. of acute tonsillitis, 362
Amyl nitrite in chronic endocarditis, 229
Anæmia, diff. fr. endocarditis, 226; fr. tubercular laryngitis, 437
Anæmic, hæmic or organic murmurs, 196, 204; diff. fr. atheroma, 256
Anæsthesia of the larynx, 499, 500; etiol., symp., diag., prog., treat., 499; of the pharynx, etiol., prog., treat., 388
produced generally, 495, 582, 606
produced in tubercular laryngitis, 442
produced locally, 74, 80, 266, 407, 409, 422, 457, 484, 495, 544, 568, 597, 598, 425, 442
Anæsthetics, pigment, 655
Anatomy and physiology of the heart, 177–180
Anchylosis of the arytenoid cartilages, 514, 515; diag., treat., 515
Anemone pratensis, unsatisfactory in pertussis, 155
Aneurism, aortic or thoracic, 16, 256–266
of the aorta, diff. fr. acute pleurisy, 70; fr. solid tumors, 262; fr. aortic pulsation, fr. pulsating empyema, 263; fr. dilated auricle, fr. consolidation of the lung, 264; fr. aneurism of the pulmonary artery, 265
of the arch of the aorta, 257
of the arteria innominata, 265, 266
of the ascending aorta, illus., 209
of the descending aorta, 257
of the heart, etiol., diag., prog., treat., 245
of the pulmonary artery, 264, 265
diff. fr. aneurism of the aorta, 265
of the sinuses of Valsalva, 257
Aneurismal murmur, diff. fr. mitral, 198
Aneurismatiscope, the, 261
Angina diphtheritica, syn. of diphtheria, 328
epiglottidea, syn. of acute laryngitis, 394
laryngea, syn. of acute laryngitis, 394
membranosa, syn. of diphtheria, 328
pectoris, 250–253
etiol., 250; symp., diag., prog., 251; treat., 252
diff. fr. pseudo-angina, fr. intercostal neuralgia, fr. acute pleurisy, fr. myalgia, 251, 252
Angiomata, or vascular tumors, illus., 467, 468
of the nose, syn. of vascular nasal tumors, 570
Annales de Gynécologie et d'Obstétrique, diphtheria, Roux and Yersin, 336
des Maladies de l'Oreille, fractures of the larynx, Panas, 489
Annual of the Universal Medical Sciences, distoma pulmonale, 151; pseudo-diphtheria, Smith and Warner, 329; diphtheria infectious through clothing or furniture, Grancher, 334; spasm of the glottis, Lubet-Barbon, 496; rhinitis, Raulin, 532; nasal osseous cysts, Macdonald quoted by Charles E. Sajous, 570; adenoid growths in deaf-mutes, Wróblewski, 614
Anomalous heart sounds, 205
Anorexia in tubercular laryngitis, 437
Anosmia, 591, 592
etiol., 591; symp., diag., prog., treat., 592
Anstie, F. E., value of sphygmograph, 211

Antipneumotoxin in pneumonia, blood serum or, 123
Antipyrine in whooping cough, 155; in rheumatic sore throat, 317
Antiseptic gargles, formulæ for, 647
lozenges, formulæ for, 648
vapor sprays, formulæ for, 653
and stimulant insufflations, formulæ for, 657
pigments, formulæ for, 656
Antispasmodic vapor inhalations, formulæ for, 650
Antrum, empyema of the, 579–583
of Highmore, illus., 302, 579
Aorta, the, 179
aneurism of the (see aortic or thoracic aneurism)
aneurism of the ascending, 209
aneurism of the descending, 257
atheroma of the, 254–256
coarctation of the, 266, 267
rupture of the, 255
Aortic aneurism, diff. fr. pulmonary cancer, 148
area, illus., 198, 199
endarteritis, syn. of atheroma of the aorta, 254
murmurs, 198–200
obstruction, illus., 209, 225, 230
or thoracic aneurism, 14, 256–266
anat., path., etiol., 256; symp., 258; diag., 262; prog., treat., 265
diff. fr. chronic endocarditis, 226, 227; fr. eccentric cardiac hypertrophy, 238; fr. solid tumors, fr. aortic pulsation, fr. pulsating empyema, fr. dilatation of the auricle, fr. aneurism of the pulmonary artery, fr. consolidation of the lung, 262–265
pulsation, diff. fr. aneurism, 263
regurgitant murmurs, 200
regurgitation, illus., 209, 225, 228, 230
semilunar valves, 178
valves, 7; disease of, 256
Aortitis, 254
Apex-beat of the heart, 10, 182, 184–185
Apex, pleurisy of the, 82
Aphonia, functional, hysterical, or nervous, syn. of bilateral paralysis of the lateral crico-arytenoid muscles, 508
Aphonic pectoriloquy, 58
Aphthous sore throat, syn. of simple membranous sore throat, 324
Apneumatosis, syn. of pulmonary collapse, 139
Apoplexy, pulmonary, 15, 137, 138
Applicator, chromic acid, 409; for intubation tubes, illus., 420; cotton, 568; post-nasal snare, 623
Arch of the aorta, aneurism of the, 257
Arching of the tongue an obstacle to laryngoscopy, 290
Archives Générale de Médecine, erysipelatous sore throat, Cornil, 314; erysipelatous laryngitis, Cornil, 428
Archives of Laryngology, trachoma of the vocal cords, Carlo Labus, 408; lupus of the larynx, F. I. Knight, 451
Archives of Pediatrics, influenza, Charles W. Earle, 520
Area of cardiac impulse, 185; of cardiac dulness, flatness, 189; of cardiac sounds, 191; valvular, 197; of endocardial murmurs, illus., 198
Argand lamp for laryngoscopy, 279, 281
Arizona for phthisis, 175
Arteria innominata, aneurism of the, 265
Arterial diseases, cardiac and, 212–268
Artificial light to illuminate the larynx, direct, indirect, 275
Ary-epiglottic folds, 296
muscles, paralysis of the thyro-epiglottic and, 505
Aryteno-epiglottidean folds, 296
Arytenoid cartilages, illus., 296
cartilages, anchylosis of the, 514, 515
muscle, paralysis of the, 511
Asch, Morris, lupus of the larynx, 451
Aspiration in acute pleurisy, 72; in subacute pleurisy, 73–75; in chronic pleurisy, 78; in abscess of the lung, 131; in pericarditis, 217
Aspirator, mode of using the, 73–75
Asthenia in diphtheria, 333; in acute tubercular sore throat, 352
Asthma, 102–105
anat., path., 102; etiol., 103; symp., 104; diag., prog., 105; treat., 106
diff. fr. bronchitis, 92; fr. capillary bronchitis, fr. spasmodic laryngeal affections, fr. emphysema, fr. cardiac dyspnœa, 105; fr. stenosis of the larynx, 457; fr. hay fever, 554
Asthmatic hay fever, 554
Astringent and stimulant insufflations, formulæ for, 657
and stimulant spray inhalations, formulæ for, 652
gargles, formulæ for, 649
lozenges, formulæ for, 643
pigments, formulæ for, 656
Asystolism, 241
Atelectasis, syn. of pulmonary collapse, 139
Atheroma of the aorta, 254–256
syn., anat., path., etiol., 254; symp., diag., 255; treat., 256
diff. fr. disease of the valves, fr. anæmic murmurs, 256
Atheromatous degeneration of the aorta, syn. of atheroma of the aorta, 254
Atomizer, 401, 402; for oil, 536
Atrophic follicular pharyngitis, 343
rhinitis, 528, 547–552
anat., path., 547; etiol., symp., 548; diag., prog., 549; treat., 550
diff. fr. lupus, fr. syphilis, fr. suppuration, fr. rhinoliths, fr. foreign bodies, 549; fr. chronic suppurative ethmoiditis, 586
Atrophy of the heart, syn. diag., 242
of the vocal cords, 515
Auricles of the heart, 178

Auricular systole, 182; illus., 201
Auscultation, 9, 34-47; mediate, immediate, 34; rules for, 38; in health, 39-41; in disease, 41-47; over the heart, 189; in aneurism of the aorta, 261
Auscultatory percussion, 32-33
Austria, rhinosclerma in, 588
Austrian mountains for phthisis, 175
Autumnal catarrh, syn. of hay fever, 553
Avenbrugger, percussion, 21
Avery, laryngoscopy, 272
Axillary region, 4, 8

Babbington, laryngoscopy, 272
Bacilli, tubercle, 157; transmitted to fœtus, 158; staining, 164, 165; in endocarditis, 222; in lupus of the larynx, 451
Bacillus, Klebs-Löffler, diph., 329
mallei, glanders, 589
tuberculosis, 578
Bacteria in pericarditis purosa, 212; in ulcerative pericarditis, 222; in hypertrophy of the tonsils, 370
Balfour, G. W., quality of murmurs of the heart, 200; heart disease, 247; bradycardia, 250; mode of administering chloroform in angina pectoris, 252
Barker, Fordyce, turpeth mineral in croup, 417
Barrel-shaped chest, 12
Base of heart, to find, 188
Basedow's disease, syn. of exophthalmic goitre, 632
Battery, galvano-cautery, 345
Baumes, laryngoscopy, 272
Bazin, leucoplakia buccalis, 360
Belfield, W. T., guaiacol in phthisis, 173; iodine trichloride in surgery, 441
Bell sound in percussion, 31
Bellocq, laryngoscopy, 272; canula, 306
Benign growths in the larynx, illus., 466-476
symp., 466; diag., 467; prog., treat., 469
diff. fr. syphilis, fr. tubercular laryngitis, fr. lepra, lupus, outgrowths, fr. eversion of the ventricles, fr. malignant tumors, 467-469; fr. malignant tumors, 479, 573
Bennatti, laryngoscopy, 272
Berberine, identical with hydrastine, 95
muriate in chronic laryngitis, 407; in rhino-pharyngitis, 610
Berliner klinische Wochenschrift, tubercles in lung, Virchow, 107; sound in emphysema, Gerhardt, 109; pneumonia contagious, Kuhn, 116; blood serum or antipneumotoxin in pneumonia, Klemperer, 123; dislocation of the larynx, H. Braun, 490; operations on the antrum, Krause, 582
Best, J. E., furunculosis of the nose, 559
Biegauski, pleurisy, 66
Bilateral paralysis of the lateral crico-arytenoid muscles, illus., 508-510
syn., etiol., symp., 508; diag., treat., 510
paralysis of the posterior crico-arytenoid muscle, illus., 511-513
Bilateral paralysis of the posterior crico-arytenoid muscle, anat., path., etiol., symp., 512; diag., prog., treat., 513
diff. fr. adhesion of the inner surfaces of the arytenoid cartilages, fr. spasm, 513
Bilious pneumonia, 128, 129
Bilocular pleurisy, diff. fr. other forms, 83
Birch-Hirschfeld, F. V., bacilli transmitted to fœtus, 158
Bird, hydatid cysts of the lungs, 149
Bizot, aortitis, 254
Black, G. V., cinnamon water antiseptic, 336
Blake, Clarence, snare for polypi, 567
Blanden, deflection of the nasal septum, 595
Blood serum or antipneumotoxin in pneumonia, 123
Blue disease, the, syn. of morbus cæruleus, 246
Bocelli, Guido, distinction between serum and pus, 77
Boileau, aortic regurgitation, illus., 209
Bokai, retropharyngeal abscess, 384
Bollinger, case of glanders eleven years, 590
Bone drill, 582
Bony tumors, nasal, 571, 572
Borgiotti, case of œsophageal spasm five hundred and thirty-one days, 638
Boric acid in cinnamon water highly effective in diphtheria, 336
Bosworth, tongue-depressor, illus., 271; tubercular laryngitis, 436; cancer in the larynx, 476; chronic rhinitis, 537, 545; mucous polypi in asthma, 565; saws, 601
Bougie, œsophageal, 390; olivary, 635
Boundaries of the heart, 188
Bouveret, L., pleurisy, 76; tachycardia, 249
Bowditch, danger in washing pleural cavity, 78
Boyle, immediate auscultation, 34
Bozzini, laryngoscopy, 272
Bradycardia, treat., 250
Brainard, bone drill, illus., 582
Braun, H., dislocation of the larynx, 490
Bristle extractor, illus., 642
British Medical Journal, cause of angina pectoris, Douglas Powell, 250; diphtheritic bacilli, Armand Ruffer, 329
Broad condylomata, 353
Brodie, mode of applying mercury to infants, 577
Bronchial cough, 59
fremitus, 16
glands enlarged, 152, 153
respiration, 41, 45
tubes, fremitus in dilatation of the, 15
whisper, normal, exaggerated, cavernous, 58
Bronchiectasis or bronchicatasis, syn. of dilatation of the bronchial tubes, 100; syn. of fibroid phthisis, 156
Bronchitis, 89-100; acute and subacute, 89, 90; chronic, 89, 90-95; capillary, 95-98; plastic, 99, 100
diff. fr. abscess of the lung, 130; fr. pulmonary gangrene, 145; fr. tracheitis, 461

Broncho-cavernous respiration, 46
Bronchocele, syn. of goitre, 629
Bronchophony, 56; normal, 55; whispering, 58
Broncho-pneumonia, syn. of lobular pneumonia, 123
Bronchorrhagia, 134
Bronchorrhœa, 92
Bronchotomy, 495
Broncho-vesicular or harsh respiration, 41, 44
Brooklyn Medical Journal, pneumonia contagious, Matheson, 116
Brower, Daniel R., mode of ventilation in diphtheria, 334; exophthalmic goitre, 632
Brown induration, symp., diag., treat., 134
Browne, Lennox, diphtheria, 328, 334, 336; acute tubercular sore throat, 350, 351; hypertrophy of the tonsils, 373; spasm of the pharynx, 390; definition of croup, 411; syphilitic laryngitis, 443, 448, 449; lupus of the larynx, 453; lepra of the larynx, 454; endo-laryngeal cauterization in cancer, operation of resection of the larynx, 481
Walton, epistaxis, 562
W. N., large rhinolith, 604
Bruit de diable, syn. of venous murmur, 207
de pot fêlé, syn. of cracked-pot resonance, 31
Bruns, Paul, pincette, illus., 291; infra-thyroid laryngotomy, 476
Bulbar paralysis, progressive, 391
Bulletin de la Société de Chirurgie, deflection of the nasal septum, Chassaignac, 595
médicale des Vosges, cause of angina pectoris, Liégeois, 250
Burns of the pharynx, scalds and, 392
Burrs, nasal, 546, 598
Bursa pharyngea, illus., 309

Cabot, A. T., pleurotomy, 76; drainage tubes, illus., 79
Calculus of the tonsil, syn. of concretions of the tonsils, 375
California for bronchitis, 95; for phthisis, 175
Calomel in lobular pneumonia, 122; in acute sore throat, 313; in diphtheria, 338
Camman, stethoscope, illus., 32, 36
and Clark instituted auscultatory percussion, 32
Campbell, see Harries and Campbell
Canadian Practitioner, siphon drainage in pleurisy, Powell, 79
Cancer (see also malignant)
Cancer, diff. fr. leukoplakia buccalis, 362
of the larynx, diff. fr. chronic laryngitis, 403, 404; fr. syphilitic laryngitis, 447; fr. lupus, 453
of the pharynx, anat., path., symp., 386; diag., treat., 387; diff. fr. chronic rheumatic sore throat, 320; fr. syphilis, fr. fibrous tumors, 387
of the tonsil, 380, 381
diag., 380; prog., treat., 81
diff. fr. tubercular ulceration of the tonsils, 378; fr. hypertrophy, fr. syphilitic ulceration, 380, 381; fr. rhinoliths, 605
Cancer, pulmonary, 70, 146, 148
Cancerous growths, diff. fr. nasal mucous polypi, 566; fr. nasal bony tumors, 572
Capillary bronchitis, 95-98
anat., path., 95; etiol., symp., diag., 96; prog., treat., 98
diff. fr. phthisis, 98; fr. asthma, 97, 105; fr. lobar pneumonia, fr. lobular pneumonia, fr. pulmonary œdema, 97, 98
Carbon dioxide in asthma, 106
Cardiac and arterial diseases, 11, 183, 212-268
aneurism, 245
dilatation syn. of dilatation of the heart, 239
displacement, diff. fr. hypertrophy and dilatation of the heart, 238
dulness, 188-190
hypertrophy, 14
hypertrophy, eccentric, 236
hypertrophy, simple, 234-236
impulse, 185
murmurs, 195-211
origin of dropsy, indicated, 11
pulsation, 185, 187
region, form of the, 184
resonance, 25
sound, modified by disease, 185
Cardialgia, 247
Cardiectasis, syn. of dilatation of the heart, 239
Cardio-pleuritic friction murmurs, 196
Carious teeth, a soil for leptothrix buccalis, 376
Carroll, stethometer, illus., 17
Cartilages, arytenoid, 296, 514; of Santorini, of the larynx, of Wrisberg, 296; cricoid, tracheal, 299
Cartilaginous tumors, illus., 467
diff. fr. nasal mucous polypi, 566; fr. hæmatoma of the nasal septum, 602
Cary, Frank, mode of feeding after intubation, 421
Caseous pneumonia, 156
Casselberry, Wm. E., mode of feeding after intubation, 421
Catarrh, epidemic, 519; acute nasal, 522; chronic, 527; autumnal, 553
Catarrhal diathesis, 607
fever, epidemic, 519
hay fever, 554
laryngitis, illus., 399
diff. fr. diphtheria, 331; fr. croup, 413
pneumonia, syn. of lobular pneumonia, 123
sore throat, syn. of acute sore throat, 311
stage of croup, 412
Catarrhus æstivus, syn. of hay fever, 553
Caustics—pigments; stimulants and, 656
Cautery electrodes, 346
in diseases of the throat, *passim*, 240-485; in diseases of the nose, *passim*, 530-637
Cavernous sound, 41; respiration, 46; whisper, 58; cough, 59

Centralblatt für klinische Medicin, spasm of the œsophagus, Borgiotti, 638
Cerebral croup, syn. of spasm of the glottis, 496
Chancre in the throat, 353
Chassaignac, relation of generative organs and tonsils, 375; deflection of the nasal septum, 595; retro-nasal fibrous tumors, 621
Cheesy infiltration of the lung, 156
Chest, dimensions of the, 3–8; form of healthy, 9–12; pigeon breast, 10; barrel-shaped, 12; size of the, 17
Cheyne-Stokes respiration, 243
Chiari and Riehl, lupus of the larynx, 451
Chicago Medical Journal and Examiner, tympanitic resonance in pleurisy, Ingals, 66
Medical Record, resection of the ribs in pleurisy, A. B. Strong, 78
China, distoma pulmonale in, 150
Chloride of iron in erysipelatous sore throat, 316
Chlorine inhalation in phthisis, 172
Chloroform for angina pectoris, mode of administering, 252; a preferred anæsthetic for children, 373, 495, 618; for chronic laryngitis, 407; for general anæsthesia, 422, 582; preferred to ether in tracheotomy, 425; for cough, 501; for myasis narium, 606
Choanæ, the, illus., 309
Chondritis and perichondritis of the laryngeal cartilages, 433, 434
etiol., symp., 433; diag., prog., treat., 434
Chorditis tuberosa, syn. of trachoma of the vocal cords, 408
Chorea laryngis, 501, 502
anat., path., etiol., symp., 501; diag., prog., treat., 502
diff. fr. hysteria, 502
Chromic acid applicator, 409
acid in trachoma of the vocal cords, 409; effect in rhinitis compared with that of galvano-cautery, 537, 541; in hypertrophy of the pharyngeal tonsil, 616
Chronic abscess of the nasal septum, diff. fr. mucous polypi, 565
bronchitis, 14, 89, 90–95
anat., path., 90; etiol., symp., 91; diag., 92; prog., 93; treat., 94
catarrh, syn. of chronic rhinitis, 527; syn. of intumescent rhinitis, 531
catarrh of the larynx, syn. of chronic laryngitis, 398
coryza, syn. of chronic rhinitis, 527
endocarditis, 223–230
etiol., symp., 224; diag., 226; prog., 228; treat., 229
diff. fr. functional diseases of the heart, fr. pericarditis, fr. anæmia, fr. thoracic aneurism, fr. fatty heart, fr. congenital deformity, 226, 227
follicular glossitis, 347, 348
Chronic follicular glossitis, symp., diag., prog., treat., 348
diff. fr. rheumatic sore throat, 319
follicular pharyngitis, illus., 340–346
syn., 340; anat., path., etiol., 341; symp., 342; diag., prog., treat., 344
diff. fr. chronic rheumatic sore throat, 319; fr. syphilis, fr. tubercular sore throat, 344
follicular tonsillitis, syn. of hypertrophy of the tonsils, 370
inflammation and elongation of the uvula, 358–369
diag., treat., 359
laryngitis, illus., 398–408
syn., anat., path., 398; etiol., symp., 399; diag., 402; prog., treat., 404
diff. fr. paralysis of the vocal cords, fr. œdema of the larynx, fr. tubercular or syphilitic laryngitis, fr. cancer, 402–404
myocarditis, 231
œsophagitis, 633, 634
etiol., symp., diag., prog., treat., 633
pericarditis, 213
pharyngitis, syn. of chronic follicular pharyngitis, 340
pleurisy, 12, 76–82, 130
anat., path., etiol., symp., 76; diag., prog., 77; treat., 78
diff. fr. pneumothorax, fr. hydro-pneumothorax, 88; fr. pulmonary cancer, 147
pneumonia, syn. of lobular pneumonia, 123, 128; syn. of fibroid phthisis, 167
rheumatic laryngitis, syn. of chronic rheumatic sore throat, 318
rheumatic sore throat, 318–321
syn., anat., path., etiol., symp., 318; diag., 319; prog., 320; treat., 321
diff. fr. chronic follicular tonsillitis, glossitis or pharyngitis, fr. tuberculosis, fr. cancer, fr. neuralgia, fr. tobacco sore throat, 319, 320
rhinitis, 527–552
syn., 527
stenosis of the larynx, illus., 456–459
anat., path., etiol., symp., diag., 456; prog., treat., 457
diff. fr. asthma, fr. foreign bodies, fr. compression, fr. tumors, fr. paralysis of the abductors, 457
suppurative ethmoiditis, 585–587
etiol., symp., diag., 585; prog., treat., 586
diff. fr. mucous polypi, fr. atrophic rhinitis with œdema, fr. suppuration of the antrum, fr. emphysema of the sphenoidal and frontal sinuses, 585
tonsillitis, syn. of hypertrophy of the tonsils, 370
tuberculosis, 156
diff. fr. other forms of phthisis, 166
Ciniselli, galvanic puncture in thoracic aneurism, 265
Circumscribed pleurisy, 82

Circumscribed pleurisy, diff. fr. hydatid cysts of the lungs, 150
Cirrhosis or scirrhus of the lungs, syn. of dilatation of the bronchial tubes, 100; syn. of fibroid phthisis, 156, 167
Clark, see Camman and Clark
J. E., immunity to tubercular virus secured, 172; solution of iodine for goitre, 631
Clavicular region, 4
Clergyman's sore throat, syn. of chronic follicular pharyngitis, 340
Climatic treatment, subacute pleurisy, 75; bronchitis, 95, 100; asthma, 106; emphysema, 112; lobula rpneumonia, 128; pulmonary phthisis, 174–178; influenza, 522; hay fever, 555, 558
Clinical Diagnosis, Jasch, bacilli in phthisis, 164
Closure of the post-palatine space obstructing rhinoscopy remedied, 305
Cloves in laryngitis, solution of, 442
Coarctation of the aorta, 266, 267
syn., 266; diag., treat., 267
Cocaine as an anæsthetic, 74, 80, 266, 290, 370, 374, 377, 407, 409, 422, 425, 457, 484, 491, 495, 537, 544, 568, 597, 598, 603, 616, 617, 655
as a sedative, 389, 501, 525, 527, 530, 538, 551, 556, 584, 587, 651, 657
caution in the use of, 352, 530, 556, 568
not to be used as a sedative in acute sore throat, 314
Cog-wheel respiration, 41, 43
Cohen, J. Solis, laryngeal illumination, 282; laryngeal examination, illus., 286; larynx of woman, illus., 295; simple membranous sore throat, 327; chronic follicular pharyngitis, illus., 343; scrofulous sore throat, 348; scalds and burns of the pharynx, 392; hypertrophy of the larynx, 455; benign laryngeal tumors, 463; malignant tumors on the larynx, 476; laryngectomy, 482; nervous cough, 499; laryngeal paralysis, 509; spasm of the œsophagus, 637
Cohnheim, pulmonary thrombosis, 138
Coil of tubing to apply cold water in pneumonia, diphtheria, croup, 122, 335, 369, 416 (see Leiter coil)
Cold applications in pneumonia, 122; in certain diseases of the throat, 307, 329, 335, 361, 363, 369, 379, 386, 392, 408, 410, 416, 633; in nose bleeding, 552, 553 (see also Ice)
Collapse of the jugular veins, 207
pulmonary, 139–142
Colorado for asthma, 106; for phthisis, 175; rhinitis in, 527
Compendium de Chirurgie Pratique, deflection of the nasal sectum, Blanden, 596
Complete extirpation of the larynx described, 482
Compression of the œsophagus, 637
Concretions in the tonsil, syn., etiol., symp., prog., treat., 375
Condylomata, syphilitic, 153, 468, 575
Congenital deformities of the heart diff. fr. chronic endocarditis, 226, 227
deformity of the nose, treat., 593
murmurs, 204, 246
syphilis of the nose, etiol., symp., diag., prog., treat., 577
Consolidation of the lung, diff. fr. hypertrophy and dilatation of the heart, 237; fr. aortic aneurism, 264
Convulsive disorders diff. fr. retropharyngeal abscess, 384, 385
Corea, distoma pulmonale in, 150
Corniculum laryngis, syn. of cartilage of Santorini, 296
Cornil, erysipelatous sore throat, 314; erysipelatous laryngitis, 428
Corvisart, syphilitic disease of the heart, 245
Coryza, acute, 522, 591; chronic, 527; syphilitic, 567; in measles, 591
Cotton applicator, illus., 568
Cough, amphoric, bronchial, cavernous, 59; laryngeal, 59, 400; in hypertrophy of the tonsils, 371; irritative, nervous, 498
Cracked-pot resonance, 28, 31
Cramp of the œsophagus, syn. of spasm of the œsophagus, 637
Creaking or crumpling sounds, 53
Creasote for pulmonary phthisis, 173
Crepitant râles, 48, 51
râle redux, 118
Crequy, removal of foreign bodies in the œsophagus, 642
Crico-arytenoid muscles, paralysis of the, 508–514
Cricoid cartilage, illus., 299
Crico-thyroid muscles, paralysis of the, 506
Croup, membranous, 14, 411–426
tent, 416
Croupous bronchitis, syn. of plastic bronchitis, 99
pneumonia, syn. of lobar pneumonia, 113
Crumpling sounds, creaking or, 53
Crushing tumors with forceps, 474, 572
Csokor, transmission of bacilli to fœtus, 158
Cuneiform cartilages, syn. of cartilages of Wrisberg, 296
Curable mitral regurgitant murmurs, 202
Curschmann, cause of asthma, 103
Curtis, H. Holbrook, chronic rhinitis, 537; wash-bottle, illus., 586; nasal trephining, 601; vaporizer, illus., 612
Curved line of flatness in pleurisy, illus., 64, 65
Cutting forceps, right angle, 597
operations on laryngeal tumors, 474
Cyanosis, syn. of morbus cæruleus, 246
Cyclopedia of the Diseases of Children, pleurotomy, A. T. Cabot, 78; asthma among Hebrews, Saltmann, 103; double pneumonia, 115
Cyclopedia of Practical Medicine, rhinitis, C. J. D. Williams, 525
Cynanche laryngea, syn. of acute laryngitis, 394
pharyngea, syn. of acute sore throat, 311
tonsillaris, syn. of acute tonsillitis, 362

Cyrtometers, 17, 18
Cystic growths, illus., 466; retro-nasal, 526
Cysts of the lungs, hydatid, 148–150
Czermak, laryngoscopy, 272

Da Costa, J. M., divisions of the chest, 3; tympanitic resonance, 29, 30, 66; pneumopericardium, 218; irritable heart of soldiers, 249
Dakota for phthisis, 175
Daly, William H., hay fever related to conditions in nasal passages, 553
Damoiseau, pleuritic symptoms, 64
Danforth, J. N., mixed sarcoma, 478
Davidson, atomizer, illus., 405, 406; oil atomizer, illus., 536
Deafness, throat, 610–613
De Cérenville, epilepsy following irritation of pleural surfaces, 78
Deferred expiration, 43
Deflection of the nasal septum, 594–597
anat., path., etiol., 594; symp., diag., prog., treat., 595
Delafield, pneumonia infective, 115
Delavan, D. Bryson, acute tubercular sore throat, 352; hemorrhage after tonsillotomy, 375; leptothrix buccalis, 376; electricity in rhinitis, 552; empyema of the antrum, 579; deflection of the nasal septum, 594, 595
Demulcents, trochisci or lozenges, formulæ, 648
Dennison, Charles, binaural stethoscope, 37
Dental Review, cinnamon-water antiseptic, G. V. Black, 336
Derbyshire neck, syn. of goitre, 629
Descending aorta, aneurism of the, 257, 258
Des Maladies du Sinus Maxillaire, multiple secretion of pus in the antrum, Giraldes, 579
Deutsche Chirurgie, tracheotomy, Max Schüller, 486
Klinik, benign growths in the larynx, Lewin, 465
medicinische Zeitung, heredity in asthma, Lazarus, 100
medicinische Wochenschrift, pneumonia contagious, Mosler, 116; transmission of bacilli to fœtus, F. v. Birch-Hirschfeld, 158; nasal tuberculosis, F. Hahn, 578; differentiation of nasal affections, Max Schaeffer, 586
Medizinal-Zeitung, transmission of bacilli to fœtus, Csokor, 158
Deutsches Archiv für klinische Medicin, danger from heart in pleurisy, Leichtenstern, 71
Deviation of the septum, diff. fr. polypi, 565
Diagnosis, physical, 3–59
Diaphragmatic hernia, diff. fr. pneumothorax, 88
pleurisy, 71, 82
Diastole of the heart, 180
Diastolic murmurs, 203
Dicrotism, 210
Dictionnaire Encyclopédie des Sciences médicales, inflammation in removal of nasal tumors, Ollier, 622
Diffuse abscess of the larynx, syn. of phlegmonous laryngitis, 427
aneurism, 256
pulmonary hemorrhage, syn. of pulmonary apoplexy, 137
Dilatation in laryngeal diseases, 449, 457, 459, 472, 488, 515; in stricture of the œsophagus, 635, 636
of the aorta, diff. fr. aortic aneurism, 264
of the bronchial tubes, 15, 100–102
syn., anat., path., etiol., 100; symp., diag., 101; prog., treat., 102
diff. fr. phthisis, 101; fr. gangrene, 145
of the heart, 238–242
syn., anat., path., etiol., 239; symp., 240; diag., prog., 241; treat., 242
diff. fr. pericarditis, 241; fr. myocarditis, 232; fr. eccentric cardiac hypertrophy, 237
hypertrophy and, 236–239
of the larynx, 457, 458
Dilated auricle, diff. fr. aneurism of the aorta, 264
Dilator, cutting, laryngeal, 458; for stricture of the œsophagus, 636
Diminished resonance, 55
Diphtheria, 328–338
syn., anat., path., 328; etiol., 329; symp., 330; diag., 331; prog., 332; treat., 333
diff. fr. sore throat of scarlet fever, 323; fr. simple catarrhal or rheumatic pharyngitis, fr. tonsillitis, fr. erysipelas, fr. scarlatina, fr. simple membranous sore throat, fr. phlegmonous or erysipelatous sore throat, fr. phlegmonous or erysipelatous sore throat, 331, 332; fr. hypertrophy of the tonsils, 332; fr. acute tonsillitis, 365; fr. croup, 415; fr. phlegmonous laryngitis, 427
Diphtheritic laryngitis, 455
diff. fr. phlegmonous laryngitis, 427
Diphtheritis, syn. of diphtheria, 328
Diplococcus pneumoniæ of Fraenkel, 115
Disease of the aortic valves, diff. fr. atheroma, 256
Disinfection in diphtheria, extreme, 334
Dislocation of the larynx, 490
of the nasal bones, treat., 594
Dissecting aneurism, 256
Disseminated pneumonia, syn. of lobular pneumonia, 123
Distoma pulmonale, 150, 151
symp., diag., treat., 151
Divisions of the chest, illus., 3–8
supra-clavicular, 4; clavicular, 4; infra-clavicular, 4, 5; mammary, 4, 5; infra-mammary, 4, 6; supra-sternal, 4, 6; sternal, 4, 6; superior sternal, 4, 16; inferior sternal, 4, 7; supra-scapular, scapular, inter-scapular, 7; infra-scapular, 8; axillary, 4, 8; infra-axillary, 4, 8

Donaldson, F., treatment of nasal polypi, 566
Douches, nasal, instruments, 551
nasal, formulæ, 658
Dover's powder in acute laryngitis, 396
Drainage tubes for chronic pleurisy, 79–81; in abscess of the lung, 131; for empyema of the antrum, 583
Drill, bone, 582; for cutting cartilage, 598
Dropsy, diseases indicated by, 11
Dry inhalations, formulæ, 654
pleurisy, 61
râles, 48
Drzewiecki, J., pleurisy, 72
Dulness, 25, 26, 28, 29; triangle of, 64; cardiac, 188–190
Dupuytren, retro-nasal fibrous tumors, 621
Duration of sound, 23, 39

Earle, Charles Warrington, influenza, 520
Eccentric cardiac hypertrophy, syn. of hypertrophy and dilatation of the heart, 236
Ecchondroma and exostosis of the nasal septum, illus., 597–601
diag., prog., treat., 598
Ecchondroses, diff. fr. nasal cartilaginous tumors, 571
Eclectic inhaler, 649
Écrasement in hypertrophy of the tonsils, mode of, 373, 374
Écraseur, galvano-cautery, 567, 569, 571, 573, 622; guarded wheel, 474
Edinburgh Medical Journal, bradycardia, Balfour, 250; anæsthesia of the larynx, McBride, 499; empyema of the antrum, McBride, 580; large rhinolith, W. N. Browne, 604
Egypt for phthisis, 176; nasal syphilis in, 574
Electric lamp for transillumination, 581
light for laryngeal illumination, 281
Electricity in rhinitis, 552
Electrodes, cautery, 346; laryngeal, 509, 511
Electrolysis, 372, 601; method of, in retronasal tumors and goitre, 622, 631; for stricture of the œsophagus, 637
Ellis, curved line of flatness in pleurisy, illus., 64, 65
Elongation of the uvula, chronic inflammation and, 358
Elongated uvula, an obstruction to laryngoscopy, 289; remedied, 305
Emballometer, 33
Embolism, pulmonary thrombosis and, 138, 139
Emphysema, subcutaneous, 11; pulmonary, 107–112; atrophous, 109
Empyema, chronic pleurisy or, 61, 76–82
of the antrum, illus., 579–584
etiol., 579; symp., diag., 580; prog., treat., 582
diff. fr. empyema of the frontal sinus, fr. suppuration of the anterior ethmoid cells, fr. polypus, fr. ozæna, fr. foreign bodies, fr. syphilis, fr. caries, fr. disease of the sphenoidal sinus, 580, 581
Empyema of the frontal sinus, diff. fr. empyema of the antrum, 581
of the sphenoidal sinuses, 583
symp., treat., 583
diff. fr. empyema of the antrum, 581
Encephaloid cancer of the larynx, 476
Endocardial murmurs, 195, 196, 198
Endocarditis, acute, 219–222
ulcerative, 222, 223
chronic, 223–230
Endocardium, the, 178
England, goitre in, 629
Engorgement, in lobular pneumonia, 113
Enlarged bronchial glands, 152, 153
anat., path., etiol., symp., 152; diag., prog., treat., 153
diff. fr. phthisis, 153
glands at the base of the tongue, diff. fr. chronic rheumatic sore throat, 319
tonsils, an obstacle to laryngoscopy, 290
Enlargement of the heart, syn. of simple cardiac hypertrophy, 234
or bulging of the præcordial region, 184
Epidemic catarrh, syn. of influenza, 519
catarrhal fever, syn. of influenza, 519
Epigastric pulsation, 187
Epiglottis, large or pendent, obstructs laryngoscopy, 291; illus., 294, 295
ulceration of the, 395
Epistaxis, 559–563
syn., anat., path., etiol., symp., 559; diag., prog., treat., 560
diff. fr. pulmonary hemorrhage, 136
Epithelioma, 361, 480; diff. fr. lupus of the nares, 588; fr. rhinoscleroma, 589
Erichsen, nasal syphilis, 577
Erysipelatous laryngitis, 428, 429
etiol., symp., diag., prog., 428; treat., 429
sore throat, 314–316
etiol., symp., diag., prog., treat., 315
diff. fr. diphtheria, 332
Erythematous sore throat, syn. of acute sore throat, 311
Ether for general anæsthesia, 582, 618
Ethmoiditis, chronic suppurative, 585–587
Eustachian orifice, 308
Eversion of the ventricle of Morgagni, diag., treat., 483
of the ventricle of the larynx, diff. fr. benign tumors, 469
Evulsion of nasal mucous polypi, 566
of tumors, in the larynx, 473
Exaggerated bronchial whisper, 58
pulmonary resonance, 28
respiration, 42
Examination of the chest, physical, 3–59; of the fauces, 271–310
of the heart, physical, 183–194
of the trachea, illus., 300
Exocardial friction sounds or murmurs, 195
Exophthalmic goitre, 632
syn., 632
Exostosis of the nasal septum, ecchondroma and, 597–601
Exostoses, diff. fr. nasal cartilaginous tumors,

571; fr. bony tumors, 572; fr. foreign bodies, 603
Expiratory power greater than inspiratory, 20
Extirpation of the larynx, partial, complete, 481, 482
Extractor, for intubation, 420; bristle, 642
Exudative bronchitis, syn. of plastic bronchitis, 99
laryngitis, syn. of membranous croup, 411
stage of croup, 412

Fagge, Hilton, surgery in croup, 415
Fahnestock, tonsillitome, illus., 372
False croup, syn. of spasm of the glottis, 496
Falsetto voice, 503, 504
Faradism or faradization, 511, 513, 514, 640
Fasciculated sarcomata, 467
Fatty heart, 242-244
etiol., symp., 242; diag. prog. treat., 244
diff. fr. chronic endocarditis, 226, 227; fr. chronic myocarditis, 232
Fauces, diseases of the, 311-381
examination of the, 271-310
Fauvel, malignant tumors in the larynx, 476
Feeble respiration, 42
Fetid form of tracheitis, 461, 462
Fibrinous bronchitis, syn. of plastic bronchitis, 99
Fibro-cellular tumors, in the larynx, illus., 466
Fibroid degeneration of the lungs, syn. of fibroid phthisis, 156, 167
disease of the heart, syn. of chronic myocarditis, 231
disease of the lungs, diff. fr. emphysema, 111
phthisis, 156, 167-169
syn., 156, 167; anat., path., 167; etiol., symp., 168; prog., 169; treat., 170
diff. fr. other forms, 166, 167
phthisis, syn. of dilatation of the bronchial tubes, 100
tumors, diff. fr. adenoid growths, 616
Fibroma of laryngo-pharynx, illus., 386
of the vocal cords, illus., 466, 467
Fibromata of the nares, syn. of nasal fibrous polypi, 569
Fibro-mucous tumors, retro-nasal, 624, 625
diff. fr. nasal fibromata, 621
Fibrosis, syn. of fibroid phthisis, 167
Fibrous growths, diff. fr. nasal mucous polypi, 566
polypi, nasal, 569
tumors of the naso-pharynx, 620-624
diff. fr. cancer of the pharynx, 387; fr. retro-nasal fibro-mucous tumors, 624
Filer's phthisis, syn. of dilatation of the bronchial tubes, 100
First stage of lobar pneumonia, 117; of pericarditis, 213; of phthisis, 161-164
Fissures, pulmonary, 8
Flat chest, illus., 12
nasal probe, illus., 537
Flatness, hepatic, cardiac, 25, 26
diff. fr. dulness, 29
Flexible œsophageal forceps, illus., 641
Flint, Austin, cyrtometer, illus., 17, 18; hammer and pleximeter, illus., 21; percussion, 25, 26, 28; tympanitic resonance, 66; pulmonary gangrene, 141; pulmonary phthisis, 162
Florida for phthisis, 175
Fluctuation of fluid in the pleural cavity, signs of, 16
Follicular disease of the naso-pharynx, syn. of rhino-pharyngitis, 607
glossitis, acute, chronic, 347, 348
pharyngitis, acute, 339, 340; chronic, 340-346
Fontaine, citric acid in diphtheria, 335
Force of the heart, increased, diminished, 186, 187
Forceps, tonsil, 373; laryngeal, 471; tube, 472; punch, 485; nasal dressing, 576; septum, 596; right angle cutting, 597; removing pharyngeal gland with, 618-620; flexible œsophageal, 641
Foreign bodies in the larynx, 490-492
symp., 490; diag., prog., treat., 491
diff. fr. abscess, 384, 385; fr. acute laryngitis, 396; fr. phlegmonous laryngitis, 428; fr. stenosis of the larynx, 457
bodies in the nose, 603, 604
symp., diag., 603; prog., treat., 604
diff. fr. atrophic rhinitis, 549; fr. empyema, 581; fr. nasal mucous polypi, 565; fr. nasal malignant tumors, 573; fr. exostosis, fr. rhinoliths, fr. simple catarrh, fr. polypi, 603
bodies in the œsophagus, 640-642
symp., 640; diag., prog., 641; treat., 642
diff. fr. stricture of the œsophagus, 635 fr. globus hystericus, fr. paræsthesia, 641
bodies in the pharynx, 382, 383
symp., diag., prog., treat., 383
bodies in the trachea, 492-495
symp., 492; diag., 493; prog., treat., 494
Formula for focal distance of reflector, 276
Formulæ for prescriptions, 645-658
Fornix pharyngis, syn. of vault of the pharynx, 309
Fort, A., electrolysis for stricture of the œsophagus, 637
Fossa innominata, 297
of Rosenmueller, illus., 309
Foster, illustrations of the action of the heart, 208, 210
Fowler, George B., laryngectomy, 483
Fox, Higston, acute tonsillitis, 363
Fracture of the larynx, 489, 490
anat., path., etiol., symp., diag., prog., treat., 489
Fractures of the nose, 593, 594
symp., diag., prog., 593; treat., 594
Fraenkel, diplococcus pneumoniæ, 115; staining bacilli, 165; illuminator, 279, 280; rhinoscope, illus., 303; cause of infantile coryza, 522
Fraentzel, resonance in pleurisy, 66

Frænum obstructs laryngoscopy, a short, 290
France for phthisis, 175; goitre in, 629
Frank, aortitis, 254
Fremitus, normal vocal, 15; friction, bronchial or rhonchial, 16
French, Thomas R., registers of male and female voice, 298
Friction fremitus, 16
sounds or murmurs, 48, 51, 52, 53; exocardial, pericardial, cardiac, 195; endocardial pleuritic, cardio-pleuritic, 196
treatment in laryngeal tumors, 472
Friedländer, diplococcus pneumoniæ, micrococcus, 115
Frog face, 620
Frontal sinus, inflammation of the, 584, 585
Fuming inhalations, formulæ, 654, 655
Functional aphonia, syn. of bilateral paralysis of the lateral crico-arytenoid muscles, 508
disease of the heart, neurotic or, 247–249
Furunculosis of the nose, 558, 559; treat., 558
Fütterer, L. G., treatment of chronic pleurisy, 78

Gags, 419, 618
Gairdner, diagram of physiological action of the heart, 181
Galvano-cautery in various diseases of the throat and the nose, 266, 340, 346, 348, 367, 372, 373, 374, 380, 386, 410, 453, 470, 501, 537, 538, 539, 544, 563, 568, 569, 570, 571, 576, 578, 586, 588, 617, 622
compared with chromic acid, 537
écraseur, 573
handle with écraseur, illus., 567
snare, illus., 623, 624
Gangrene, amphoric resonance in, 31; in lobar pneumonia, 115; pulmonary, 144, 145
Garcia, Manuel, laryngoscopy, 272
Gargles, formulæ, 647
Garland, G. M., curved line of flatness in pleurisy, illus., 64
Gazette des Hôpitaux, sterilized air in pneumothorax, Potain, 88; potassium iodide for angina pectoris, Huchard, 253; removal of foreign bodies with skein of thread, Crequy, 642
Gazette Hebdomadaire, fracture of the larynx, Henoque, 489
Gee, cyrtometer, 17; tympanitic resonance, 30
Generative organs to tonsils, relation of, 375
Georgia mountains for phthisis, 175
Gerhardt, pulmonary emphysema, 109
Germain Sée, lactose diuretic, 230
German mountains for phthisis, 175
student's lamp for laryngeal illumination, 279, 281
Germany for phthisis, 175; rhinoscleroma in, 588
Gibb, erysipelatous laryngitis, 429
Gibbes, Heneage, bacilli, illus. (colored plate), 165; secured immunity to tubercular virus, 172
Giraldes, multiple secretions of pus in the antrum, 579
Glanders, 589, 590
anat., path., etiol., symp., 589; diag., prog., treat., 590
diff. fr. rheumatism, fr. pyæmia, fr. typhoid fever, fr. syphilis, fr. scrofulous eruptions, 590
Glands, enlarged bronchial, 152, 153
enlarged at base of tongue, 319, 389
Gleitsmann, tubercular sore throat, 352
Globe nebulizer, illus., 174
Globus hystericus, 500
diff. fr. foreign bodies in the œsophagus, 641
Glossitis, acute follicular, 347
chronic follicular, 347, 348
Glottis, 298
spasm of the, 496, 497
Goitre, 629–631
syn., anat., path., etiol., 629; symp., diag., prog., treat., 630
diff. fr. exophthalmic goitre, fr. malignant tumors, 630
aerial, 486
exophthalmic, 632
Gold and sodium chloride for immunity to tubercular virus, 172; for syphilitic laryngitis, 448
Gottstein, malignant tumors in the larynx, 476; wool tampons, 552
Gouty affections diff. fr. chronic rheumatic sore throat, 319
Grancher, diphtheria propagated by infected clothing or furniture, 334
Granular sore throat, syn. of chronic follicular pharyngitis, 340
Graves' disease, syn. of exophthalmic goitre, 632
Gray hepatization, 113, 114; illus., 117
Great Lakes, rhinitis near the, 527
Grippe, syn. of influenza, 519
Gross, S. D., foreign bodies, 492, 494; instruments for removing foreign bodies from cavities of nose and ears, illus., 604
Guaiacol, for phthisis, 173
Guaiacum for acute tonsillitis, 366; unsatisfactory in phlegmonous tonsillitis, 369
Gueneau, Noel, diaphragmatic pleurisy, 82
Guido Boeelli, pus diff. fr. serum, 77
Guillotines for throat, 473
Gumma, 353, 354
Gurgles, 48, 52
Gussenbauer, artificial larynx, 482, 483
Guttmann, tympanitic resonance, 30

Hack, hay fever, related to conditions in nasal passages, 553
Hæmadynamometer, 19
Hæmatemesis diff. fr. hæmoptysis, 135
Hæmatoma of the nasal septum, etiol., symp., diag., prog., treat., 602
diff. fr. mucous polypi, fr. cartilaginous tumors, fr. hypertrophy of the turbinated body, fr. ecchondroma, 602

Hæmic murmurs, 204
Hæmoptysis, 134, 135, 259
diff. fr. hæmatemesis, 135; fr. epistaxis, fr. hemorrhage of the gums or the pharynx, 136
Hæmostatics, spray inhalations, formulæ, 653
Hahn, F., nasal tuberculosis, 578
Haines, W. S., iodine trichloride in tubercular laryngitis, 441
Hairy heart, 212
Hamilton, milk spots, 212; pneumo-pericardium, 218; acute endocarditis, 219, 220; myocarditis, 231
Hammer for percussion, 21
Hammond, hæmadynamometer, illus., 19; expiratory force greater than inspiratory, 20
Harkin, epistaxis, 561
Harries and Campbell, etiology of lupus of the larynx, 452
Harsh respiration, syn. of broncho-vesicular or rude respiration, 44
Hay asthma, syn. of hay fever, 553
fever, 553-558
syn., anat., path., etiol., 553; symp., diag., 554; prog., treat., 555
diff. fr. acute rhinitis, 524; fr. simple chronic rhinitis, 529; fr. simple acute rhinitis, fr. spasmodic asthma, 554, 555
Hayden, illustration of motion of the heart, 209, 210
Head, sections of, 302, 541, 579, 584
for laryngoscopy, good and poor positions of, 284, 285
lower than the body in taking food in certain throat diseases, 442, 506
Heart, the, 177-211
aneurism of the, 245
apex beat of the, 10, 180, 182, 184
atrophy of the, 242
congenital deformity of the, 227
diastole of the, 180
dilatation of the, 239-242
failure in atheroma of the aorta, 255
fatty, 242-244; degeneration, infiltration, 242
force of the, modified, 184, 186, 187
hairy, 212
neoplasms of the, 246
neurotic or functional disease of the, 247-249
physical examination of the, 183-194
physiological action of the, 180-183
rupture of the, 245
sounds, how caused, 190, 191; modified by disease, 191-194; anomalous, 205
syphilis of the, 245
systole of the, 180
to find the limits of the, 188
tumors of the, 246
valvular disease of the, 223-230
Heath, Christopher, empyema of the antrum, 582
Helcosis laryngis, syn. of tubercular laryngitis, 434
Hemiplegia causes exaggerated respiration, 42
Hemming, Hugh, syrup of chloral in diphtheria, 336
Hemorrhage, pulmonary, 134-136; after abscission of the uvula, 359; after tonsillotomy, 374
Hemorrhagia narium, syn. of epistaxis, 559
Hemorrhagic infarctus, syn. of pulmonary apoplexy, 137
pleurisy, 61
Henoque, fracture of the larynx, 489
Henrotin, gag, illus., 419, 618
Hepatic dulness, flatness, 25, 26
pulsation, 187
Hepatization, red, yellow, gray, 113, 114
Heredity of phthisis, 158
Hernia, diaphragmatic, 88
Herpetic sore throat, syn. of simple membranous sore throat, 324
ulceration, 395
Herynge (see Krause and Herynge)
Hilton, sacculus laryngis, 297
Himalayas, goitre in the, 629
Hippocrates acquainted with succussion, 20; percussion, 21
Holden, E., chorea laryngis, 501
Home and its comforts best for advanced cases of phthisis, 176
Hooper, F. H., operating on benign tumors in the larynx, 473
Hopmann, nasal papillary tumors, 569
Hospital sore throat, syn. of chronic follicular pharyngitis, 340
Hot applications in pneumonia, 122; in diphtheria, 335; in phlegmonous tonsillitis, 369; in croup, 416; in tracheitis, 461
Hotz, F. C., throat deafness, 611
Huber, myocarditis, 231
Huchard, free protracted use of potassium iodide to cure angina pectoris, 253
Hungary, rhinoscleroma in, 588
Hunter, John, empyema of the antrum, 579, 582
Hutchinson, spirometer, 18
Hydatid cysts of the lungs, 148-150
anat., path., etiol., 148; symp., diag., 149; treat., 150
diff. fr. phthisis, 149; fr. circumscribed pleurisy, 150
Hyde, J. Nevins, treatment of lepra of larynx, 455
Hydrastine identical with berberine, 95
for chronic follicular pharyngitis, 344
Hydro-pericardium or pericardial effusion, 15, 218, 219
anat., path., etiol., symp., diag., 218; prog., treat., 219
diff. fr. hypertrophy and dilatation of the heart, 238
Hydrothorax, 13, 15, 84
etiol., symp., diag., prog., treat., 84
diff. fr. pneumonia, 120; fr. pulmonary collapse, 143
Hyperæmia, pulmonary, 132-134
Hyperæsthesia of the larynx, 82, 500, 501
anat., path., etiol., symp., diag., 500; prog., treat., 501

Hyperæsthesia of the pharynx, 388, 389
Hypersarcosis cordis, syn. of simple cardiac hypertrophy, 234
Hypertrophic rhinitis, illus., 528, 540–547
anat., path., etiol., symp., 540; diag., 542; prog., treat., 543
diff. fr. intumescent rhinitis, 534, 542; fr. syphilis, fr. nasal mucous polypi, 542, 543
Hypertrophy, simple cardiac, 14, 234–236
and dilatation of the heart, illus., 211, 236–239
syn., symp., 236; diag., prog., treat,, 239
diff. fr. retraction or consolidation of the lung, fr. cardiac dilatation, fr. pericardial effusion, fr. cardiac displacement, fr. thoracic aneurism, 237–239
of the larynx, 455
of the liver diff. fr. pleurisy, 70
of Luschka's tonsil, syn. of hypertrophy of the pharyngeal tonsil, 613
of the pharyngeal tonsil, illus., 613–620
syn., anat., path.,613; etiol., symp.,614; diag., prog., treat., 616
diff. fr. nasal mucous polypi, fr. fibroid tumors, 616; fr. fibromata, 621
of the spleen or of the liver, diff. fr. pleurisy, 70
of the tonsils, 370–375
syn., etiol., symp., 370; diag., prog., treat., 371
diff. fr. diphtheria, 332; fr. cancer, 380, 381
of the turbinated body, diff. fr. hæmatoma of the nasal septum, 602
Hypodermic syringe, illus., 568
Hypostatic congestion, 133
Hysteria, diff. fr. chorea laryngis, 502
Hysterical aphonia, syn. of bilateral paralysis of the lateral crico-arytenoid muscles, 508
Hysterical or pseudo angina pectoris, diff. fr. angina pectoris, 251

Ice in diphtheria and other diseases of the throat, 334, 367, 369, 416, 428, 633
Ichthyosis linguæ, syn. of leucoplakia buccalis, 360
Illumination of the throat, 275–384
Immediate auscultation, 34
percussion, 21
Immunity to tubercular virus, how secured, 172
Incipient hypertrophy due to Bright's disease, illus., 210
Incompetency of heart valves produced, 224
Increased vocal resonance, 56
India, myasis narium in, 605
Induration of the lungs, syn. of fibroid phthisis, 167
Infants, syphilitic sore throat in, 356; syphilitic laryngitis in, 449; acute rhinitis in, 526; syphilis of the nose in, 577
Infectious endocarditis, syn. of acute endocarditis, 219
Inferior costal breathing, 11
meatus, illus., 309
sternal region, 4, 6
turbinated bodies, illus., 308
Inflammation of the antrum or frontal sinuses diff. fr. acute rhinitis, 524
of the frontal sinuses, illus., 584, 585
symp., treat., 584
of the larynx, syn. of acute laryngitis, 394
of the lungs, popular name for pneumonia, 113
of the uvula, acute, chronic, 358–360
Influenza, 519–522
syn., anat., path., etiol., symp., 519; diag., 520; prog., treat., 521
diff. fr. rhinitis, fr. inflammation of the larnyx, 521
Infra-axillary region, 4, 8
Infra-clavicular region, 4, 5
Infra-glottic dropsy, syn. of œdema of the larynx, 430
laryngoscopy, illus., 292
Infra-mammary region, 4, 8
Infra-scapular region, 4, 8
Infra-thyroid laryngotomy, 476
Ingals, emballometer, illus., 33; flat trocar, illus., 79; drainage tubes for empyema, illus., 81; nasal speculum, illus., 301; modification of Shurly's battery, illus., 345; cautery electrodes, illus., 346; tonsil forceps, illus., 373; laryngeal applicator, illus., 405; chromic acid applicator, galvano-cautery handle, illus., 409; punch forceps, illus., 485; nasal scissors, illus., 545; nasal syringe, illus.,550; snare, illus., 567; nasal dressing forceps, illus., 576; electric lamp for transillumination, 581; drainage tube for the antrum, illus., 583; septum forceps, illus., septum knife, illus., 596; right-angle cutting forceps, 597; nasal saws, illus., 599; nasal spatula, illus., heavy-bone scissors, illus., nasal bone forceps, illus., 600; post-nasal snare applicator, illus., 623
Inhalations, formulæ, vapor, 649–651; spray, 651–653; dry, 654; fuming, 654, 655
Inhaler, 649, 654
Injections for pleurisy, stimulating, 81
Inspection, 9–14, 86, 88, 183, 184, 272, 302
Insufflations, formulæ, 656, 657
Insufflator, illus., 536
Intensity of sound, 22, 39, 41
of heart sounds, modified by disease, 191
of vocal resonance, modified by disease, 55
Inter-arytenoid fold, illus., 299
Intercostal neuralgia or pleurodynia, diff. fr. pleurisy, 68; fr. pneumonia, 119; fr. angina pectoris, 251
Interlobular emphysema, 107
pneumonia, often included in lobular pneumonia, 123
Intermittent dilatation preferred in stenosis of the larynx, 459
rhythm of the heart, 193

Intermittent venous murmurs, 207
Internal treatment, diphtheria, 337
International clinics, operating on benign tumors in the larynx, F. H. Hooper, 473
Congress Laryngology and Otology, Transactions, myxomata transformed into sarcomata, Schiffers, 566
Journal of Surgery and Antiseptics, nasal vascular tumors, J. O. Roe, 570
Medical Annual, tachycardia, L. Bouveret, 249
Medical Congress, Transactions, epistaxis, Harkin, 561; Walton Brown, 562
Internationale klinische Rundschau, pericarditis, von Stoffela, 214; nasal tuberculosis, Michelson, 578; adenoid growths in deaf-mutes, Wròblewski, 614
Interrupted or cog-wheel respiration, 43
Interscapular region, 4, 7
Interstitial pneumonia, often included in lobular pneumonia, 123
pneumonia, syn. of fibroid phthisis, 128, 167
Intubation in diphtheria, croup, and other throat diseases, 338, 397, 415, 418–421, 428, 429, 432, 450, 453, 458, 459, 472, 484, 490, 513, 515
described, 418–421, 458, 459
instruments, 418
Intumescent rhinitis, 528, 531–540
anat., path., etiol., symp., 531; diag., prog., treat., 534
diff. fr. simple chronic rhinitis; fr. nasal mucous polypi, 534; fr. hypertrophic rhinitis, 534, 542
Inversion of a patient to remove foreign bodies from the trachea, 491
Involution of the trachea, 485, 486
etiol., symp., diag., prog., treat., 486
Iodine for immunity to tubercular virus, 172; for tuberculosis, 631
trichloride in surgery, 441
Inspiratory power less than expiratory, 20
Irritability of the tongue remedied for rhinoscopy, 304
Irritable fauces an obstacle to laryngoscopy, 289; remedied, 305
heart of soldiers, 249
Irritative cough, treat., 498
Italy, rhinoscleroma in, 588; goitre in, 629

Jaccoud, pleurisy, 83
Jackson, Hughlings, nose-bleeding preceding apoplexy, 560
Japan, distoma pulmonale, 150
Jarvis, small nasal speculum, illus., 301; tubercular laryngitis, 441; snare forceps, 473; rhinitis, 545; snare, 567; nasal vascular tumors, 570; drill, 598
Jaworski, pneumonia contagious, 116
Johnson, H. A., inspection in phthisis, 162
Journal American Medical Association, pneumonia contagious, Jaworski, 116
de Médecine de Paris, epileptic asthma, Poulet, 104
Journal of Laryngology, lepra of the larynx, Morell Mackenzie, 454
Jugular veins, collapse of the, 207
June cold, syn. of hay fever, 553

Keloid diff. fr. rhinoscleroma, 589
Kennedy, fatty heart, 242
Klebs-Löffler bacillus a cause of diphtheria, 329
Klemperer, G. and F., experiments with blood serum or anti-pneumatoxin in pneumonia, 123
Knife, laryngeal, 474; septum, 596, 599
Knife-grinder's rot, syn. of dilatation of the bronchial tubes, 100
Knight, stethoscope, illus., 36
Charles H., galvano-cautery in chronic follicular tonsillitis, 372; nasal osseous cysts, 570
F. I., lupus of the larynx, 451; chorea laryngis, 501, 502; laryngeal vertigo, 504
Koch, bacilli in lupus of the larynx, 451
tubercle bacillus, 159
tuberculin, disastrous use of, 454; in tuberculosis of nares, 579; curative in lupus of the nares, 588; inactive in rhinoscleroma, 589
König, canula, 486, 488
Kramer, head-band for reflector in laryngoscopy, 277
Krause and Herynge, treatment of acute tubercular sore throat, 352
operations on the antrum, 582
Krishaber, illuminator, illus., 278; thyrotomy, 475
Kuhn, pneumonia contagious, 116

Labus, Carlo, trachoma of the vocal cords, 408
Lactic acid in diseases of the throat and nose, 335, 336, 380, 381, 417, 578
Lactose diuretic, 230
Laennec, theory of the cause of pulmonary emphysema, 20; mediate auscultation, 34; bronchial respiration, 45; râles, 51
La France Médicale, carbon dioxide in asthma, Weill, 106
Lamp for laryngoscopy, German student's, 279; for transillumination of the nasal cavities, electric, 581
Lancet, laryngeal, 397
Larry, aerial goitre, 486
Laryngeal and tracheal respiration, 41
applicator, 405
cough, 59
electrodes, 509
forceps, illus., 471
knives, 474
lancet, 397
phthisis, syn. of tubercular laryngitis, 434
tuberculosis, syn. of tubercular laryngitis, 434
tubes, 418, 459 (see intubation)
tumors, illus., 463–485
diff. fr. syphilis, 447
vertigo, treat., 504

Laryngectomy, modes described, 482, 483
Laryngismus stridulus, syn. of spasm of the glottis, 496
Laryngitis, acute, 393–397
chronic, 398–408
chronica, syn. of chronic laryngitis, 398
due to small-pox, 455
erysipelatous, 428, 429
exudative, syn. of membranous croup, 411
of measles, 455
of scarlet fever, of small-pox, 455
phlegmonosa, syn. of phlegmonous laryngitis, 427
sero-purulenta, syn. of phlegmonous laryngitis, 427
subacute, 397, 398
submucosa purulenta, syn. of phlegmonous laryngitis, 427
syphilitic, 443–450; in infants, 449, 450
traumatic, 398
tubercular, 434–443
Laryngo-pharyngeal sinuses, 296
Laryngophony, 54
Laryngoscope, a, 272; preferred form, 282; manipulation of, 283–289
Laryngoscopic mirror in position, illus., 286
Laryngoscopic reflector, illus., 283
Laryngoscopy, illus., 272–292
infraglottic, 292
obstacles to, 289–292
Laryngotomy, supra-thyroid, infra-thyroid, 475, 476, 642
Larynx, a normal, illus., 293, 295; of women, in forming head tones, illus., 298
abscess of the, 429, 430
anæsthesia of the, 499, 500
artificial, 482
benign tumors of the, 465–476
cancer of the, 476–483
chronic stenosis of the, 456–459
cystic growths of the, 466
diseases of the, 394–515
dislocation of the, 490
extirpation, partial, complete, 481–483
foreign bodies in the, 490–492
fracture of the, 489, 490
hyperæsthesia of the, 500, 501
hypertrophy of the, 455
illumination of the, 275–283
lepra of the, 454
lupus of the, 451–454
malignant tumors of the, 476–483
morbid growths of the, 463–483
neuralgia of the, 500, 501
œdema of the, 430–433
paræsthesia of the, 500, 501
resection of the, 481
spasm of the, in adults, 497, 498
ventricles of the, 297
La Semaine Médicale, causes of angina pectoris, 251
Lateral region, 3
La Tribune Médicale, lactose diuretic, Germain Sée, 230
Laugenbeck, retro-nasal fibrous tumors, 621
Lawrence, retro-nasal fibrous tumors, 621
Lazarus, heredity in asthma, 103
Leared, binaural stethoscope, 35
Lefferts, George M., history of lupus in the larynx, 451; eversion of the ventricles of Morgagni, 483; chorea laryngis, 501; retro-nasal cystic tumors, 626
Leichtenstern, pleurisy, 71; empyema in children, 77
Leidy, Joseph, thyrotomy, 475
Leiter coil for applying cold through a circulation of water, in tonsillitis, 369; in croup, 416
Lepra of the larynx, illus., 454, 455
path., etiol., symp., diag., prog., treat., 454
diff. fr. benign tumors, 469
Leptothrix buccalis, 376
Leucoplakia buccalis, 360–362
syn., anat., path., etiol., 360; symp., diag., 361; prog., treat., 362
diff. fr. professional patches, 357; fr. smoker's patches, fr. mercurial patches, fr. syphilitic patches, fr. cancer, fr. psoriasis linguæ, 361, 362
buccalis et lingualis, syn. of leucoplakia buccalis, 360
Levret, laryngoscopy, 272
Lewin, benign growths in the larynx, 465
Lewis, foreign bodies in the trachea, 492
Leyden, cause of asthma, 103
Liégeois, cause of angina pectoris, 250
Ligation for extirpation of tumors, 622
Lime-water vapors in diphtheria, 416
Lincoln, R. P., nasal cancerous tumors, 573; extirpation of nasal tumors, 621
Linsley's translation Fränkel's Bacteriology, staining bacilli, 165
Lipomata, 467
Liston, laryngoscopy, 272
Litten, pulmonary thrombosis and embolism, 138
Liver, enlargement or hypertrophy of, 68, 70
Lobar pneumonia, 113–123
syn., anat., path., 113; etiol., 115; symp., 116; diag., 119; prog., 121; treat., 122
diff. fr. capillary bronchitis, 97; fr. lobular pneumonia, 127
Lobular pneumonia, 123–128
syn., anat., path., 123; etiol., symp., 124; diag., 125; prog., 127; treat., 128
diff. fr. capillary bronchitis, 97; fr. capillary bronchitis, fr. pulmonary collapse, fr. lobar pneumonia, fr. acute tubercular phthisis, 125–128
Local anæsthesia produced by a pigment of morphine, carbolic acid, tannic acid, glycerin, water, 442
anæsthesia, produced by cocaine, 457, 470, 495, 544, 557, 568, 582, 603, 616, 617, 623
anæsthesia, pigments, formulæ, 655
Loewenberg, forceps, illus., 617
London Hospital Clinical Lectures and Reports, nose-bleeding preceding apoplexy, Hughlings Jackson, 560
Lancet, diagnosis of congenital disease of

the heart in children, Sansom, 246; removing foreign bodies from the trachea, Padley, 494; goitre, Morell Mackenzie, 631; tube used in stricture of the œsophagus, Charters J. Symonds, 636
London Practitioner, treatment of ulcerative endocarditis, Sansom, 223
Loomis, A. L., percussion sounds, 28; treatment of pleurisy, 72, 78; double pneumonia, 115; treatment of pulmonary hemorrhage, 136; mortality in infants from atelectasis following bronchitis, 141; rhythm of heart sounds, illus., 183; reduplication of heart sounds, 194; endocarditis, 221; simple cardiac hypertrophy, 234; thoracic aneurisms, 265
Henry P., bacilli in healthy persons, 159
Lozenges, trochisci or, formulæ, 647-649
Lubet-Barbou, spasm of the glottis, 496
Lumniczer, Josef, cause of putrid bronchitis, 91
Lung fever, popular syn. of pneumonia, 113
Lungs, apoplexy of the, 15
collapse of the, 70
consolidation of, 237, 264
hydatid cysts of the, 148-150
retraction of the, 237
syphilitic disease of the, 151, 152
L'Union Médicale, Klebs-Löffler bacillus, Roux and Yersin, 329; case of excessive nose-bleeding, Martineau, 560
Lupus exedens, non-exedens, 587
of the larynx, illus., 451-454
anat., path., etiol., 451; symp., diag., 452; prog., treat., 453
diff. fr. tuberculosis, fr. syphilis, fr. cancer, 453, 454, 479; fr. benign tumors, 469
of the nares, 587, 588
anat., path., etiol., symp., diag., 587; prog., treat., 588
diff. fr. atrophic rhinitis, 549; fr. syphilis, fr. epithelioma, fr. tuberculosis, 587
of the pharynx, diff. fr. scrofulous sore throat, 349
vulgaris, 549
vulgaris laryngis, Chiari and Riehl, 451
Luschka's tonsil (see hypertrophy of the pharyngeal tonsil)
Lyon Médicale, cause of angina pectoris, 251

McBride, anæsthesia of the larynx, 499; empyema of the antrum, 580
McDonald, Greville, atrophic rhinitis most common in girls, 547; atrophic rhinitis, 552; nasal osseous cysts, 570; empyema of the antrum, 581; hypertrophy of the pharyngeal tonsil, 614
Mackenzie, John N., syphilitic sore throat in infants, 356; syphilitic laryngitis in infants, 449; hay fever related to condition in nasal passages, 553; forceps, 617
Mackenzie, Morell, rack movement bull's-eye condenser, illus., 278, 279; fossa innominata, 297; erysipelatous sore throat, 316; lactic acid in diphtheria, 335; syphilitic sore throat, 356; laryngeal lancet, illus., 397; identity of diphtheria and croup, 411; syphilitic laryngitis, 443, 445; lepra of the larnyx, 454; laryngeal dilator, illus., 458; laryngeal tumors, 463, 465; tube forceps, illus., 472; guarded wheel écraseur, 474; thyrotomy, 475, 476; laryngeal cancer, 477; mode of complete extirpation of the larynx, 482; tracheocele, 486; syphilis of the trachea, 487; laryngeal electrodes, illus., 509; rhinitis, 525; hay fever, 556; mucous polypi, 565; nasal papillary tumors, 569; nasal syphilis, 574, 577; tonsillitis, 574; anosmia, 592; deflection of the septum, 594; rhino-pharyngitis, 607, 609; throat deafness, 613; goitre, 631; paralysis of the œsophagus, 639, 640; electric inhaler, 649
MacNamara, epistaxis, 561
Maggots in the nose, syn., of myosis narium, 605
Malformations and new growths of the uvula, 359
Malignant (see also cancer) disease of the œsophagus diff. fr. paralysis, 639
endocarditis, syn. of acute endocarditis, 219
growths on uvula, 360
tumors diff. fr. benign tumors, 469
tumors, nasal, 572, 573
tumors of the larynx, illus., 476, 483
anat., path., symp., 476; diag., 479; prog., treat., 480
diff. fr. syphilis, fr. chronic catarrhal inflammation, fr. lupus, fr. tubercular laryngitis, fr. benign growths, 478, 479
tumors of the naso-pharynx, anat., path., etiol., symp., diag., prog., treat., 625
diff. fr. retro-nasal fibro-mucous tumors, 625; fr. nasal cartilaginous tumors, 571
Mammary or nipple line, 6
region, 4, 5
Marey, sphygmograph, illus., 208
Martineau, case of excessive nose-bleeding, 560
Massage with foreign bodies in the œsophagus, 642
Matheson, pneumonia, contagious, 116
Mathieu, tonsillitome, illus., 372
Measles, sore throat of, 222, 323; laryngitis due to, 455; nasal affections in, 591
Meatus, inferior, middle, superior, 309
Mediastinal tumors, solid, 193, 267, 268
diff. fr. pericarditis, 216
Mediate auscultation, 34
percussion, 21
Medical News, danger in washing pleural cavity, Bowditch, resection of ribs, W.

M. Strickler, 78; promotion of renal secretion in children with capillary bronchitis, 98; asthma due to poison in the blood, Robinson, 104;
Medical Press and Circular, pneumonia — contagious, Mosler, 116
Record, acute pleurisy, Drzewiecki, 72
Register, pneumonia contagious, Wells, 116
Society of London, Transactions, acute tonsillitis, Higston Fox, 363
Membranous croup, 14, 411–426
syn., anat., path., etiol., 411; symp., diag., 412, 413; prog., 415; treat., 416
diff. fr. acute laryngitis, 396; fr. catarrhal laryngitis, fr. laryngismus stridulus, fr. diphtheria, 413–415
laryngitis, syn. of membranous croup, 411
sore throat, simple, 324–327
Meningitis diff. fr. pneumonia, 121
Mensuration, 9, 16–20, 86
Menthol and albolene spray, 441, 551
Mercurial patches diff. fr. leucoplakia buccalis, 361
Mercury to infants, mode of applying, 577
Mesosternal line, 7
Metallic tinkling, 20, 54, 87
Mexico for phthisis, 175; nasal syphilis in, 574; myasis narium in, 605
Michelson, nasal tuberculosis, 578
Michigan for hay fever, 555
Micrococcus of Friedlander exciting pulmonary inflammation, 15
Microscopic examination, lobar pneumonia, 114
Middle meatus, illus., 309
turbinated bodies, illus., 308
Miliary tuberculosis, acute, 165–167
Milk most important nutritious drink in diphtheria, 334
spots, 212
Minnesota for phthisis, 175
Minot, pneumonia in children, 115
Mirrors for laryngoscopy, throat, 273; position for, manipulation of, 286–289
Mitral area, illus., 198
constriction, illus., 210
murmurs, 198, 201
obstruction, 225, 228, 230
regurgitation, illus., 209, 225, 228
stenosis, 225
valves, 7, 178
Moist râles, 48, 50
Montana for phthisis, 175
Morbid growths in the larynx, 14, 463–485
anat., path., etiol., 463; symp., 464
Morbus cæruleus, 246, 247
syn., symp., diag., 246; prog., treat., 247
Morgagni, eversion of the ventricle of, 483
Morsen, creasote for phthisis, 173
Mosetig-Moorhof mode of injecting iodoform in goitre, 631
Mosler, pneumonia contagious, 116
Mountains for phthisis 175; for hay fever, 555
Mount Bleyer, tongue depressor, illus., 464
Moure, regeneration of atrophied structure, 550
Mucous click, 48, 52
patches, 353
polypi, myxomata or true, 466
polypi, nasal, 564–568
râles, 48, 50
tubercles, 353
Mulhall, J. C., falsetto voice, 503
Multilocular pleurisy diff. fr. other forms, 83
Münchener medicinische Wochenschrift, the aneurismatiscope, Ferdinand Schnell, 261
Murmurs, vesicular, 39; cardiac, 195–211; exocardial or pericardial friction sounds or, 195; endocardial, 196; diastolic, 203, 204; ventricular, congenital hæmic, 204; subclavian, 206
Myalgia diff. fr. angina pectoris, 251
Myasis narium, 605, 606
syn., etiol., symp., diag., prog., 605; treat., 606
Mycosis of the tonsils, 376, 377
anat., path, etiol., symp., diag., 376
diff. fr. acute and chronic follicular tonsillitis, 376, 377
Myocarditis, 213, 231–233
anat., path., etiol., symp., 231; diag., prog., treat., 232
Myxomata or true mucous polypi, illus., 466

Nares, tuberculosis of the, 578, 579; lupus of the, 587, 588
Nasal affections in acute diseases, 591
bone forceps, 600
bones, dislocation of the, 594
bony tumors, 571, 572
syn., anat., path., etiol. symp., 571; diag., prog., treat., 572
diff. fr. exostoses, fr. rhinoliths, fr. cancer, 572
burrs, illus., 546
cartilaginous tumors, syn., anat., path., symp., diag., prog., treat., 571
diff. fr. fibrous polypi, fr. malignant tumors, fr. exotoses, fr. ecchondroses, fr. bony tumors, 571
cavities, diseases of the, 519–606
douches, illus., 551
douches, formulæ, 658
dressing forceps, 576
fibrous polypi, syn., treat., 569
malignant tumors, 572, 573
anat., path., 572; etiol., symp., diag., prog., treat., 573
diff. fr. rhinoliths, fr. foreign bodies, fr. abscess, fr. benign growths, 573
mucous polypi, 564–568
syn., anat., path., etiol., symp., 564
diff. fr. intumescent rhinitis, 534; fr. hypertrophic rhinitis, 543; fr. deviation of the septum, fr. thickening of the turbinated bodies, fr. chronic abscess of the nasal septum, fr. foreign bodies in the nose, fr. fibrous, sarcomatous, and can-

cerous growths, 565; fr. empyema, 581; fr. chronic suppurative ethmoiditis, 585; fr. hæmatoma, 602; fr. foreign bodies, 603; fr. hypertrophy of the pharyngeal tonsil, 616; fr. retronasal fibrous tumors, 621; fr. retronasal fibro-mucous tumors, 625
Nasal myxomata, syn. of nasal mucous polypi, 564
osseous cysts, anat., path., etiol., symp., diag., treat., 570
papillary tumors, 569, 570
syn., anat., path., symp., diag., prog., treat., 569
probe, flat, 537
saws, 599, 600
scissors, 545, 600
septum, deflection of the, 594; ecchondroma and exostosis of the, 597; perforation of the, 601; hæmatoma of the, 602; abscesses of the, 603
snare, 359, 567
spatula, 600
speculum, 301
spud, illus., 599
syringe, 550
trephines, illus., 546
vascular tumors, syn., treat., 570
Naso-pharynx, cystic tumors of the, 626
diseases of the, 607–626
malignant tumors of the, 625
Natural light for laryngoscopy, 282
Navratil, dilator, 457
Nebraska for phthisis, 175
Neoplasms of the heart, rare, 246; of the larynx, 464
Nervous aphonia, syn. of bilateral paralysis of the lateral crico-arytenoid muscles, 508
cough, treat., 498, 499
Netter, diplococcus pneumoniæ, 115
Neuralgia, intercostal, 68, 252
of the larynx., 500, 501
anat., path., etiol., symp., diag., 500; prog., treat., 501
diff. fr. chronic rheumatic sore throat, 319
of the pharynx, treat., 389
Neuroses of the pharynx, 388–392
Neurotic or functional disease of the heart, 247–249
etiol., symp., 247; diag., prog., treat., 248
diff. fr. chronic endocarditis, 226
Newcomb, James E., electrolysis in disease of septum, 601
New Hampshire for hay fever, 555
New Mexico for phthisis, 175; rhinitis in, 527
New York Medical Journal, pneumonia, infective, Delafield, 115; pneumonia, contagious, Wells, 116; acute tubercular sore throat, Gleitsmann, 352; electricity in rhinitis, D. Bryson Delavan, 552; nasal vascular tumors, J. O. Roe, 570
New York Medical Record, iodide trichloride in surgery, Wm. T. Belfield, 441; eversion of the ventricles of Morgagni, 483; fracture of the nose, J. O. Roe, 594; electrolysis in disease of septum, James E. Newcomb, 601
Night sweats remedied, 171
Nipple line, mammary or, 6
Nitroglycerine for angina pectoris, 252; atheroma of the aorta, 256
Nitrous oxide gas for anæsthetic in aspiration in empyema, 80
Normal bronchial whisper, 58
bronchophony, 55
radical pulse, illus., 208
vesicular resonance, 25
vocal fremitus, 15
vocal resonance, 54, 55
North Carolina mountains for phthisis, 175
Nose bleeding, syn. of epistaxis, 559
congenital deformity of the, 593
diseases of the, 518–626
foreign bodies in the, 603, 604
fractures of the, 593, 594
furunculosis of the, 558, 559
syphilis of the, 574–577; congenital, 577
Nottinghamshire, goitre in, 629

Obstacles to laryngoscopy, 289–292
to posterior rhinoscopy, 304–306
Obstruction, aortic, mitral, tricuspid, pulmonic, 225, 226, 228, 230
Obturator for intubation tubes, illus., 418
Odontological Society Transactions, empyema of the antrum, Christopher Heath, 582
O'Dwyer, Joseph, intubation, 338, 415, 490; intubation instruments, illus., 418, 420; laryngeal tubes, 433, 434, 449, 457, 459, 485, 488, 470, 472
Œdema glottidis, syn. of œdema of the larynx, 14, 430
of the larynx, 430–433
syn., 430; etiol., symp., 431; prog., treat., 432
diff. fr. retropharyngeal abscess, 384; fr. chronic laryngitis, 402, 403; fr. tubercular laryngitis, 439
of the uvula, acute inflammation and, 358
pulmonary, 15, 42, 142–144
Œdematous laryngitis, syn. of œdema of the larynx, 430
Œnothera biennis unsatisfactory with pertussis, 155
Oertel, carbolic acid in diphtheria, 336; pilocarpine in diphtheria, 337
Œsophageal bougie, 635
forceps, flexible, 641
tube, 387, 388, 392
Œsophagismus, syn. of spasm of the œsophagus, 637
Œsophagitis, 632–634
acute, 632, 633
chronic, 633, 634
Œsophagotome, 636
Œsophagotomy, 642

Œsophagus, compression of the, 637
diseases of the, 632–643
foreign bodies in the, 640–642
paræsthesia of the, 642, 643
paralysis of the, 638–640
spasm of the, 637, 638
stricture of the, 634–637
Oil atomizer, 536
Olivary bougies, 635
Ollier, retro-nasal fibrous tumors, 621, 622
Opiates prohibited in capillary bronchitis, 98
Opium objectionable in pneumonia, 123
Orth, gangrene in lobar pneumonia, 115
Osseous cysts, nasal, 570
tumors diff. fr. nasal mucous polypi, 566
Osteoma diff. fr. rhinoliths, 605
Osteomata of the nose, syn. of nasal bony tumors, 571
Outgrowths diff. fr. benign tumors, 469
Owsley, F. D., spray of solution of cloves in laryngitis, 442
Oxyhydrogen light for laryngeal illumination, 275
Ozæna diff. fr. empyema of the antrum, 581

Packing nasal cavities to check bleeding, 619
(See Plugging; see Tampon)
Padley, method of removing foreign bodies from the trachea, 494
Palasciano, fibromata, 621
Palate retractors, 306
ulcerative destruction of, 354
Pallor in chronic pulmonary disease, 11
Palpation, 9, 14–16, 185
Panas, fracture of the larynx, 489
Papillary growths on the uvula, 359
Papillomata of the larynx, illus., 465, 476
of the nares, syn. of nasal papillary tumors, 569
Paræsthesia of the larynx, 500, 501
anat., path., etiol., symp., diag., 500; prog., treat., 501
of the œsophagus, 642, 643
etiol., symp., diag., prog., treat., 643
diff. fr. foreign bodies, 641
Paræsthesia of the pharynx, 389
etiol., prog., treat., 389
Paralysis of the abductors diff fr. stenosis of the larynx, 457 (see Paralysis of the posterior crico-arytenoid muscles)
of the arytenoid muscles, symp., diag., treat., 511
of the crico-thyroid muscles, illus., symp., diag., prog., treat., 506
of the œsophagus, 638–640
anat. path., 638; etiol., symp., diag., 639; prog. treat., 640
diff. fr. spasm of the pharynx, 390; fr. stricture of the œsophagus, 635; fr. spasm, fr. malignant disease, 639, 640
of the pharynx, 391, 392
etiol., symp., diag., prog., 391; treat., 392
diff. fr. spasm of the pharynx, 390
of the posterior crico-arytenoid muscles, bilateral, 511–513; unilateral, 514
Paralysis of the posterior crico-arytenoid muscles, diff. fr. stenosis of the larynx, 457
of the thyro-arytenoid muscles, illus., 507, 508
anat., path., etiol., symp., diag., prog., treat., 507
of the thyro-epiglottic and ary-epiglottic muscles, 505, 506
etiol., symp., diag., prog., treat., 505
of the vocal cords, diff. fr. acute laryngitis, 396; fr. chronic laryngitis, 402
Parosmia, diag., treat., 591
Partial extirpation of the larynx described, 481
Passive aneurism of the heart, syn. of dilatation of the heart, 239
hyperæmia, 133
Pathological Society Transactions, men more affected by plastic bronchitis, Peacock, 99
Pear-shaped chest, 10
Pectoriloquy, 55, 57; whispering, aphonic, 58
Pendent epiglottis an obstacle to laryngoscopy, 291
Percussion, 9, 21–33, 63, 85, 86, 88, 188; mediate, immediate, 21; in health, 21–27; in disease, 28–31; auscultatory, 32, 33
Perforated concave reflector, 275–278
Perforating ulceration in syphilitic sore throat, illus., 353
Perforation of the nasal septum, 601, 602
treat., 601
Pericardial effusion and hydro-pericardium diff. fr. eccentric cardiac hypertrophy, 238
friction sounds or murmurs, 195
Pericarditis, 13, 212–217
anat., path., 212; etiol., symp., 213; diag., 215; prog., treat., 216
diff. fr. pleurisy, 68, 215; fr. endocarditis, fr. mediastinal tumors, 216; fr. endocarditis, 220; fr. chronic endocarditis, 226, 227; fr. hypertrophy and dilatation of the heart, 238; fr. dilatation of the heart, 241
fibrinosa, serosa, 212
Pericardium, the, 177
Perichondritis of the laryngeal cartilages, chondritis and, 433, 434
Peri-pneumonia, peri-pneumonia vera, syn. of pneumonia, 113
Pertussis or whooping-cough, 153–155
anat., path., 153; etiol., symp., diag., prog., 154; treat., 155
Perverted sense of smell, 591, 592
Peter, M., devised the plessigraph, 31; pulsation on back of hands, 207
Phagedenic ulceration, 354
Pharyngeal bursa, illus., 309
tonsil, hypertrophy of the, 613–620
Pharyngitis, acute follicular, 339, 340
chronic follicular, 340–346
sicca, or atrophic follicular, 543
Pharynx, anæsthesia of the, 388
and posterior nasal cavities, vault of the, illus., 307–310

Pharynx, cancer of the, 386, 387
diseases of the, 382-393
foreign bodies in the, 382, 383
hyperæsthesia of the, 388, 389
lupus of the, 349
neuralgia of the, 389
neuroses of the, 388-392
paræsthesia of the, 389
paralysis of the, 391, 392
scalds and burns of the, 392
spasm of the, 390
tumors of the, 386
Phlebectasis laryngea, anat., path., etiol., symp., diag., treat., 409
Phlegmonous laryngitis, 427, 428, 431
syn., etiol., symp., diag., 427; prog., treat., 428
diff. fr. laryngismus stridulus, fr. retro-pharyngeal abscess, fr. foreign bodies in the larynx, fr. diphtheritic laryngitis, 427, 428
sore throat, syn. of phlegmonous tonsillitis, 368
tonsillitis, 368-370
syn., anat., path., etiol., symp., diag., 368; prog., treat., 369
diff. fr. diphtheria, 332; fr. acute tonsillitis, 365
Phthisis infectious, 170
fibroid, 167-169
pulmonary, 13, 15, 29, 31, 161-164
of the heart, syn. of atrophy of the heart, 242
Physical diagnosis, 3-59
examination, methods of, 9-58
examination of the heart, 183-194
Physiological action of the heart, illus., 180-183
Physiology of the heart, anatomy and, 177-180
Pigeon breast, illus., 12
Pigments, formulæ, 656, 656
Pilocarpine in diphtheria, 337; in erysipelas, 429; in œdema of the larynx, 432
Pincette, 291
Pineapple juice in diphtheria, 335
Pins, E., pericarditis, 214
Piorry, mediate percussion, 21
Pitch of sound, 22, 39
of heart sounds modified by disease, 191
Pityriasis as a sign, 11
Plastic bronchitis, 99, 100
syn., anat., path., etiol., symp., 99; prog., treat., 100
diff. fr. pleurisy, fr. pneumonia, 99
or dry pleurisy, 61
Plessigraph, the, 31
Plessimeter, pleximeter or, 21
Pleurisy, acute, 61-72
bilocular, 83
circumscribed, 82, 150
diaphragmatic, 82
hemorrhagic, 61
of the apex, 82
or empyema, chronic, 76-82
or pleuritis, 12, 29, 60-84
anat., path., 60
diff. fr. plastic bronchitis, 99; fr. pneumonia, 110; fr. pulmonary collapse, 141
Pleurisy, plastic or dry, 61
subacute, 72-75
multilocular, 83
unilocular, 83
sero-fibrinous, 61
Pleuritic friction sounds diff. fr. pericardial, 196
Pleuritis, pleurisy or, 60-84
Pleurodynia or intercostal neuralgia, diff. fr. pleurisy, 68; fr. pneumonia, 119
Pleurotomy, 78
Pleximeter, 21, 22
Plugging for epistaxis, 561-563, 623, 624
Pneumococci in endocarditis, 222
Pneumo-hydropericardium, etiol., symp., diag., prog., treat., 218
Pneumo-hydrothorax, illus., 85-88
diag., 87; treat., 88
diff. fr. emphysema, fr. chronic pleurisy, fr. diaphragmatic hernia, 88
Pneumonia, 113-129
syn., 113
diff. fr. pleurisy, 69; fr. plastic bronchitis, 99; fr. pulmonary œdema, 119, 143; fr. abscess of the lung, 130; fr. pulmonary collapse, 141
bilious, 129
chronic or interstitial, typhoid, 128
from disease of the heart, from Bright's disease, 128, 129
lobar, 113-123
lobular, 123-128
Pneumo-hydropericardium, etiol., symp., diag., prog., treat., 218
Pneumo-hydrothorax, 85
diag., 87; prog., treat., 88
diff. fr. emphysema, fr. chronic pleurisy, fr. diaphragmatic hernia, 87, 88
Pneumothorax, 13, 15, 31, 84, 85
etiol., 84; symp., 85; diag., 87; prog., 88; treat., 88
diff. fr. emphysema, 87; fr. chronic pleurisy, fr. diaphragmatic hernia, 88; fr. emphysema, 110
Pneumonorrhagia, syn. of pulmonary apoplexy, 134, 137
Pocket tongue-depressor, illus., 271
Polasciano, retro-nasal fibrous tumors, 621
Polikier, B., foreign bodies in œsophagus, 642
Polypi, nasal fibrous, 569
nasal mucous, 564-568
Polypus, diff. fr. phlegmonous laryngitis, 428
Porcher, self-retaining uvula and palate retractor, illus., 306
Position for rhinoscopy, illus., 304
Positions of head for laryngoscopy, good, poor, illus., 284, 285
Posterior crico-arytenoid muscles, bilateral paralysis of, 511-513
region, 3
rhinoscopy, illus., 302-306
Post-nasal catarrh, syn. of rhinopharyngitis, 607
snare applicator, 623

Post-nasal syringe, illus., 609
Post-tracheotomy vegetations, 485
etiol., symp., diag., prog., treat., 485
Potain, use of sterilized air in pneumothorax, 88
Potassium iodide for angina pectoris, 247, 253
Poulet, epileptiform asthma, 104
Powder-blower for insufflation, illus., 536
Powell, R. Douglas, siphon drainage in pleurisy, 79; cause of angina pectoris, 250; aortitis, 254
Prentiss, classification of causes of slow pulse, 250
Prescriptions, formulæ for, 645–658
Presystolic venous pulsation, cause of, 207
Probang, cotton, 405
Probe, flat nasal, 537
Processus vocales, the, illus., 299
Professional patches, diff. fr. leucoplakia buccalis, 361
Progressive bulbar paralysis, 391
Prolonged interval between inspiration and expiration, cause of, 43
respiration, cause of, 44
Prophylactic treatment most important for distoma pulmonale, 151; for acute rheumatic sore throat, 321; for diphtheria, 333, 334; for rhinitis in catarrhal tendencies, 534
Prophylaxis in phthisis, 170; in rhino-pharyngitis, 609
Prudden, T. M., streptococcus of diphtheria, 329
Pseudo-angina pectoris, diff. fr. angina pectoris, 252
Pseudo-apoplexy, 243
Pseudo-diphtheria, 329
Pseudo-membranous bronchitis, syn. of plastic bronchitis, 99
Psoriasis linguæ, diff. fr. leucoplakia buccalis, 361
Pulmonary apoplexy, 29, 137, 138
syn., anat., path., etiol., symp., 137; diag., treat., 138
area, illus., 198, 199
artery, 180; aneurism of the, 264, 265
cancer, 146–148
anat., path., etiol., symp., 146; diag., 147, prog., treat., 148
diff. fr. chronic pleurisy, fr. phthisis, fr. aortic aneurism, 148
Pulmonary collapse, 139, 142
syn., anat., path., 139; etiol., symp., 140; diag., prog., treat., 141
diff. fr. lobar pneumonia, 120; fr. lobular pneumonia, 125; fr. pneumonia, fr. pleurisy, 141
Pulmonary emphysema, 12, 107–112
anat., path., 107; etiol., symp., 108; diag., 110; prog., treat., 112
diff. fr. chronic bronchitis, 93; fr. asthma, 105; fr. pneumothorax, 110; fr. acute tuberculosis, fr. fibroid disease of the lungs, fr. asthma, 111
fissures, 8
gangrene, 144, 145
Pulmonary gangrene, anat., path., 144; etiol., symp., diag., prog., treat., 145
diff. fr. phthisis, fr. bronchitis, fr. dilatation of the bronchial tubes, 145
hemorrhage, 134–136
syn., anat., path., 134; etiol., symp., diag., 135; prog., treat., 136
diff. fr. bronchitis, 93, 94; fr. hæmatemesis, fr. epistaxis, fr. hemorrhage from the gums or the pharynx, 135, 136
hyperæmia, 132–134
anat., path., 132; etiol., symp., prog., 133; treat., 134
œdema, 30, 142–144
anat., path., etiol., 142; symp., diag., prog., treat., 143
diff. fr. capillary bronchitis, 97, 143; fr. pneumonia, 120, 143; fr. pneumonia, fr. hydrothorax, 143
phthisis, 13, 156–176
syn., 156; prog., 169; treat., 170
diff. fr. pleurisy, 69; fr. bronchitis, 93, 94; fr. capillary bronchitis, 98; fr. bronchiectasis, 101; fr. pneumonia, 120; fr. pulmonary gangrene, 145; fr. pulmonary cancer, 147; fr. hydatid cysts of the lungs, 149; fr. syphilitic disease of the lungs, 151; fr. enlarged bronchial glands, 153
resonance, exaggerated, 28
semilunar valves, 178
thrombosis and embolism, 138, 139
anat., path., 138; etiol., symp., diag., prog., treat., 139
tuberculosis, 30, 156–165, 169, 170
anat., path., 156; etiol., 158; symp., 159; diag., 164; prog., 169, 170; treat., 170
diff. fr. other forms of phthisis, 166
tumors, 148–153
Pulmonic obstruction, regurgitation, 226
Pulsating empyema, 77
diff. fr. aortic aneurism, 263
Pulsation in the veins on the back of the hands, cause of, 207
Pulse, an indication of action of the heart, 185
normal radial, illus., 208
senile, illus., 210
Punch forceps, 485
Purring tremor, 187
Pus, diff. fr. serum in the pleural sac, 77
Putrid or fetid bronchitis, 91, 102
Pyæmia, diff. fr. glanders, 590
Pyo-pericardium, 217
Pyo-pneumothorax, 88
Pyramidal, pyriform sinuses, 296
Pyrenees, goitre in, 629
Pyriform sinuses, diseases of the, 393

Quain's stethometer, illus., 17
Quality of a murmur, third in importance, 196
of sound, 23, 39, 41; of heart sounds modified by disease, 191
Quinsy, syn. of acute tonsillitis, 362; syn. of phlegmonous tonsillitis, 368

Râles or rhonchi, illus., 48–52
Ramon de la Sota, lupus of the larynx, 452
Rampolla, retro-nasal fibrous tumors, 621
Rankin, D. N., myasis narium, 605
Rapid tracheotomy, 425, 426
Raulin, rhinitis, 532
Recessus pharyngei, illus., 309
Red hepatization, 113, 114; illus., 117
Reduplication of sounds of the heart, 193
Reference Handbook of the Medical Sciences, leptothrix buccalis, 376
Reflected light for laryngoscopy, 275–278
Reflectors, laryngeal, 275–283; perforated concave, 283
Regeneration of atrophied structure, 550
Regions of the chest, illus., 4–8
Regurgitation, aortic, mitral, tricuspid, pulmonic, 225, 228, 230
Renal origin of dropsy, 11
Resection of the ribs in pleurisy, differing views, 78–80; in abscess of the lung, 131; of the larynx described, 481
Resonance, normal vesicular, 25; cracked pot, 28, 31; exaggerated pulmonary, 28; tympanitic, 28, 29; amphoric, vesiculo-tympanitic, 28, 30; normal vocal, 55, 56
Respiration, bronchial, broncho - vesicular, laryngeal and tracheal, 41; exaggerated, feeble, 42; suppressed, interrupted, or cog-wheel, 43; rude, broncho-vesicular or harsh, 44; cavernous, broncho - cavernous, amphoric, 46
Respiratory organs, physiological action of, 38, 39
Retraction of the lung, syn. of consolidation of the lung, 237
Retro-nasal cartilaginous tumors, 625
catarrh, syn. of rhino-pharyngitis, 607
fibro-mucous tumors, illus., 624, 625
anat., path., etiol., symp., diag., 624; prog., treat., 625
diff. fr. fibrous tumors, fr. mucous polypi, fr. malignant growths, 624
fibrous tumors, 620–624
anat., path., etiol., symp., 620; diag., prog., treat., 621
diff. fr. polypi, fr. sarcomata, 621
Retro-pharyngeal abscess, 383–386
anat., path., etiol., 383; symp., diag., 384; prog., treat., 385
diff. fr. croup, fr. œdema of the glottis, fr. foreign bodies, fr. convulsive disorders, 384, 385; fr. phlegmonous laryngitis, 428; fr. abscess of the larynx, 430
Revue d'Hygiène et de Police sanitaire, infection of diphtheria, Grancher, 334
de Laryngologie, d'Otologie et de Rhinologie, rhinitis, Raulin, 532
mensuelle des Maladies de l'Enfance, spasm of the glottis, Lubet-Barbon, 496; foreign bodies in the œsophagus, B. Polikier, 642
Rheumatic pharyngitis diff. fr. diphtheria, 331
Rheumatic sore throat, 316–321; acute, 316, 317; chronic, 318–321
Rheumatism, diff. fr. glanders, 590
nasal affections in, 591
Rhinitis, 522–552; simple acute, 522–526; acute in infants, traumatic, 526; chronic, 527–552; intumescent, 531–540; hypertrophic, 540–547; atrophic, 547–552; in measles, scarlet fever, 591
chronica, syn. of chronic rhinitis, 527
hyperæsthetica, syn. of hay fever, 553
Rhinoliths, 604, 605
symp., 604; diag., prog., treat., 605
diff. fr. atrophic rhinitis, 549; fr. nasal bony tumors, 572; fr. malignant tumors, 573; fr. osteoma, fr. cancer, 605
Rhino-pharyngitis, 607–610
syn., etiol., 607; symp., diag., prog., 608; treat., 609
diff. fr. adenoid growths, fr. syphilis, 608
Rhinoscleroma, 588, 589
etiol., diag., prog., treat., 589
diff. fr. syphilis, fr. epithelioma, fr. keloid, 589
Rhinoscope, a, 272
with uvula holder, illus., 306
Rhinoscopic image, illus., 307
Rhinoscopy, illus., 272, 293–310; anterior, 301, 302; posterior, 302–306
obstacles to posterior, 304, 305
Rhonchi or râles, 48–52
Rhonchial fremitus, bronchial or, 16
Rhythm of sounds, 39, 41; of a murmur, second in importance, 196, 200; of heart sounds modified by disease, 191, 193
of the heart, illus., 182, 183
Ribs, resection of the, 78–80, 131
Riegel, signs of chronic myocarditis, 232
Riehl (see Chiari and Riehl)
Right-angle cutting forceps, 597
Rima glottidis, 298
Robinson, Beverley, asthma due to poison in the blood, 104; feeding in laryngitis, 443; rhino-pharyngitis, 607, 609
Roe, J. O., hay fever related to conditions in nasal passages, 553; nasal vascular tumors, 570; fracture of the nose, 594
Rose cold, syn. of hay fever, 553
Rotch, T. M., pericarditis, 215
Rouge, retro-nasal fibrous tumors, 621
Roux and Yersin, Klebs-Löffler bacillus in mouths of healthy children, 329, carbolic or boric acid in diphtheria, 336
Rubber palate retractor, illus., 306
Rude, broncho-vesicular or harsh respiration, 44
Ruffer, Armand, diphtheritic bacilli, 329
Rupture of the heart, symp., 245

Sacculus laryngis, the, 297
Sajous, Charles E., self-retaining nasal speculum, illus., 301; simple membranous sore throat, 326; cocaine in tubercular sore throat, 352; syphilitic sore throat, 356; chromic acid in trachoma

of vocal cords, 409; hay fever related to conditions in nasal passages, 553; snare, 567; nasal osseous cysts, 570; knife, nasal saws, illus., 599
Salicylic acid, objectionable in pericarditis, 216
Salter, heredity in asthma, 103
Sands, œsophagotome, illus., 636
Sansom, treatment of ulcerative endocarditis, 223; diagnosis of congenital diseases of the heart in children, 246
Sarcomata, 467, 478
diff. fr. nasal mucous polypi, 566
Saws, nasal, 599
Scalds and burns of the pharynx, symp., diag., prog., treat., 392
Scapular region, 4, 7
Scarification of the tonsils, 367, 369
Scarlatina, diff. fr. acute sore throat, 312; fr. diphtheria, 332; fr. acute tonsillitis, 364, 365
Scarlet fever, sore throat of, 323, 324; laryngitis due to, 455; nasal affections in, 591
Schäffer, Max, nasal papillary tumors, 569; differentiation of nasal affections, 586
Schech, anæsthesia of the larynx, 499
Schiffers, myxomata transformed into sarcomata, 566
Schmidt's Jahrbuch, pleurisy, Biegauski, 66; epilepsy following irritation of pleural surfaces, De Cérenville, 78
Schnell, Ferdinand, the aneurismatoscope, 261
Schrötter, head band for reflector in laryngoscopy, illus., 278; tubes, dilators, bougies or sound, 433, 449, 457, 472, 485, 515
Schuller, Max, tracheotomy, 486
Schuster, nasal syphilis, 576
Scirrhus of the lungs, syn. of fibroid phthisis, 167
Scissors for amputating the uvula, illus., 359; nasal, 545; heavy bone, 600
Scrofulous eruptions, diff. fr. glanders, 590
sore throat, 348-350
etiol., 348; symp., diag., 234; prog. treat., 350
diff. fr. lupus of the pharynx, fr. syphilis, fr. tuberculosis, 349; fr. acute tubercular sore throat, 352; fr. syphilitic sore throat, 355
S-curve, illus., 64
Sea voyage for convalescents from subacute pleurisy, 75; for plastic bronchitis, 100; for hay fever, 555
Seashore for hay fever, 455
Seat of a murmur first in importance, 196
of heart sounds modified by disease, 191, 192
Second stage of pneumonia, period of red hepatization, 117; of phthisis, 161, 162; of pericarditis, 213, 214
Sections of head, illus., 302, 541, 579, 584
Sedatives, formulæ, gargles, 647; trochisci or lozenges, 647; vapor inhalations, 650; spray inhalations, 651; dry inhalations, 654; fuming inhalations, 655; insufflations, 656
Seiler, Carl, tube forceps, illus., 495
Self-retaining nasal speculum, 301
Senile pulse, illus., 210
Senn, Nicholas, guaiacol in phthisis, 173; laryngoscopy, 272
Septic endocarditis, syn. of acute endocarditis, 219
Septum forceps, knife, 600
Septum narium, illus., 308
abscesses of the nasal, 603
deflection of the nasal, 594-597
ecchondroma and exostosis of the nasal, 597-601
hæmatoma of the nasal, 602
perforation of the nasal, 601, 602
Sero-fibrinous pleurisy, 61
Serum diff. fr. pus in the pleural sac, 77
Sex modifies form of chest and percussion sounds, 10, 11, 27
Shattuck cites Soltmann on asthma among Hebrews, 103
Shawl-pin removed from the trachea, a, 495
Shoemaker, pilocarpine for erysipelas, 429
Short frænulum obstacle to laryngoscopy, 290
Shortened inspiration, 43
Shurly, E. L., battery, 345; iodine hypodermically for immunity to tubercuiar virus, 172, 442
Sibilant râles, 48, 49
Sibson, treatment of endocarditis, 221
Signs and symptoms differentiated, 9
of inter-thoracic disease, 16
tussive, 59
nervous, 206
Simon, capillary bronchitis in children, 98
Simple acute rhinitis, 522-526
syn., anat., path., etiol., 522; symp., 523; diag., prog., treat., 524
diff. fr. hay fever, 524, 554; fr. inflammation of the antrum or frontal sinuses, fr. measles, 524
acute sore throat diff. fr. acute follicular pharyngitis, 339
cardiac hypertrophy, 234-236
syn., etiol., symp., 234; diag., prog., 235; treat., 236
catarrhal inflammation diff. fr. syphilitic sore throat in infants, 357
chronic rhinitis, 528-530
etiol., symp., 528; diag., prog., 529; treat., 530
diff. fr. hay fever, 529; fr. intumescent rhinitis, 534
membranous sore throat, 324-327
syn., anat., path., 324; etiol., symp., 325; diag., prog., treat., 326
diff. fr. diphtheria, 326, 332; fr. syphilitic sore throat, 355
Sinus, empyema of the frontal, 581; of the sphenoidal, 583, 584
inflammation of the frontal, 584, 585
Sinuses, diseases of the valeculæ and pyriform, 393
of Valsalva, aneurism o the 257, 259
pyramidal, pyriform, laryngo-pharyngeal, 296

Siphon drainage in pleurisy, 79, 82
Skoda, bronchial sound, 45; heart sounds, 190
Small-pox, sore throat of, 321, 322; laryngitis due to, 455; nasal affections in, 591
Smeleder, support of reflector in laryngoscopy, 277
Smith and Warner, pseudo-diphtheria, 329
Smoker's patches diff. fr. leucoplakia buccalis, 361
Snare for excisions in the throat, 386, 567, 570
applicator, post-nasal, 623
forceps, 473
Sodium sulpho-carbolate in endocarditis, 223
Solid mediastinal tumors, 267, 268
symp., 267; diag., prog., treat., 268
diff. fr. thoracic aneurism, 262
Soltmann, asthma among Hebrews, 103
Sonorous râles, 48
Sore throat, acute, 311–314; erysipetalous, 314–316; rheumatic, 316–321; acute rheumatic, 316, 317; chronic rheumatic, 318–321; simple membranous, 324–327; scrofulous, 348–350; acute tubercular, 350–353; syphilitic, 353–357
throat of measles, symp., diag., prog., treat., 322
throat of scarlet fever, 323, 324
anat., path., symp., diag., 323; prog., treat., 324
throat of small-pox, 321, 322
anat., path., diag., prog., treat., 322
Sound in moving fluid transmitted in the direction of motion, 197
South America, myasis narium, 605
South Carolina for phthisis, 175
Spain for phthisis, 175
Sparteine in chronic endocarditis, 229
Spasm of the adductors diff. fr. paralysis of the abductors, 513
of the glottis, 496, 497
syn., symp., diag., 496; prog., treat., 497
diff. fr. acute laryngitis, 395, 396; fr. phlegmonous laryngitis, 427; fr. true croup, 414, 497
of the larynx in adults, 497, 498
etiol., 497; symp., diag., prog., treat., 498
diff. fr. asthma, 105
of the œsophagus, 637, 638
syn., etiol., symp., 637; diag., prog., treat., 638
diff. fr. stricture of the œsophagus, 635; fr. paralysis, 639
of the pharynx, etiol., symp., diag., prog., treat., 390
diff. fr. stricture of the œsophagus, fr. paralysis, fr. paralysis of the pharynx or the œsophagus, 390
of the vocal cords, 502, 503
anat., path., 502; symp., treat., 503
Spasmodic asthma diff. fr. hay fever, 554
stricture of the œsophagus, syn. of spasm of the œsophagus, 637
croup, syn. of spasm of the glottis, 496
Spasmus glottidis, syn. of spasm of the glottis, 496
Spatula, nasal, 600
Sphenoidal sinuses, empyema of the, 583, 584
Sphygmograph, the, illus., 208–211, 260
Spirometer, illus., 18
Spleen, variable in size, 27; enlargement of, diff. fr. pleurisy, 70
Spray inhalations, formulæ, 651–653
Sprays, powders, pigments, of successive value for chronic laryngitis, 405
Spud, nasal, 599
Staining tubercular bacilli, 164, 166
Staphylococci in pleurisy, 61
Starvation treatment of aortic aneurism, 266
Stenosis of the aorta, syn. of coarctation of the aorta, 266
of heart valves produced, 224, 228
of the larynx, chronic, 456–459
of the trachea, diag., prog., treat., 460
Sterilized air in pneumothorax, 88
Sternal region, 4, 6, 7
Sternberg, diplococcus pneumoniæ, 115
Stethogoniometer, 18
Stethometer, 17
Stethoscopes, 34–37; disadvantages of, 34
Stimulant and caustic pigments, 656 (see astringents and stimulants, antiseptics and stimulants)
Stimulants, formulæ, gargles, 647; trochisci or lozenges, 643; vapor inhalations, 650, 651; dry inhalations, 654
Stimulating injections for pleurisy, 81
Stirling, inhalation of lime water in plastic bronchitis, 100
Stoerk, écraseur, guillotines, forceps, blades, illus., 473
Stokes, pseudo-apoplexy and fatty heart, 244
Stowell, C. H., sections of head, illus., 302, 541, 579, 584
Streptococci in pleurisy, 61
Streptococcus erysipelatosus, 315
Strickler, W. M., resection of ribs for pleurisy, 78
Stricture of the œsophagus, 634, 637
anat., path., etiol., symp., 634; diag., prog., 635; treat., 636
diff. fr. spasm of the larynx, 390; fr. tubercular laryngitis, fr. tumors in the pharynx, larynx, œsophagus, fr. spasm, fr. paralysis, fr. foreign bodies, fr. spasm, 635
Strong, A. B., resection of ribs for pleurisy, 78; drainage tubes, illus., 79
Strophanthus in exopthalmic goitre, 632
Struma, syn. of goitre, 629
Subacute bronchitis (see acute bronchitis)
laryngitis, 397, 398
prog., treat., 397
pericarditis, 212
pleurisy, 12, 72–75
anat., path., etiol., 72; symp., diag., prog., treat., 73
Subclavian murmurs, 206
Subcrepitant râles, 48–50
Subcutaneous emphysema shown, 11
Subglottic hypertrophy, 401

Submucous infiltration of the sides of the vomer, illus., diag., treat., 547
laryngitis, syn. of phlegmonous laryngitis, 427
laryngitis, syn. of œdema of the larynx, 430
Succussion, 9, 20, 86
Suffocative laryngismus, syn. of spasm of the glottis, 496
stage of croup, 412
Superficial ulceration in syphilitic sore throat, 353; of vocal cords, of epiglottis, illus., 395
Superior costal breathing, 11
meatus, illus., 309
sternal region, 4, 6
turbinated bodies, illus., 309
Suppressed respiration, 43
Suppuration of the anterior ethmoid cells diff. fr. empyema of the antrum, 581
of the antrum, diff. fr. atrophic rhinitis, 549; fr. chronic suppurative ethmoiditis, 586
Suppurative ethemoidhtis, chronic, 585-587
tonsillitis, syn. of phlegmonous tonsillitis, 368
Supra-arytenoid cartilages, 296
Supra-clavicular region, 4
Supra-glottic dropsy, syn. of œdema of the larynx, 430
Supra-scapular region, 4, 7
Supra-sternal region, 4, 6
Supra-thyroid laryngotomy, 475
Swallowing the tongue, 392, 393
treat., 393
Swiss mountains for phthisis, 175; goitre in, 629
Symonds, Charters J., gum-elastic tube to keep stricture of œsophagus pervious, 636
Symptoms and signs differentiated, 9
Syphilis of the nose, 574-577
anat., path., etiol., 574; diag., prog., treat., 575
diff. fr. atrophic rhinitis, 549, 575; fr. simple catarrhal rhinitis, fr. lupus, 575; fr. empyema of the antrum, 581; fr. lupus of the nares, 588; fr. rhinoscleroma, 589; fr. glanders, 590; fr. rhino-pharyngitis, 608
of the trachea, illus., 487, 488
anat., path., etiol., symp., 487; diag., prog., treat., 488
Syphilitic condylomata of the larynx diff. fr. benign growths, 468
disease of the heart, 245
disease of the lungs, 151, 152
symp., diag., 151; prog., treat., 152
laryngitis, illus., 443, 450, 456
etiol., symp., 444; diag., 446; prog., treat., 448
diff. fr. chronic laryngitis, 403; fr. tubercular laryngitis, 439, 440; fr. tubercular laryngitis, fr. tumors, 446-448; fr. lupus, 453; fr. benign tumors of the larynx, 468; fr. cancer, 479
Syphilitic laryngitis in infants, 449, 450
diag., 449; prog., treat., 450
patches diff. fr. leukoplakia buccalis, 362
sore throat, illus., 353-357
anat., path., 353; etiol., symp., diag., 354; prog., treat., 355
diff. fr. chronic rheumatic sore throat, 320; fr. chronic follicular pharyngitis, 344; fr. scrofulous sore throat, 349; fr. catarrhal sore throat, fr. scrofulous sore throat, fr. tubercular sore throat, 354, 355; fr. acute tonsillitis, 366; fr. cancer of the pharynx, 387
sore throat in infants, 356, 357
anat., path., etiol., diag., prog., treat., 357
diff. fr. simple catarrhal inflammation, 357
ulceration of the tonsil diff. fr. tubercular ulceration of the tonsils, 378; fr. cancer, 380, 381
Syringe, nasal, 550; hypodermic, 568; post-nasal, 609
Systole of the heart, 180; auricular, illus., 201; ventricular, illus., 202
Systolic murmur, 201, 202
souffle, 244
venous pulsation, cause of, 207

Tachycardia, 249
prog., treat., 249
Taenia echinoc, coccus, cause of hydatid cysts of the lungs, 148
Tait's Cliniques de Laryngotomie, thyrotomy, Krishaber, 475
Tampon, for the nose, wool, 552; surgeons' lint, 561, 562; lint or gauze, 600; styptic gauze, 624
Teeth, empyema of the antrum from diseased, 579
Tennessee mountains for phthisis, 175
Texas for phthisis, western, 175
Thickening of turbinated bodies diff. fr. mucous polypi, 565
Third stage of pneumonia, period of gray hepatization, 117; of phthisis, 161-164; of pericarditis, 213, 215
Thompson, R. E., percussion sounds, 28, 30; pulmonary emphysema, 110
Thoracic aneurism, aortic or, 256-266
arteries, diseases of the, 254-268
Three stages of acute pleurisy, 81; of pneumonia, 117; of phthisis, 161; of pericarditis, 213; of croup, 412
Throat, the, 271-310
acute rheumatic sore, 316-317
acute sore, 311-314
acute tubercular sore, 350-353
chronic rheumatic sore, 318-321
consumption, syn. of tubercular laryngitis, 434
deafness, 610-613
etiol., symp., 610; diag., prog., 611; treat., 612
diseases of the, 271-515

Throat, erysipelatous sore, 314-316
gouty affections of the, 319
mirrors for laryngoscopy, illus., 273
of measles, sore, 322
of scarlet fever, sore, 323, 324
of small-pox, sore, 321, 322
rheumatic sore, 316-321
scrofulous sore, 348-350
simple membranous sore, 324-327
syphilitic sore, 353-357
Thrombosis and embolism, pulmonary, 138, 139
Thymus vulgaris, unsatisfactory with pertussis, 155
Thyro-arytenoid muscles, paralysis of the, 507, 508
Thyro-epiglottic and ary-epiglottic muscles, paralysis of, 505
Thyroid gland, diseases of the, 629-632
Thyrotomy described, 474-476, 483
Tinkling, metallic, 20, 54, 87, 88
Tobacco smoking a cause of leukoplakia buccalis, 360
sore throat, diff. fr. chronic rheumatic sore throat, 320
Tobold, illuminator, 280; laryngeal knives, illus., 474
Tongue, arching of the, 290; swallowing the, 392; enlarged glands and veins at base of the, 319 (see paræsthesia of the pharynx); depressors, illus., 271, 464
Tonsil forceps, 373
Tonsilla pharyngea, 310
Tonsillitis, acute, 362-367; phlegmonous, 368, 369; chronic (see hypertrophy of the tonsil)
Tonsillitome, the, 372, 373
Tonsillotomy, 373, 374
Tonsils, concretions in the, 375
cancer of the, 380, 381
hypertrophy of the, 369-375
to generative organs, relation of the, 375
hypertrophy of the pharyngeal, 613-620
Luschka's, 613
mycosis of the, 376, 377
obstacle to laryngoscopy, enlarged, 290
removal of the, 371-375
tubercular ulceration of the, 378-380
Tornwaldt, nasal tuberculosis, 578; rhinopharyngitis, 607
Trachea, examination of the, 300
involution of the, 485, 486
stenosis of the, 460
syphilis of the, 487, 488
Tracheal cartilages, illus., 299
respiration, laryngeal and, 41
tumors, illus., 483, 484
etiol., symp., diag., prog., treat., 484
Tracheitis, 460-462
anat., path., etiol., symp., 460; diag., prog., treat., 461
diff. fr. laryngitis, fr. bronchitis, 461
Tracheocele, 486, 487
syn., anat., path., etiol., symp., 486; diag., prog., treat., 487
Tracheophony, 54
Tracheotomy described, 421-426
in aneurism of the aorta, 266
in various throat diseases, 338, 397, 432, 434, 442, 448, 450, 454, 455, 457, 459, 470, 472, 474, 481, 484, 486, 488, 495
rapid, 425, 426
vegetations after, 485
Trachoma of the vocal cords, illus., 408, 409
syn., anat., path., etiol., symp., diag., prog., treat., 408
Transillumination of the antrum, 580; electric lamp for, 581
Traube, pulmonary percussion, 66
Traumatic laryngitis, symp., diag., prog., treat., 398
rhinitis, 526, 527
symp., treat., 527
Traveller's nasal douche, illus., 551
Trephines, nasal, 546
Triangle of dulness, illus., 64
Tricuspid area, illus., 198, 199
obstruction, 226, 228
regurgitation, 225, 228, 230
stenosis, 225, 228
valves, 7, 178
Trocar, flat, 79
Trochisci or lozenges, formulæ, 647-649
Trousseau, percussion, 32; laryngoscopy, 272; tracheal forceps, 495
True croup, syn. of membranous croup, 411
Tube for antrum, drainage, 583
forceps, 472
to keep stricture of œsophagus pervious, 636
Tubes for chronic pleurisy, drainage, 79-83
for intubation, 418
Tubercle bacilli, illus., 157; staining, 164; in lupus of the larynx, 451
bacillus, Koch, 159
Tubercles, mucous, 353
Tubercular laryngitis, illus., 434-443
syn., 434; anat., path., 435; etiol., symp., 436; diag., 437; prog., treat., 431
diff. fr. chronic laryngitis, 403; fr. anæmia, fr. œdema of the larynx, fr. catarrhal laryngitis, fr. syphilis, 437-440; fr. syphilitic laryngitis, 447; fr. lupus, 452; fr. benign tumors, 468; fr. cancer, 479
sore throat (see acute tubercular sore throat)
ulceration of the tonsils, 378-380
anat., path., symp., diag., 378; prog., 379; treat., 380
diff. fr. syphilis, fr. cancer, 379
Tuberculin of doubtful value in phthisis, 173; disastrous results in lupus of the larynx, 453; in tuberculosis of the nares, 579; curative in lupus of the nares, 588; inactive in rhinoscleroma, 589
Tuberculosis (see acute tubercular sore throat)
acute miliary, 165-167
of the nares, 578, 579
anat., path., etiol., symp., diag., prog., treat., 578

Tuberculosis of the nares diff. fr. lupus of the nares, 588
pulmonary, 156–165
Tufnell, treatment of thoracic aneurism, 266
Tumors, see also aneurism
nasal: fibrous, 569; papillary, 569, 570; vascular, 570; cartilaginous, 571; bony, 571, 572; malignant, 572, 573
of the heart, diag., prog., treat., 246
of the larynx: benign, 465–476; cartilaginous, 468; malignant, 476–483
of the naso-pharynx: malignant, 625; cystic, 626
of the pharynx, illus., treat., 386
pulmonary, 148–153
retro-nasal. fibrous, 620–624; fibro-mucous, 624; cartilaginous, 625
solid mediastinal, 193, 267, 268
tracheal, 483, 484
Turbinated bodies, 308; hypertrophy of, 541, 542
Türck, tongue depressor, illus., 271; laryngoscopy, 272; attempt to magnify laryngeal image, 282; syphilitic laryngitis, 446
Turgescence, venous, 206, 262, 267
Tussive signs, 59
Tympanitic resonance, 26, 28, 29, 30, 66
Typhoid fever, nasal affections in, 591; diff. fr. pneumonia, 121; fr. glanders, 590
pneumonia, 128

Ulceration of the pharynx, 357
of the tonsils: tubercular, 378–380; syphilitic, 379, 381
Ulcerative endocarditis, 222, 223
etiol., symp., diag., prog., 222, treat., 223
Unilateral paralysis of the lateral crico-arytenoid muscles, illus., 510, 511
etiol., symp., diag., 510; treat., 511
paralysis of the posterior crico-arytenoid muscles, illus., symp., diag., prog., treat., 514
Unilocular pleurisy diff. fr. other forms, 83
United States, goitre in, 629
Utah for phthisis, 175
Uvula, abscission of the, 359
acute inflammation and œdema of the, 358
and palate retractor, self-retaining, 306
diseases of the, 358–360
elongated, 289, 305, 343
chronic inflammation and elongation of the, 358, 359
malformations and new growths of the, 359, 360
malignant growths in the, 360
Uvulatome scissors, illus., 359

Valeculæ, the, illus., 295
and pyriform sinuses, diseases of the, 393
Valsalva, sinuses of, 257, 259; treatment of aortic aneurism, 266
Valves of the heart, 7, 178; position of the, 179
Valvular disease of the heart (see chronic endocarditis)
murmurs, 203
Vapor inhalations, formulæ, 649–651
Vaporizer, 612
Vapors from lime water in membranous croup, 416
Varicose veins at base of tongue, 389
diff. fr. chronic rheumatic sore throat, 319
Vascular tumors, angiomata or, 467
tumors, nasal, 570
Vault of the pharynx and posterior nasal cavities, 307–310
Vegetations, post-tracheotomy, 485
Veins at base of tongue, varicose, 319, 389
Velum palati attacked in syphilitic sore throat, 353, 354
Venous murmur or hum, 207
pulsation, presystolic, systolic, 206, 207
signs, 206–208
Ventilation with diphtheria, mode of, 334
Ventricle of Morgagni, eversion of the, 483
Ventricles of the heart, right and left, 178
of the larynx, the, 297
Ventricular bands, illus., 297
murmurs, 204
systole, 182; illus., 202
Verneuil, œsophageal dilator, 638
Vertigo, laryngeal, 504
Vesicular emphysema, 107
murmur, the standard of comparison, 39, 40
resonance, normal, 25
Vesiculo-tympanitic resonance, 30
Vierteljahresschrift für Dermatologie und Syphilis, lupus of the larynx, Chiari and Riehl, 451; nasal syphilis and lupus of the larynx, Shuster, 576
Virchow, pulmonary emphysema, 107; malignant endocarditis, 219
Virchow's Archiv, nasal papillary tumors, Hopmann, 569
Virginia mountains for phthisis, 175
Vocal cords, illus., 297
cords, atrophy of the, 515
cords, paralysis affecting the, 505–514
cords, spasm of the, 502, 503
cords, trachoma of the, 408, 409
cords, tumors of the, 465–468
cords, ulcers of the, 395
fremitus, normal, 15, 16
resonance, normal, diminished, 55; increased or exaggerated, 56; whispering, 58
sounds, 54–59
Voltolini, attempt to magnify laryngeal image, 282; staff for lifting the epiglottis, 291; friction in laryngeal tumors, 472; transillumination of the antrum, 580
Vomer or septum, illus., 307; submucous infiltration of the sides of the, illus., 547
Von Stoffella, pericarditis, 214
Vulsella forceps, 367

Wagner, Clinton, pneumonia contagious, 116; retro-nasal cystic tumors, 626
Walsham, deflection of the nasal septum, 596

Warden, laryngoscopy, 272
Warner (see Smith and Warner)
Wash bottle, 586
Waxham, gag, illus., 419
Weber, cause of asthma, 103
Weber-Liel, throat deafness, 610
Weichselbaum, diplococcus pneumoniæ, 115
Weill, carbon dioxide in asthma, 106
laryngeal illumination, 280
Wells, pneumonia contagious, 116
Wertheim, attempt to magnify laryngeal image, 282
Whisper, normal bronchial, exaggerated, cavernous, amphoric, 58
Whispering bronchophony, pectoriloquy, vocal resonance, 58
Whistler, cutting dilator, illus., 458
White Mountains for hay fever, 555
Whittaker, James T., transmission of bacilli to fœtus, 158
Whooping cough, pertussis or, 153–155
Wiener medizinische Presse, cause of putrid bronchitis, Josef Lumniczer, 91; pericarditis, E. Pins, 214
Williams, C. J. D., rhinitis, 525
Winter cough, 91
Wintrich, tympanitic resonance, 66; pleurisy, 83; cause of asthma, 103
Woakes, Edward, mucous polypi, 564; throat deafness, 610
Wolff, pneumonia contagious, 115
Wool tampons, 552
Wright, C. H., burr for nasal surgery, 598
Wròblewski, adenoid growths in deaf mutes, 614
Wyoming for phthisis, 175

Yellow hepatization, 113, 114
Yeo, J. Burney, pleurisy of the apex and low-necked dresses, 82
Yersin (see Roux and Yersin)

Zeitschrift der Bakterienkunde, contagious pneumonia, Wolff, 115
für klinische Medicin, signs of chronic myocarditis, Riegel, 232
Ziehl, solution for staining bacilli, 164
Ziemssen, chorea laryngis, 501
Ziemssen's Cyclopedia of Medicine, carbolic acid in diphtheria, Oertel, 336; glanders eleven years, Bollinger, 590
Zimmermann, siphon drainage in pleurisy, 79
Zuckerkandl, nasal papillary tumors, 569; deflector of the nasal septum, 594
Zwillinger, H., nasal osseous cysts, 570

COLUMBIA UNIVERSITY

This book is due on the date indicated below, or at the expiration of a definite period after the date of borrowing, as provided by the rules of the Library or by special arrangement with the Librarian in charge.

DATE BORROWED	DATE DUE	DATE BORROWED	DATE DUE
C28(638)M50			

RC941 In4
1894
Ingals