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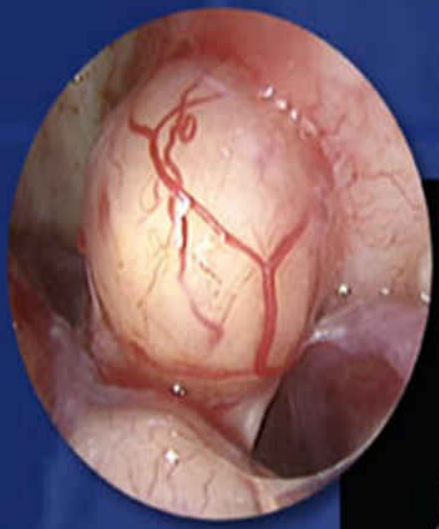

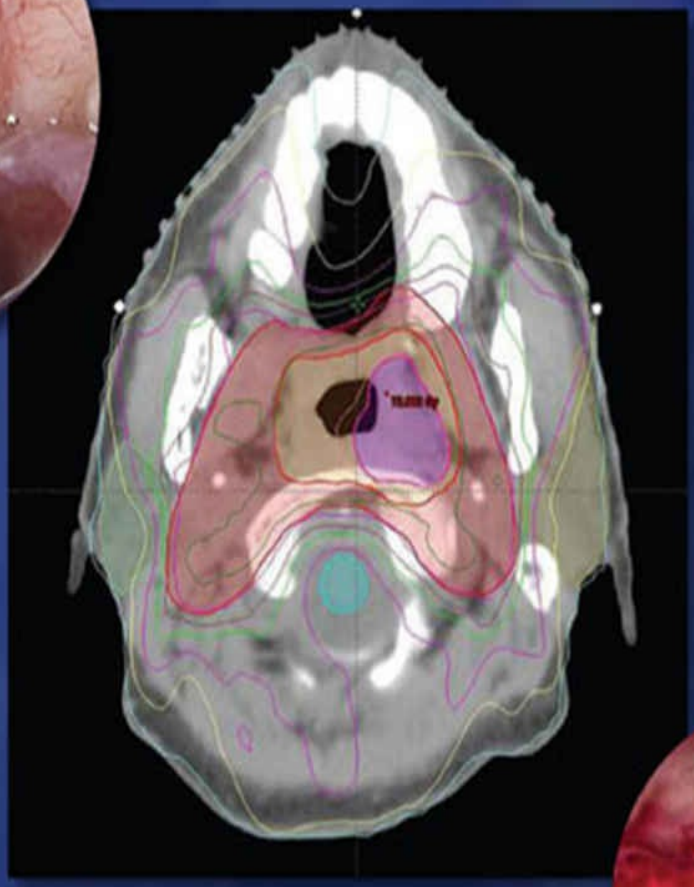

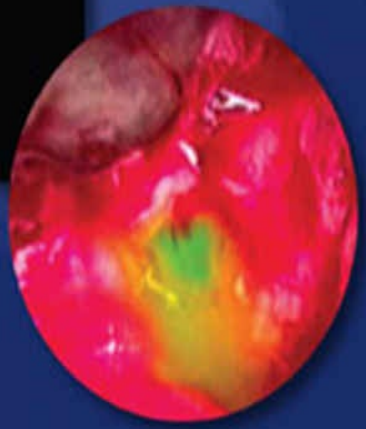

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OTORHINOLARYNGOLOGY 18

HEAD AND NECK SURGERY

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9. Diagnostic Audiology, Hearing Instruments, and Aural Habilitation

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114. Diseases of the Thyroid and Parathyroid Glands

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86. Disorders of Speech and Language

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27. Autoimmune Inner Ear Disease and Other Autoimmune Diseases with Inner-Ear Involvement

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112. Neoplasms of the Larynx and Laryngopharynx

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79. Speech and Resonance Disorders in Children

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4. Physiology of the Auditory and Vestibular Systems

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88. Laryngopharyngeal Reflux, Infections and Manifestations of Systemic Diseases

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110. Neoplasms of the Oral Cavity

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72. Pediatric Sleep Disordered Breathing

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36. Stereotactic Radiosurgery and Radiotherapy

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66. Otoplasty

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15. Eustachian Tube Dysfunction

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58. Rejuvenation of the Upper and Mid-face
60. Scar Revision and Skin Resurfacing

Sharon G. Kujawa, PhD
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22. Noise-Induced Hearing Loss

John F. Kveton, MD
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18. Intratemporal and Intracranial Complications of Acute and Chronic Otitis Media

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90. Airway Control and Laryngotracheal Stenosis in Adults

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29. Presbyacusis and Presbyastasis

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104. Targeted Therapuetic Approaches to Head and Neck Cancer

Andrew P. Lane, MD
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Neck Surgery Johns Hopkins School of Medicine Baltimore, Maryland
53. Revision Paranasal Sinus Surgery and Surgery of the Frontal Sinus

Charles J. Limb, MD
Francis A. Sooy, MD Professor and Chief Otology/Neurotology
and Skull Base Surgery Director, Douglas Grant Cochlear Implant Center University of California San Francisco School of Medicine San Francisco, California
8. Central Auditory Processing and Functional Neuroimaging

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Riverside Medical Group Newport News, Virginia
97. Esophagology

Yinda Liu, AuD
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8. Central Auditory Processing and Functional Neuroimaging

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Milwaukee, Wisconsin
48. Acute Rhinosinusitis and Its Complications
102. Navigational Systems, Surgical Simulators and Robotic Surgery

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14. Diseases of the External Ear

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91. Neurogenic Disorders of the Larynx

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11. Imaging of the Temporal Bone

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81. Congenital Head and Neck Masses

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102. Navigational Systems, Surgical Simulators and Robotic Surgery

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16. Acute Otitis Media and Middle-Ear Effusions

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16. Acute Otitis Media and Middle-Ear Effusions

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39. Cellular Biology of the Immune System

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108. Neoplasms of the Anterior Skull Base

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84. Development, Anatomy, and Physiology of the Larynx

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76. Acquired Anomalies of the Larynx and Trachea

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44. Allergy Evaluation

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108. Neoplasms of the Anterior Skull Base

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114. Diseases of the Thyroid and Parathyroid Glands

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29. Presbyacusis and Presbyastasis

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Sciences University of Washington Seattle, Washington
94. Imaging of the Larynx, Trachea, and Esophagus

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107. Nutrition of the Patient with Head and Neck Cancer

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27. Autoimmune Inner Ear Disease and Other Autoimmune Diseases with Inner-Ear Involvement

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29. Presbyacusis and Presbyastasis

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28. Menière Disease, Vestibular Neuritis, Benign Paroxysmal Positional Vertigo, Superior Semicircular Canal Dehiscence, and Vestibular Migraine

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93. Muscle Misuse Disorders of the Larynx

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106. Immunotherapy for Squamous Cell Carcinoma of the Head and Neck

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103. Imaging of the Oral Cavity, Pharynx, Salivary Glands and Neck

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59. Rejuvenation of the Lower Face and Neck

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Allergic Rhinitis
Allergy Evaluation

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64. Regional Flaps and Free Tissue Transfer

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12. Pathologic Correlates in Otology and Neurotology

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17. Chronic Otitis Media and Cholesteatoma

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33. Cochlear Implant Coding Strategies and Device Programming

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7. Cochlear Biophysics

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102. Navigational Systems, Surgical Simulators and Robotic Surgery

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21. Trauma to the Middle Ear, Inner Ear and Temporal Bone

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68. Congenital Anomalies of the External, Middle and Inner Ear

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56. Functional Rhinoplasty

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47. Acute and Chronic Nasal Disorders

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84. Development, Anatomy, and Physiology of the Larynx

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89. Trauma to the Larynx

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105. Chemoradiation for Head and Neck Cancer

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18. Intratemporal and Intracranial Complications of Acute and Chronic Otitis Media

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82. Vascular Tumors and Malformations of the Head and Neck

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57. Upper and Lower Blepharoplasty

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100. Mechanisms of Immune Evasion of Head and Neck Cancer

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98. Sleep Medicine and Surgery

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4. Physiology of the Auditory and Vestibular Systems

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64. Regional Flaps and Free Tissue Transfer

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15. Eustachian Tube Dysfunction

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42. Etiology of Infectious Diseases of the Upper Respiratory Tract

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97. Esophagology

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37. Anatomy and Physiology of the Nose and Paranasal Sinuses

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79. Speech and Resonance Disorders in Children

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58. Rejuvenation of the Upper and Mid-face

Alicia M. Quesnel, MD
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20. Otosclerosis

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75. Congenital Anomalies of the Larynx and Trachea

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93. Muscle Misuse Disorders of the Larynx

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24. Idiopathic Sudden Sensorineural Hearing Loss

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81. Congenital Head and Neck Masses

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78. Aerodigestive Tract Foreign Bodies and Caustic Ingestions

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34. Facial Paralysis
58. Rejuvenation of the Upper and Mid-face

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5. Gene Therapy and Inner-Ear Drug Delivery

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54. Endoscopic Surgery of the Skull Base, Orbits and Benign Sinonasal Neoplasms

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95. Laryngoscopy

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33. Cochlear Implant Coding Strategies and Device Programming

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105. Chemoradiation for Head and Neck Cancer

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49. Chronic Rhinosinusitis and Polyposis

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36. Stereotactic Radiosurgery and Radiotherapy

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70. Congenital Anomalies of the Mandible and Maxilla

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69. Anatomy and Physiology of the Oral Cavity, Oropharynx, Salivary Glands and Neck

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93. Muscle Misuse Disorders of the Larynx

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96. Bronchology

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97. Esophagology

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21. Trauma to the Middle Ear, Inner Ear and Temporal Bone

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5. Gene Therapy and Inner-Ear Drug Delivery

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52. Primary Paranasal Sinus Surgery

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92. Laryngeal Paralysis

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69. Anatomy and Physiology of the Oral Cavity, Oropharynx, Salivary Glands and Neck

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113. Diseases of the Salivary Glands

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82. Vascular Tumors and Malformations of the Head and Neck

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3. Molecular Biology of Hearing and Balance

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11. Imaging of the Temporal Bone

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99. Molecular Biology of Squamous Cell Carcinoma

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87. Benign Laryngeal Lesions

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34. Facial Paralysis
36. Stereotactic Radiosurgery and Radiotherapy

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64. Regional Flaps and Free Tissue Transfer

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109. Neoplasms of the Nasophaynx

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102. Navigational Systems, Surgical Simulators and Robotic Surgery

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21. Trauma to the Middle Ear, Inner Ear and Temporal Bone

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10. Evaluation of the Vestibular

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73. Drooling, Dysphagia and Aspiration

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71. Infectious and Inflammatory Disease of the Oral Cavity, Oropharynx, and Nasopharynx

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98. Sleep Medicine and Surgery

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85. Assessment of Vocal Function

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113. Diseases of the Salivary Glands

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49. Chronic Rhinosinusitis and Polyposis

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105. Chemoradiation for Head and Neck Cancer

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71. Infectious and Inflammatory Disease of the Oral Cavity, Oropharynx, and Nasopharynx

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84. Development, Anatomy, and Physiology of the Larynx

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41. Imaging of the Nasal Cavities, Paranasal Sinuses, Nasopharynx, Orbits, Infratemporal Fossa, Pterygomaxillary Fissure and Base of Skull

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13. Outcomes Research, Clinical Trials and Clinical Research
112. Neoplasms of the Larynx and Laryngopharynx

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80. Voice Disorders in Children

FOREWORD

It is my pleasure to write the foreword for this 18th edition of *Ballenger's Otorhinolaryngology Head and Neck Surgery*, edited by my colleagues and friends P. Ashley Wackym and James B. Snow, Jr. I first read an early edition of this book as a resident beginning to accumulate knowledge in the field to which I would dedicate my professional career.

In this 18th edition, the editors have assembled an all-star cast of authors who are international experts in their respective fields. The book is a current and comprehensive compendium of current knowledge in the field of otorhinolaryngology head and neck surgery. The authors have abridged the current literature to present to the reader a concise, yet powerful view of the important topics of the field. From basic anatomy, physiology and clinical standards, to emerging fields with an emphasis on the molecular biology of disease and restorative medicine, this book is a wonderful teaching tool for the resident, medical student, researcher or ancillary personnel with interest in the ever-changing field of otorhinolaryngology head and neck surgery.

The editors, authors and illustrators are to be commended for the tremendous effort necessary to produce a five-star book. Trainees will find this study of great value and practitioners will be grateful for an up-to-date reference.

Congratulations to Ashley and Jim for the development and execution of this marvelous and comprehensive book.

D. Bradley Welling, MD, PhD
March 2016

PREFACE

It has been a great pleasure to work with James B. Snow, Jr. in the development and execution of the centennial and this 18th edition of *Ballenger's Otorhinolaryngology Head and Neck Surgery*. Our vision for this edition was two-fold: 1) to highlight the pivotal role molecular medicine continues to play in understanding the pathogenesis of disease and patient diagnosis and therapy and 2) to include the technological advances which continue to shape our specialty. All chapters have been updated and 15 new chapters added. Sectional editors and senior authors were selected based on their contributions of new knowledge and highly-respected research and intellectual leadership in their specialties. These new contributions have greatly enhanced the book. Our editorial aim was to produce a comprehensive compendium with an absolute minimum of redundancy which includes all new and important information in all specialties relating to disorders of hearing, balance, smell, taste, voice, speech and language that are the principal responsibilities of the otorhinolaryngologist–head and neck surgeon of the 21st century.

When Jim Snow invited me to collaborate with him to develop the centennial edition and then assume the primary editorial leadership of this 18th edition, the timing was perfect both professionally and personally which allowed me to accept. He was the perfect mentor to guide me through this endeavor and for this I am forever grateful. It is a monumental undertaking to synthesize into one volume all the information needed for current and future students, young otorhinolaryngologists–head and surgeons, and experienced surgeons. It has been an honor and privilege to work with Jim. He furthered my skills honed from other mentors, Paul Ward, Brian McCabe, Bruce Gantz, and Vicente Honrubia. With his superb intellect, thoughtful organization and introspection, he served as an honest critic, enthusiastic supporter and even father figure. Jim always treated me as an equal, and we established a comfortable

relationship in which we valued each other's perspective and opinion and always reached agreement because of our shared vision and purpose to enhance the quality of the book. This common goal facilitated our work and ultimately produced an exceptional reference text, and I am proud to have played a role in its development.

My hope is that all those using the book or online version will benefit immensely from the organization and content, and our patients will ultimately reap the full benefits of the knowledge and expertise contained within.

P. Ashley Wackym, MD
March 2016

INTRODUCTION

As Ballenger's book enters its second century of publication, we have endeavored to make this 18th Edition a comprehensive guide to current clinical practice and provide the basic knowledge upon which that practice is based. The majority of the sectional editors are new, individuals who have earned a standing in their area of specialization to lend unmatched authority to their sections. Senior authors of each chapter were selected because of their contributions to new knowledge in their specialty areas. P. Ashley Wackym is clearly one of the leading intellects of our field, and it is his leadership and organizational abilities that ultimately brought this publication into a new era. It is a remarkable integration, really synthesis, of state-of-the-art basic science and clinical practice. We hope that we have succeeded in making it reader-friendly with a minimum of redundancy.

Just as the role of libraries is expanding from being repositories of printed matter to helping educate patrons to gain digital access to information, so too has this book undergone such change. Our text should not solely be a repository of information at the time of writing, but also serve to guide users to easy and rapid access to all the most recent findings. In this edition, the editors and authors have added digital information and website links for many sources in the various specialties of our field. Each contributor has strived to inform the reader how to efficiently access new and better organized information. For example, in the chapter on hereditary hearing loss, access to the Online Mendelian Inheritance of Man is presented along with how to make maximum use of it. This source provides a six digit reference number for each phenotype so that new information about the molecular genetics and clinical findings related to the phenotype is constantly being updated from around the world. As new genes are found, the descriptions of the phenotypes are expanded and the concepts about them are modified.

The development of massively parallel sequencing provides the capability equivalent to sequencing the whole exome (genome). With it, a patient's exome can be obtained. An alternative to sequencing the whole exome, for example, is to sequence the deafness exome, the exons comprising all of the known deafness genes. This book will enable you to learn when to do one or the other of these possibilities. Which should be done at the current state-of-the-art will change with progress and as we learn more about the risks and benefits of each. Be ready for it.

The ever-increasing role of molecular biology in genetics, physiology and pharmacology is emphasized throughout the book. It is hoped that this information will anticipate the realization of many advances between now and the next edition. Through progress in the molecular genetics of the senses of hearing, balance, smell and taste and of the production of voice, speech and language, the realization of the individualization of the practice of otorhinolaryngology head and neck surgery is at hand.

Above all, we hope that this 18th edition will benefit your patients and that you will enjoy reading it as much as we have enjoyed writing and editing it.

James B. Snow, Jr., MD
March 2016

DEDICATION

This work is dedicated to my wife, Jeremy, and my son, Ashton, from whom I have stolen innumerable hours in pursuit of the highest standard of academic otorhinolaryngology head and neck surgery; and to my patients, students, residents, faculty members, collaborators and mentors who have all given me purpose and immeasurable satisfaction.

P. Ashley Wackym, MD

This first edition in Ballenger's second century is dedicated to Anna Jane Mercer, marvelous intellect and friend, avid reader, teacher of the youngest to the oldest, builder of the future for family and associates, and my dearest companion.

James B. Snow, Jr., MD

OTOLOGY AND NEUROTOLOGY

1

Anatomy of the Auditory and Vestibular Systems

Richard R. Gacek, MD

The temporal bone (TB) is a complex portion of the skull base that contains the labyrinth with its nerve supply (cranial nerve VIII) but also other cranial nerves such as the facial, trigeminal, vagus, glossopharyngeal, spinal accessory, and hypoglossal nerves. A thorough knowledge of the gross and microscopic anatomy[1,2] of the TB and the physiology of the labyrinthine sense organs is essential for the specialist who strives for accuracy in diagnosis and precision in surgery of the TB. This knowledge is gained first from dissection of cadaveric whole TB specimens but is greatly enhanced by study of prepared histologic sections from normal and pathologic TB.

OSTEOLOGY

Four major components of the TB contribute to the skull base: the squamous, mastoid, tympanic, and petrous.

The *squamous* portion of the TB provides attachment for the temporalis muscle, which is bounded inferiorly by the temporal line (Figure 1-1). The temporal line provides an external landmark for the floor of the middle cranial fossa. The zygomatic process projects forward from the lower portion of this bone, and together they form the anterior border of the mandibular fossa, which receives the condyle of the mandible.

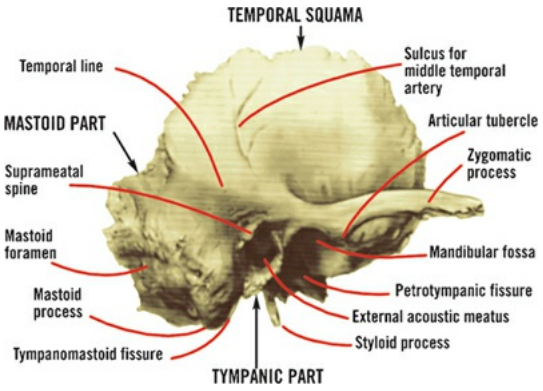


Figure 1-1 Right temporal bone, lateral view. (Reproduced with permission from Anson and Donaldson.[2])

The *tympanic* portion of the TB is an incomplete cylindrical portion of the TB that, together with the squamosal portion, forms the medial part of the external auditory canal. This portion of the external auditory canal is 2 cm in length by 1 cm in diameter. Its anterior boundary is the posterior limit of the mandibular fossa; medially, its border is the tympanic membrane. The posterior part fuses with the mastoid component of the TB at the tympanomastoid suture. Failure in development of this part of the TB is responsible for congenital aural atresia, a form of conductive hearing loss correctable by surgery.

The major portion of the TB formed by the *mastoid* portion attributes its large size to extensive pneumatization. The mastoid process projects posteriorly and inferiorly behind the external auditory meatus and serves as the attachment for the sternocleidomastoid muscle. A deep groove in its inferior aspect houses the posterior belly of the digastric muscle, which is innervated by the facial nerve. The superior surface of the mastoid compartment is formed by a thin plate of bone known as the tegmen mastoidea. Posteriorly, it forms the anterior plate of the posterior cranial fossa and is indented by a groove for the sigmoid sinus. The superior and inferior petrosal sinuses travel medially along the superior and inferior aspects of this part of the TB.

The *petrous* portion of the TB forms its medial part inferior to the middle cranial fossa; posteriorly, it forms the anterior surface of the posterior cranial fossa (Figure 1-2). The superior surface of the petrous bone is highlighted by the prominence of the superior semicircular canal, a landmark in surgery within the middle cranial fossa. Anterior to this portion of the petrous bone is the hiatus for the greater superficial petrosal nerve, which joins with the geniculate ganglion of the facial nerve. In some TBs, this hiatus is enlarged, and the geniculate ganglion may be exposed in the middle cranial fossa. Anterior and medial to this region is a concave area for the semilunar ganglion of the trigeminal nerve. On the

posterior surface of the petrous bone are several important landmarks. The most obvious aperture is the internal auditory meatus (canal) that transmits the seventh and eighth cranial nerves as well as the labyrinthine artery or loop of the anterior inferior cerebellar artery (Figure 1-3). The lateral end (fundus) of the internal auditory canal (IAC) is divided horizontally by the falciform crest.[1–3] The superior compartment contains the facial nerve anteriorly and the superior division of the vestibular nerve posteriorly (Figure 1-4). The inferior compartment transmits the cochlear nerve anteriorly and the inferior division of the vestibular nerve posteriorly. The endolymphatic sac may be found in a depression covered by a bony shelf (operculum) anterior to the sigmoid groove. It narrows down into the vestibular aqueduct as the endolymphatic duct. The depression for the semilunar ganglion and the fifth cranial nerve on the anterior surface of the petrous bone also carries the sixth cranial nerve through a dural canal referred to as Dorello's canal. These two nerves may be involved in inflammatory or neoplastic processes that occupy the petrous apex (PA) and are responsible for the clinical syndrome known as Gradenigo syndrome (fifth cranial nerve pain, diplopia from lateral rectus muscle palsy, and otorrhea).

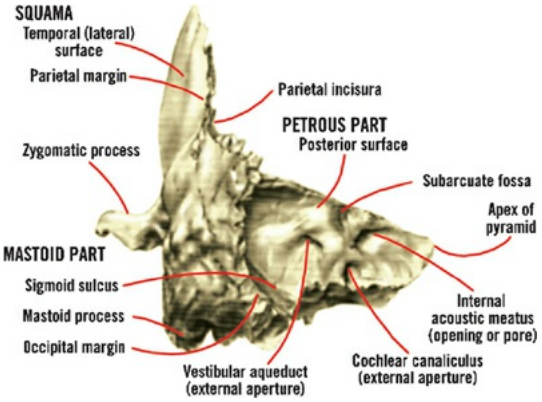


Figure 1-2 Left temporal bone, posterolateral view. (Reproduced with permission from Anson and Donaldson.[2])

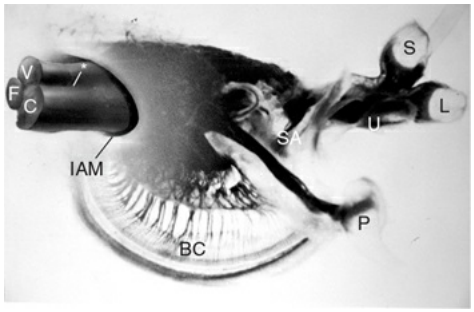


Figure 1-3 This orientation of the human inner ear dissection shows the anatomical relationship of the nerves and sense organs when viewed from the posterior surface of the temporal bone. IAM = internal auditory meatus; V = vestibular nerve trunk; C = cochlear nerve trunk; F = facial nerve; BC = basal turn of cochlea; SA = saccule; U = utricular

nerve; S.L.P = superior, lateral, and posterior semicircular canal ampullae; * = cleavage plane between vestibular and cochlear nerve trunks.

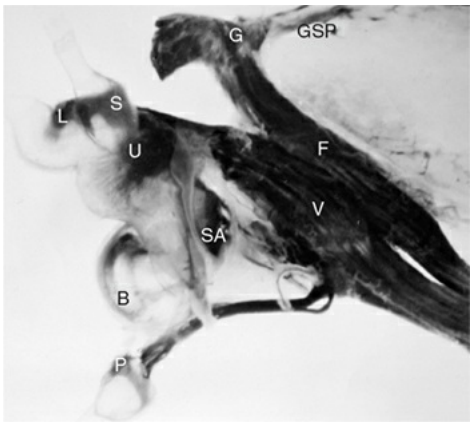


Figure 1-4 Human inner ear dissection with nerve supply demonstrates the relationship of the facial (F) and superior vestibular nerve (V) in the superior compartment of the internal canal. B = basal turn of cochlea; GSP = greater superficial petrosal nerve; G = geniculate ganglion; L = lateral canal crista; P = posterior canal crista; S = superior canal crista; SA = saccule; U = utricle.

AUDITORY SYSTEM

External Ear

The external or outer ear is that portion of the ear that is lateral to the tympanic membrane (Figure 1-5). It consists of the external auditory canal as well as the auricle and cartilaginous portion of the ear.

The auricle is a semicircular plate of elastic cartilage characterized by a number of ridges or grooves. The major ridges of the auricle are the helix and antihelix, the tragus and antitragus, which surround the concha, which is the scaphoid depression posterior to the external auditory meatus. The cartilage of the external auditory meatus is continuous with that of the outer portion of the ear canal and auricle.

The external auditory canal is made up of a cartilaginous extension of the auricle in its outer half and the mastoid and tympanic portion of the TB in its medial half. It is bounded medially by the tympanic membrane and is lined with skin that is thin with little subcutaneous tissue medially but laterally contains numerous hair follicles and ceruminous and sebaceous glands. The bony external auditory canal averages 3.5 cm in length, with a diameter of 1 cm. The tympanic membrane is composed of three layers: the outer squamous cell epithelial layer,

the medial mucosal layer facing the middle ear, and the fibrous layer or tunica propria, forming the substance of the tympanic membrane.[4] The fibrous layer gives the tympanic membrane its shape and consistency. Radial fibers of the tunica propria insert into the manubrium, circumferential fibers providing strength without interfering with vibration, whereas tangential fibers reinforce the architecture of the tympanic membrane. These physical characteristics are important for the vibratory characteristics necessary for sound transmission.

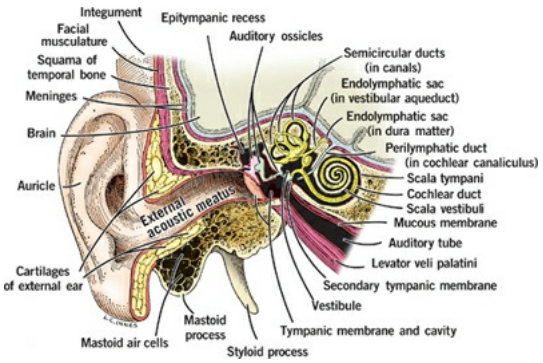


Figure 1-5 General relationship of parts of the ear (semidiagrammatic). (Reproduced with permission from Anson and McVay.[3])

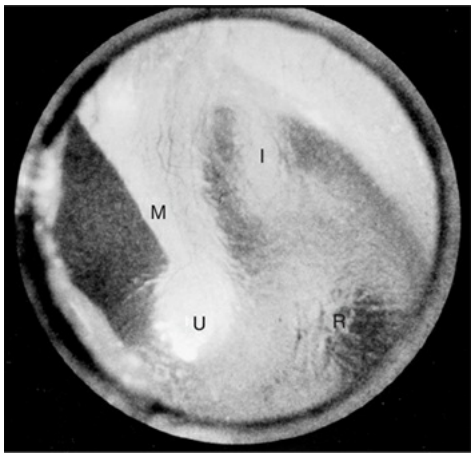


Figure 1-6 Photograph of a normal left tympanic membrane. I = long process of incus; M = malleus; R = round window niche; U = umbo.

The tympanic membrane is identified by a prominent landmark, the manubrium of the malleus, which is limited superiorly by its lateral or short process and inferiorly by a rounded end referred to as the umbo (Figure 1-6). The umbo forms the deep apex of the conical shape formed by the tympanic membrane. The tympanic membrane is incomplete superiorly, where it lacks a fibrous layer in the portion superior to the short process of the manubrium.[5] Since it lacks a fibrous layer, this portion is called the pars flaccida (Shrapnell's membrane). The major or inferior portion of the tympanic membrane is referred to as the pars tensa.

Middle Ear

The space between the tympanic membrane and the bony capsule of the labyrinth in the petrous portion of the TB contains the ossicular chain with its associated muscles, the aperture of the eustachian tube, and the vascular system. The tympanic cavity is divided into the epitympanic, mesotympanic, and hypotympanic regions. The *hypotympanic* portion is that portion of the middle ear that lies inferior to the aperture of the eustachian tube and the round window niche (RWN). This portion of the middle ear contains various bony trabeculae and the bony covering of the jugular bulb. This bony surface may be dehiscent, exposing the jugular bulb in the hypotympanic region. Inferiorly, a small channel (the inferior tympanic canaliculus) transmits Jacobson's nerve (a branch of cranial nerve IX).

The *mesotympanic* portion of the middle ear is limited superiorly by the horizontal portion of the facial canal and inferiorly by the RWN. This region contains the oval and round windows, the stapes bone, the stapedius muscle posteriorly, and the canal for the tensor tympani muscle anteriorly. The oval window is kidney bean shaped with a convex superior rim and a concave inferior rim. In the oval window, the footplate of the stapes bone is held in place by the annular ligament. The RWN forms a deep recess often covered with various mucous membrane configurations that obscure the round window membrane (RWM). The RWM is a fibrous membrane covered with a layer of mucosa that is roughly kidney bean shaped, with a major component anterior and inferior and a minor component located posteriorly and horizontally in the RWN. Posteriorly in the mesotympanum there are two bony recesses of clinical importance.[6] The recess lateral to the vertical segment of the facial canal is called the facial recess. The space medial to the facial canal is called the sinus tympani (Figure 1-7). These two recesses are important clinically as they frequently harbor chronic middle ear infection and must be controlled in surgery. The facial recess also provides access to the middle ear space and RWN in those procedures in which the ear canal wall is preserved, that is, intact canal wall mastoidectomy, cochlear implantation. A bony projection from the facial canal (pyramidal eminence) contains the tendon of the stapedius muscle before its insertion into the neck of the stapes bone. The most anterior portion of the middle ear space is called the *protympanum* and is bordered superiorly by the orifice of the eustachian tube and anteriorly by the canal for the internal carotid artery (see Figure 1-7).

The *epitympanum* is the portion of the middle ear that is limited superiorly by

the bony roof of the middle ear called the tegmen tympani. This bony landmark is continuous posteriorly as the tegmen mastoidea. The medial wall of the epitympanum is formed by the bony prominence of the lateral and superior semicircular canal ampullae as well as the epitympanic portion of the facial (fallopian) canal. The head and neck of the malleus and its articulation with the body and short process and a portion of the long process of the incus occupy most of the space in the epitympanum. These two ossicular masses are held in place by ligaments anteriorly and posteriorly to provide an axis of rotation for the ossicular chain (Figure 1-8). The epitympanic space communicates posteriorly through a narrow opening called the aditus ad antrum to the central mastoid tract of the mastoid cavity. Anteriorly, the epitympanum is separated at the cochleariform process from an anterior epitympanic cell of variable size by a bony and mucous membrane barrier, which may completely or incompletely separate the two compartments. This anterior epitympanic space is formed by pneumatization from the protympanum (see Figure 1-8). The anterior epitympanic space is also important surgically as it may contain inflammatory tissue (ie, cholesteatoma) that has extended from the protympanum.

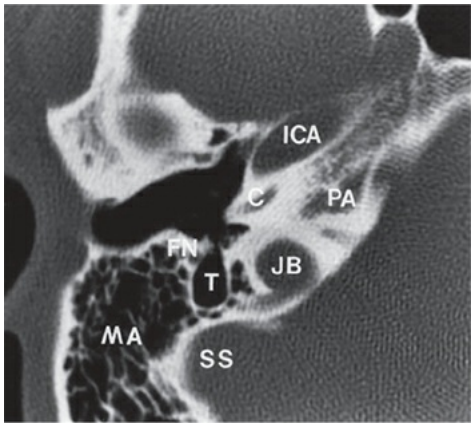


Figure 1-7 This axial computed tomographic scan of the temporal bone illustrates a normal mastoid cell system (MA), the horizontal segment of the internal carotid artery (ICA), the jugular bulb (JB), the sigmoid sinus (SS), and a nonpneumatized petrous apex (PA). C = basal turn of the cochlea; FN = facial nerve; T = sinus tympani.

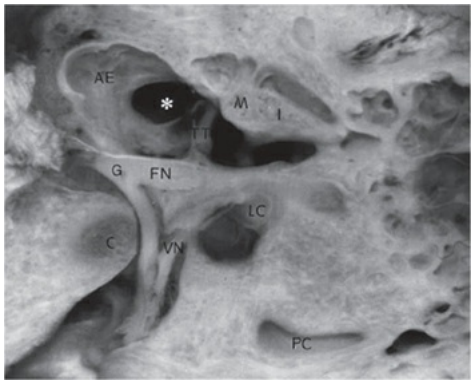


Figure 1-8 This horizontal cut through a celloidin-embedded temporal bone illustrates the relationship of the facial nerve (FN) to the superior division of the vestibular nerve (VN) in the internal auditory canal. The axis of rotation of the head of the malleus (M) and body of the incus (I) with their ligamentous attachments in the epitympanum is shown. AE = anterior epitympanic space ventilated into the protympanum (*); LC = lateral semicircular canal crista and ampulla; PC = posterior semicircular canal; C = endosteum of the cochlea (basal turn); TT = tensor tympani tendon; G = geniculate ganglion.

Auditory Ossicles. Sound pressure energy is transmitted from the tympanic membrane across the middle ear space by the ossicular chain comprised of the malleus, incus, and stapes (Figure 1-9). The head of the malleus and body of the incus function as a unit suspended by ligaments in the epitympanum. The tip of the long process of the incus articulates at a right angle with the head of the stapes so that the sound energy transmission initiated by medial displacement of the tympanic membrane is carried by the parallel displacement of the elongate processes of the malleus and incus to the head, crura, and footplate of the stapes (see Figure 1-9). Since the surface area of the tympanic membrane is larger than that of the stapes footplate by a ratio of 25 to 1, the sound pressure density in the oval window and the inner ear fluids is similarly increased. Maintaining this ratio by various reconstructive methods constitutes an important principle in middle ear surgery. The stapes therefore acts in a piston-like fashion in the oval window. The stapes bone is shaped like a stirrup with a head, neck, and footplate or base. The crura are bowed, the posterior one more so than the anterior, and fused with the footplate, which is formed from both otic capsule and periosteal bone. These auditory ossicles are controlled to some degree by two middle ear muscles, the tensor tympani and the stapedius. The tensor tympani muscle is housed in a bony semicanal in the anterior mesotympanum just superior to the orifice of the eustachian tube (Figure 1-10). The muscle converges posteriorly into a tendinous segment that is anchored at the cochleariform process and turns abruptly lateralward to insert on the neck of the malleus. The tensor tympani muscle is innervated by a branch of the fifth cranial nerve. Its motoneurons are located centrally in the parvocellular division of the trigeminal motor nucleus, and its action causes the drumhead to be pulled medially, thus raising the resonant frequency of the sound conduction system.

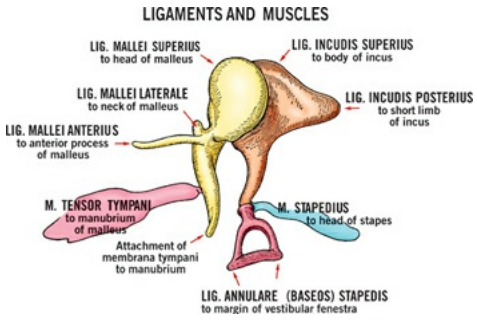


Figure 1-9 Auditory ossicles: their adult form and muscles and ligaments. (Reproduced with permission from Anson.[6])

The stapedius muscle arises within either its own or the fallopian canal and is accompanied by the motor portion of the facial nerve. It converges superiorly and anteriorly to form the stapedius tendon, which emerges through the pyramidal eminence to insert at the neck of the stapes. The stapedius muscle is innervated by a branch of the seventh nerve and its motoneurons are located in the brainstem in the interface between the facial nucleus and the lateral superior olivary nucleus. Contraction of the stapedius muscle displaces the stapes posteriorly and attenuates sound transmitted by the ossicular chain. Since reflex contraction of the stapedius muscle is activated by sound, it is regarded as a protective mechanism for the cochlea.

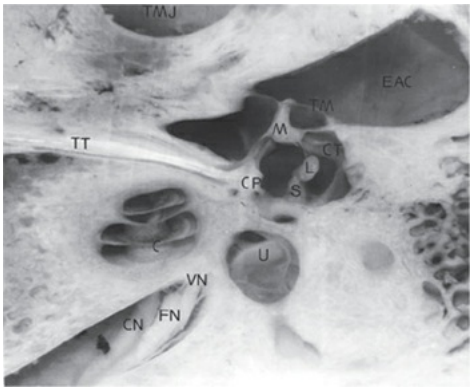


Figure 1-10 A more inferior cut through the same temporal bone as in Figure 1-8 demonstrates the cochlea (C), the utricular macula and its nerve (U), the cochlear nerve (CN), the facial nerve (FN), and vestibular nerves (VN) in the internal auditory canal. The muscle and tendon of the tensor tympani muscle (TT) overlie the cochlea as it turns laterally in the cochleariform process (CP) to attach near the neck of the malleus (M). The articulation of the long process (L) of the incus with the stapes head (S) can be seen in the mesotympanum. The chorda tympani nerve (CT) passes between the malleus and incus. TM = tympanic membrane; EAC = external auditory canal; TMJ = temporomandibular joint space.

Eustachian Tube. The eustachian tube is an essential communication between the nasopharynx and the middle ear (Figure 1-11). It is responsible for

pneumatization of the middle ear and the mastoid and for maintaining normal pressure between the middle ear and the atmosphere. It represents the pharyngeal extension of the first branchial arch and extends from the lateral wall of the nasopharynx.

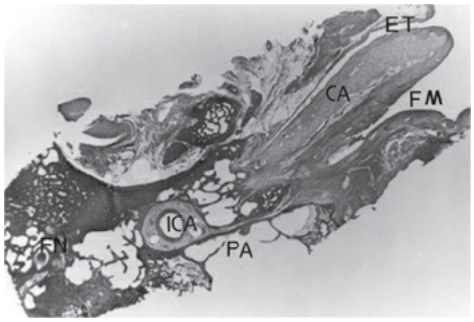


Figure 1-11 Low-power horizontal section through the eustachian tube (ET) as it passes into the protympanum. CA = cartilage of the ET; FM = fossa of Rosenmüller; ICA = internal carotid artery; FN = facial nerve; PA = petrous apex air cells.

The skeleton of the medial three-fourths of the eustachian tube is cartilage that is surrounded by soft tissue, adipose tissue, and respiratory epithelium. The cartilage of the eustachian tube, which is hook shaped on cross-section, is stabilized and displaced by contraction of the tensor veli palatini and levator veli palatini muscles on swallowing or yawning. The eustachian tube is thereby opened, allowing for pressure equalization. The lining epithelium of the cartilaginous portion is similar to that of the pharynx with pseudostratified columnar cell epithelium and many mucous glands. Posterior to the union of the cartilaginous and osseous portion of the eustachian tube where the isthmus is located, the mucosa undergoes transition to cuboidal or low columnar cell epithelium similar to the tympanic cavity epithelium. Neoplastic compression of the eustachian tube lumen near its pharyngeal orifice (Rosenmüller's fossa) will cause fluid to fill the middle ear space (serous otitis media), usually in an adult patient (see Figure 1-11). Investigation of this occult region by endoscopy, radiologic imaging, and biopsy is necessary in such instances.

In some patients fullness in the ear, autophony without hearing loss and a normal eardrum may be produced by an overly patent eustachian tube. Such patency of the tube may be caused by a decrease in the fat cells surrounding its cartilaginous segment associated with weight loss.

Nerve Supply of the External and Middle Ear. The auricle and the external auditory canal receive the sensory nerve branches from the fifth nerve via the auriculotemporal nerve and the greater and lesser auricular nerves. Branches from the glossopharyngeal and vagus nerves also contribute to this innervation.

The branch of the vagus nerve is referred to as Arnold's nerve, which travels in the posterior part of the ear canal in the tympanomastoid suture. When this nerve is stimulated, it produces a cough reflex as when the external auditory canal is being cleaned with an instrument. It may also participate in heralding a neoplastic or infectious process in distant regions of the aerodigestive tract also innervated by the vagus nerve (ie, larynx, hypopharynx) when pain is referred to the ear.

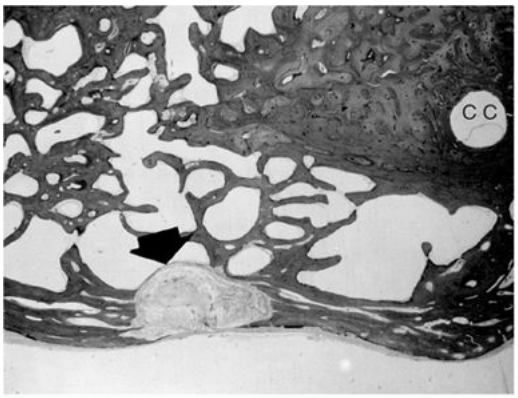
The main innervation to the middle ear space is through the tympanic plexus and Jacobson's nerve, which receives a major contribution from the glossopharyngeal nerve through the inferior tympanic canaliculus. This nerve travels in a bony sulcus or canal over the promontory along with the inferior tympanic artery anterior to the oval window and finally anteriorly to become the lesser superficial petrosal nerve. This nerve ultimately carries the fibers of the preganglionic neurons of the ninth nerve to the otic ganglion, where they synapse with postganglionic neurons and are carried over the auriculotemporal nerve to the parotid gland. The glossopharyngeal nerve provides sensory innervation to the pharyngeal tonsillar fossa and may be responsible for referred otalgia from neoplasms in this organ. Such referred ear pain is commonly encountered after tonsillectomy. Sympathetic fibers from the carotid plexus also contribute to the tympanic plexus. The chorda tympani nerve, which is a sensory branch of the facial nerve, will be discussed in the section on the facial nerve.

Mastoid Compartment. The air cell system of the mastoid bone represents an extension of the air compartment in the middle ear from the first pharyngeal pouch. This process occurs in development of the TB and may result in a variable degree of pneumatization in the mastoid compartment (see Figure 1-7). Recurrent infection in the middle ear and mastoid has been identified as a factor that may limit the extent of pneumatization of the mastoid air cell system, whereas absence of such infection may favor full development of the air cell system. The air cells in the mastoid compartment extend from the aditus ad antrum in the epitympanum to the central mastoid tract (antrum) from which further extension in several directions may occur. [7] The *posterior superior* cell tract extends medially at the level of the superior semicircular canal toward the PA, and the *posterior medial* cell tract extends toward the PA at the level of the posterior semicircular canal. The *supralabyrinthine* cell system extends medially superior to the labyrinth, whereas the *retrofacial* cell system extends posteriorly and inferiorly along the bony ear canal to pneumatize the mastoid tip. These cell tracts may vary considerably and are important for the surgeon to know as a guide in tracing infection into deep recesses of the mastoid compartment,

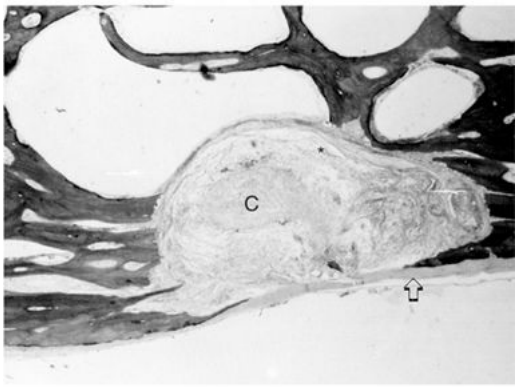
particularly the PA.

Normal structures may be aberrantly located near pneumatized portions of the TB where they may become important in adult life. Such are the arachnoid villi whose function is the circulation of cerebrospinal fluid into the dural venous sinuses. Aberrant arachnoid villi in the middle or posterior cranial fossa may be located adjacent to the bone of pneumatized areas of the skull base where they may enlarge with time into arachnoid granulations which erode into the mastoid compartment in adult life (Figure 1-12). Their clinical presentation as spontaneous cerebrospinal fluid otorrhea or rhinorrhea[8] requires timely diagnosis and treatment for the astute clinician (Figure 1-13). The prevention of intracranial morbidity (meningitis, brain abscess) from acute infection in these bony cavities is the goal of early recognition.

Cell tracts arising from the middle ear space also are important for the surgeon. These cell tracts, particularly those that may lead to air cell development in the PA, are those that course inferior to the labyrinth or those that extend around the canal of the internal carotid artery (see Figure 1-14). Extensive pneumatization in the development of the PA may create air cells that can become isolated when the cell tract is obliterated by bone or fibrous tissue leading to the formation of cholesterol cysts over a period of many years. Over time, these cysts erode the surrounding bone and may reach considerable size in early or late adulthood. Compression of the trigeminal nerve and the sixth nerve near the PA may present a clinical picture similar to Gradenigo syndrome, which is the clinical manifestation of petrous apicitis.



(A)



(B)

Figure 1-12 (A) Medium power view of an arachnoid granulation (*arrow*) located on the posterior surface of the temporal bone. CC = Crus Commune. (B) Higher power photo shows the arachnoid core (C) surrounded by the subarachnoid space (*) which contains spinal fluid. Arrow indicates the dura mater. Communication with the mastoid air cells is eminent.

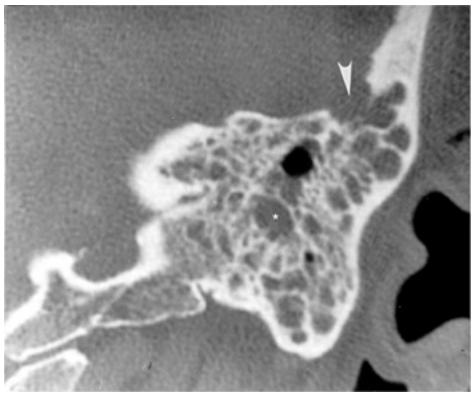


Figure 1-13 This coronal CT demonstrates filling of a mastoid compartment (*) with CSF from a middle fossa arachnoid granulation (*arrow*).

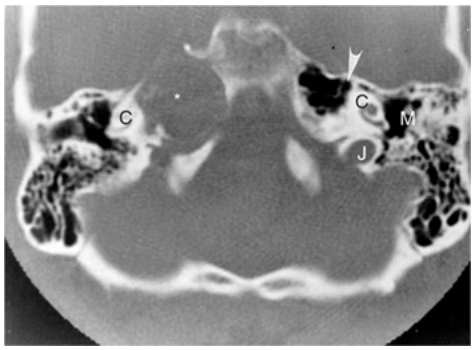


Figure 1-14 Axial CT in a patient with an expanding cholesterol cyst in the petrous apex (*) while the contralateral temporal bone illustrates pneumatization of the apex region (*arrow*) which leads to cyst formation. C = cochlea; M = mastoid compartment; J = jugular foramen.

The PA is of special interest because of a variety of lesions that may involve this region. The clinical manifestation may be subtle and requires a high index of suspicion to pursue the diagnosis.[9] Imaging (both computed tomo- graphy and magnetic resonance imaging) is especially sensitive to the identification of pathology in the PA. Since the composition of the PA can include air cells (see Figure 1-14), bone marrow (Figure 1-7), the internal carotid artery (Figure 1-7), and the cartilage of the foramen lacerum, the list of lesions that occur here is lengthy. Cholesterol cysts and congenital cholesteatomas are the most common; however, infection, bone marrow neoplasms, cartilage tumors, metastatic malignancies, neurogenic tumors, and aneurysms of the internal carotid artery have been reported. Clinical signs of a progressive lesion in the PA relate to nearby structures: eustachian tube obstruction, facial pain or anesthesia, and lateral rectus muscle palsy.

Inner Ear

The petrous portion of the TB houses the labyrinth with its attendant sensory structures responsible for auditory and balance function. Within the bony labyrinth is contained the membranous labyrinth, which represents a continuous series of epithelial lined tubes and spaces of the inner ear containing endolymph and the sense organs of hearing and balance. The membranous labyrinth can be divided into three regions that are interconnected: the pars superior or the vestibular labyrinth with the exception of the saccule, the pars inferior (cochlea and the saccule), and the endolymphatic duct and sac. All of the sense organs of the labyrinth have in common that they contain hair cells with rigid cilia and are innervated by afferent and efferent neurons.[10,11] Displacement of the cilia of the hair cells is responsible for opening potassium and calcium channels that initiate

the electrical potential within the hair cell that is then leaked into the afferent neuron and carried to the brainstem.

Cochlea. The cochlear duct, the auditory portion of the labyrinth, extends approximately 35 mm.[12] The cochlear duct and associated sensory and supportive structures assume the form of a spiral similar to a snail shell of 2.5–2.75 turns (Figure 1-15). This allows the long cochlear duct to be contained in a small space.[13] A cross-section of a cochlear turn (Figure 1-16) demonstrates the essential structures in this sense organ. The scala media or cochlear duct containing endolymph is triangular in shape in cross-section. The basilar membrane forms the horizontal limb of the triangle, Reissner membrane, the superior limb, and the stria vascularis with spiral ligament on the vertical side. The cochlear duct is filled with a fluid referred to as endolymph, whereas the fluid in the scala vestibuli and scala tympani is perilymph. Perilymph of the two scalae communicates through the helicotrema at the apex of the cochlea (Figure 1-17). All of the structures of the cochlear duct and, particularly, the basilar membrane have a morphologic gradient whereby the width of the basilar membrane is narrowest at the basal end and widest at the apex. The spiral ligament and epithelial elements in the organ of Corti also have a morphologic gradient from base to apex (see Figure 1-15). This morphologic gradient, to a large degree, determines the location of maximal stimulation of the basilar membrane and inner hair cells by a given tone or frequency that is introduced to the inner ear. In this way, high frequencies are located at the base and low frequencies at the apex, with the frequency scale laid out in an orderly fashion over the remainder of the basilar membrane. The cochlear duct ends in a blind pouch (cecum) that is located near the RWM.

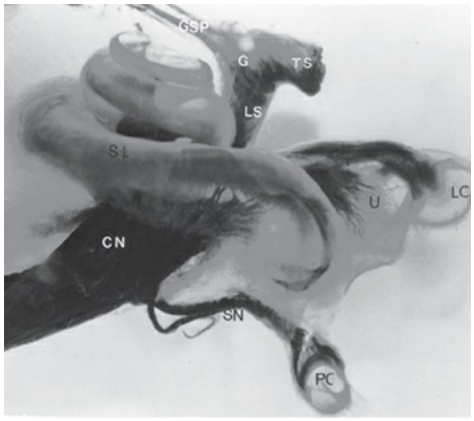


Figure 1-15 Photograph of a dissection of the human labyrinth and its nerve supply that demonstrates the 2.5 turns of the cochlear duct and the spiral ligament (SL). U = utricular nerve and macula; LC = lateral duct ampulla; PC = posterior duct ampulla; SN = singular nerve. Facial nerve: labyrinthine segment (LS) and tympanic segment (TS). G = geniculate

ganglion; GSP = greater superficial petrosal nerve; CN = cochlear nerve. (Reproduced with permission from Gacek.[13])

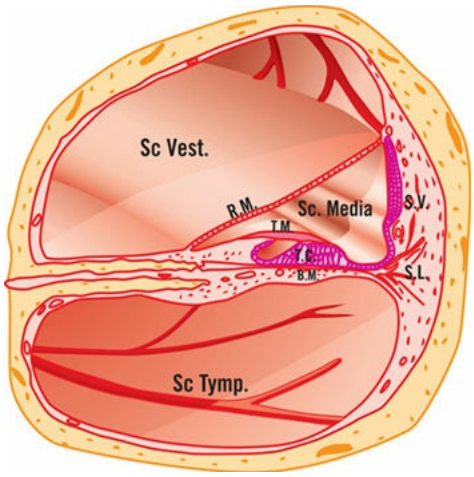


Figure 1-16 Section (diagrammatic) through the cochlea. Sc Vest = scala vestibuli; RM = Reissner membrane; Sc Media = scala media; TM = tectorial membrane; OC = organ of Corti; BM = basilar membrane; SV = stria vascularis; SL = spiral ligament; OSL = osseous spiral lamina; Sc Tymp = scala tympani.

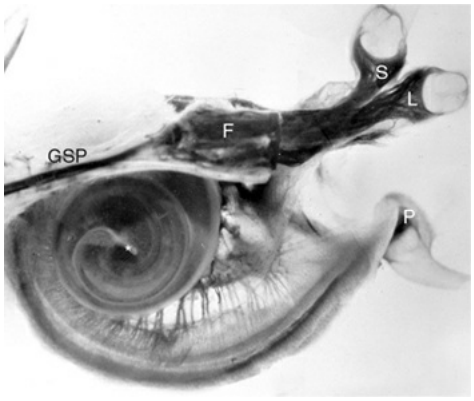


Figure 1-17 Human inner ear specimen viewed from the middle ear demonstrates communication of the vestibular and tympanic scalae at the helicotrema (*).

Perilymph of the scala vestibuli fills the vestibule under the stapes footplate. This perilymphatic compartment extends up the scala vestibuli of the cochlea and communicates with the perilymph in the scala tympani, which extends down the cochlea to terminate at the RWM. The perilymphatic compartment also communicates with the subarachnoid space through the periotic duct by way of the cochlear aqueduct that is filled with a trabecular meshwork of connective tissue capable of allowing some exchange of cerebrospinal fluid and perilymph. However, perilymph is primarily formed by filtration from the vascular network in the spiral ligament.

The organ of Corti is a complex sense organ that contains inner and outer hair cells and supporting cells resting on the basilar membrane, with the ciliated ends

of the hair cells protruding into or near a covering structure, the tectorial membrane (Figure 1-18). The apical portions of the hair cells are anchored in the cuticular plate,[8] with the stereocilia (usually 100–150 per cell) protruding through the cuticular plate.[7] The stereocilia of the outer hair cells make contact with the tectorial membrane, whereas the stereocilia of the inner hair cells lie free in the endolymphatic space inferior to the tectorial membrane. There are a single row of inner hair cells and three to five rows of outer hair cells. These cells differ morphologically in that the inner hair cells are more flask shaped and tightly surrounded by supporting cells and have stereocilia that are arranged in a linear fashion, whereas the outer hair cells are columnar and incompletely surrounded by phalangeal or supporting cells lying free in the perilymph of the organ of Corti.[11] The stereocilia of the outer hair cells form an inverted "W," and a basal body representing a rudimentary kinocilium located on the spiral ligament side of the ciliary tuft. The inner hair cells are supported by interphalangeal cells, whereas the outer hair cells are supported by Deiters cells inferiorly and laterally by Hensen cells. The tectorial membrane is anchored medially at the limbus and attached to the Hensen cells laterally by a fibrous net. The basilar membrane and tectorial membrane are displaced vertically by the traveling wave created by sound energy delivered to the oval window. Since the fulcrum of these two structures is separate, they will slide horizontally when stimulated, resulting in a shearing action between the tectorial membrane and the cuticular plate. The resultant displacement of stereocilia initiates an electrical event in the hair cell. The organ of Corti contains approximately 15,500 hair cells, with about 3,500 of them being inner hair cells and 12,000 being outer hair cells. These hair cells are innervated by afferent and efferent neurons in a complex but orderly manner. The afferent neurons to the auditory sense organ are bipolar neurons referred to as spiral ganglion cells that are located in Rosenthal canal of the bony modiolus. Approximately 30,000 spiral ganglion cells innervate the human organ of Corti (see Figure 1-16). The spiral ganglion takes the form of clusters of ganglion cells throughout the extent of the length of the cochlea. Ninety to 95% of the spiral ganglion neurons are type I neurons, which are large and myelinated and project a single dendrite directly to an inner hair cell. Approximately 10–20 type I spiral ganglion cells innervate one inner hair cell (Figure 1-19). These form the major afferent input from stimulation of the organ of Corti. Type I ganglion cells degenerate readily following injury to the dendrite.[14,15] The remaining 5% of afferent neurons in the spiral ganglion are type II ganglion cells, which are smaller and unmyelinated and have very thin distal processes. The dendrites of these type II neurons cross the tunnel

space along its floor enveloped by pillar cell processes and form spiral bundles between Deiters cells.[15] These dendrites then course apically between Deiters' cells to innervate several to many outer hair cells per type II dendrite. A type II ganglion survives following injury to its dendrite.

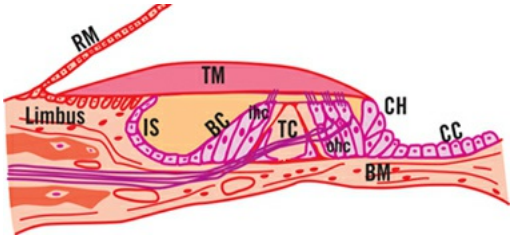


Figure 1-18 Detail of organ of Corti. RM = Reissner membrane; TM = tectorial membrane; IS = inner sulcus; BC = border cells; ihc = inner hair cells; TC = tunnel of Corti; ohc = outer hair cells; phc = phalangeal cells; CH = cells of Hensen; CC = cells of Claudius; BM = basilar membrane.

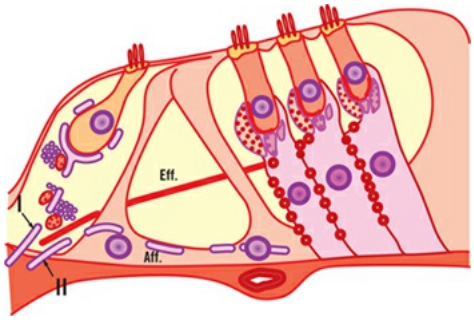


Figure 1-19 Schematic of the organ of Corti summarizing the efferent (*dark*) and afferent clear innervation termination. I = type I spiral ganglion cell afferents; II = type II spiral ganglia cell afferents.

The axons of the type I spiral ganglion cells project to the cochlear nucleus complex, which has anteroventral and posteroventral divisions of the ventral cochlear nucleus and the dorsal cochlear nucleus. Each type I afferent neuron bifurcates and also sends a trifurcating branch to the dorsal cochlear nucleus in an orderly fashion according to frequency.[16] Apical turn neurons terminate in the most medial portion of the nuclear complex, whereas the basal turn neurons terminate laterally. Remaining frequency projections are ordered between these two regions of the cochlear nucleus. The central termination of the type II ganglion cells is not known largely because the small caliber axons are difficult to trace for long distances.

This frequency organization of the auditory pathway characterizes the remainder of the afferent pathway from end-organ to cortex. Another feature of the afferent auditory pathway is that the numbers of neurons involved at the various nuclear way stations undergo a progressive increase from cochlear

nucleus to the cortex.[16] Although there are 30,000 spiral ganglion cells in the monkey auditory nerve, 88,000 neurons are found in one cochlear nucleus in the primate. One superior olivary complex contains 34,000 neurons, whereas the nucleus of the lateral lemniscus has 38,000 neurons, and at the inferior colliculus level there are almost 400,000 neurons on each side and at the medial geniculate body 500,000. The auditory cortex has approximately 10 million neurons.

A brief description of the afferent auditory pathway follows (Figure 1-20).[17] The cells of the dorsal cochlear nucleus project axons to the dorsal acoustic stria, which crosses the midline and ascends in the contralateral lateral lemniscus to terminate in the dorsal nucleus of the lateral lemniscus and the inferior colliculus, particularly its inferior half. The cell bodies of the ventral cochlear nucleus project axons to the ipsilateral accessory and main superior olivary nuclei and to the medial dendrites of the contralateral accessory olive. The neurons of the accessory olive have bipolar dendrites arranged horizontally. This arrangement is favorable to receive input from projections of both cochlear nuclei. As such, it is an important nuclear way station for determining sound localizaton. Some fibers of the intermediate and ventral cochlear striae travel beyond the superior olivary complex and enter the contralateral lateral lemniscus to terminate in the inferior colliculus. The superior olive is thought to function as both a relay station for the auditory pathway and as a reflex center. The best-known reflex mediated through the superior olive is the stapedius reflex. Stapedius muscle motoneurons are located in the interface between the superior olivary and the facial nerve nuclei. The accessory superior olive projects bilaterally in the lateral lemnisci to terminate in the dorsal nuclei of the lateral lemnisci and the inferior colliculi. The lateral superior olive projects homolaterally in the lateral lemniscus to terminate in the dorsal nucleus of the lateral lemniscus and also in the inferior colliculus. No neurons from the superior olivary nuclei project beyond the inferior colliculus.

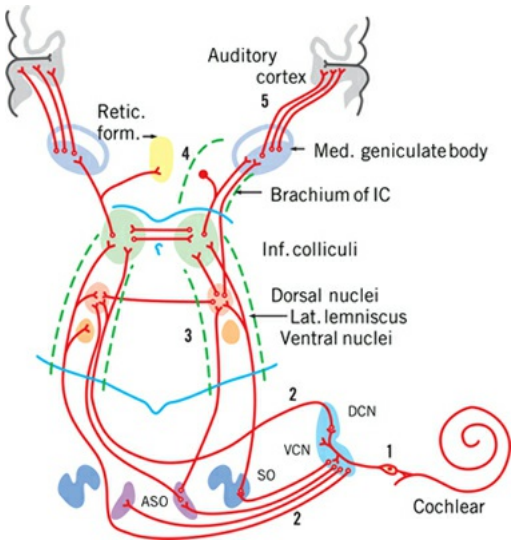


Figure 1-20 Diagram of the neuronal linkage that serves the afferent auditory pathway from one cochlea. Numerals indicate order of neuron units in the pathway. VCN = ventral cochlear nucleus; DCN = dorsal cochlear nucleus; SO = lateral superior olivary nucleus; ASO = accessory superior olivary nucleus; IC = inferior colliculus; Retic. form. = reticular formation. (Reproduced with permission from Gacek.[17])

Projections from the inferior colliculus are primarily to the medial geniculate body. However, some projections to the medial geniculate body are received from the nuclei of the lateral lemniscus. All ascending neurons terminate in the medial geniculate body so that the final projection pathway to the auditory cortex, which is a major one, is from the medial geniculate body to the auditory cortex. Furthermore, the only commissural or interconnections between the two sides of the auditory pathway are at the superior olivary level, the level of the nuclei of the lateral lemniscus, and the inferior colliculus. No commissural projections are present superior to the inferior colliculus.

The ascending auditory pathway, although comprised of four to five neurons in the linkage from end-organ to auditory cortex and having an increasing volume of neural units active at each level, nevertheless is precisely organized according to the frequency scale and project bilaterally but predominantly in a contralateral pathway to the auditory cortex.

Efferent Auditory Pathways. Paralleling the afferent auditory pathway is a descending pathway originating in the auditory cortex and terminating in the end-organ (Figure 1-21). This pathway does not involve as many neurons as the ascending or afferent pathway but has the feature of extensive ramification and formation of many terminals, which contact a large number of neurons. Nevertheless, the efferent pathway does not make as many neural contacts in the auditory nuclei as the ascending pathway. The descending auditory pathway

originates in the auditory cortex and initially projects to the inferior colliculus and the dorsal nucleus of the lateral lemniscus, with some termination in the medial geniculate body and the reticular formation.

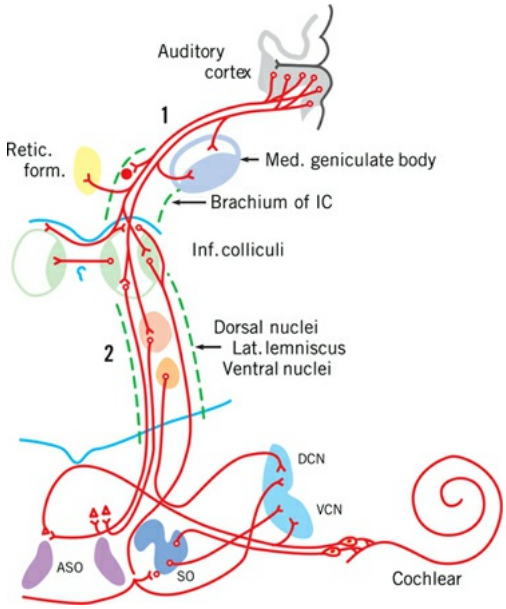


Figure 1-21 Diagram of the descending (efferent) auditory pathway. IC = inferior colliculus; DCN = dorsal cochlear nucleus; VCN = ventral cochlear nucleus; ASO = accessory superior olivary nucleus; SO = lateral superior olivary nucleus. (Reproduced with permission from Gacek.[17])

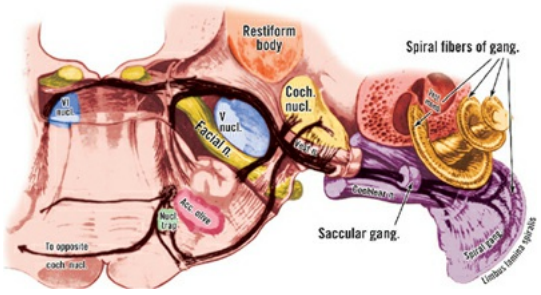


Figure 1-22 Drawing of the olivocochlear efferent pathway. See text for a description. (From Rasmussen.[19])

The next neuron in the descending chain is located in peripheral regions of the inferior colliculus and the nuclei of the lateral lemniscus. These projections terminate in brainstem neurons that give rise to efferent neurons projecting to divisions of the cochlear nucleus and to neurons that give rise to the third and final neuron in the descending auditory pathway, the olivocochlear bundle, which innervates the organ of Corti.[18–20]

The olivocochlear bundle has both an ipsilateral and a contralateral limb or component (Figure 1-22). The neurons, which number approximately 1,000–3,000, arise from neurons located within and near the superior olivary complex.

The contralateral limb of the olivocochlear bundle forms a major part of the efferent bundle, accounting for approximately three-fourths of the number of efferent neurons projecting to the organ of Corti in one ear. These axons arise from small neurons located near the accessory olivary nucleus,[18] ascend in the brainstem, and cross the midline at the level of the facial genu below the floor of the fourth ventricle. They are joined by the smaller ipsilateral component, which arises from similar olivary and periolivary neurons of the ipsilateral superior olivary nucleus before joining the contralateral limb as it enters the vestibular nerve root.[20] As it leaves the brainstem in the vestibular nerve, the bundle gives off collateral branches to the ventral cochlear nucleus and exits the vestibular nerve within the IAC just distal to the saccular ganglion. It then enters Rosenthal canal, where it travels perpendicular to the spiral ganglion cells and their dendrites, forming the intraganglionic or juxtaganglionic spiral bundle. The fibers from the efferent bundle are then given off regularly as they ascend the cochlea. These fibers penetrate the habenulae perforatae in the osseous spiral lamina along with afferent dendrites to enter the organ of Corti. The differential termination of the efferent neurons from the ipsilateral and the contralateral limbs is as follows: the fibers from the ipsilateral efferent component terminate on type I afferent dendrites and their terminals below the inner hair cell.[20,21] The contra lateral limb of the efferent pathway crosses the tunnel space and ramifies extensively to terminate on several outer hair cells in the first and second rows in a wider area than the inner hair cells at that region (Figure 1-19). The efferent innervation to outer hair cells is most extensive in the basal turn and decreases as the apex is reached.[21] This decrease is most noticeable in the outermost rows of outer hair cells of the organ of Corti. Whereas the inner hair cell type I neuron innervation provides the major afferent input to the cochlear nucleus, the efferent innervation of the outer hair cells is thought to alter mechanically the resistance at the outer hair cell level by contractile changes in the length of the outer hair cells. In this way, the sensitivity to sound stimulation of type I–innervated hair cells is modified. The predominant effect on auditory nerve transmission by stimulation of the efferent pathway has been to suppress the action potential in the auditory nerve.[22] It is thought that the outer hair cells with their type II afferent innervation and the efferent innervation sharpen the characteristic frequency discrimination of the organ of Corti. It is also probable that these hair cells, with their spontaneous and induced transition in length, may be responsible for otoacoustic emissions, which are small electrical potentials recorded from the ear.[23] In some patients in whom the inhibitory effect of the

efferent system is lost (ie, surgery, infection), the facilitated otoacoustic emissions may be perceived as ringing in the ear (tinnitus).

VESTIBULAR SYSTEM

The sense organs of the vestibular system are of two types, the cristae and the maculae (Figures 1-23 and 1-24). These sense organs have two types of hair cells,[24] type I and type II, which are secured in the neural epithelium by supporting cells (see Figure 1-24). The type I hair cells are flask shaped and are enclosed in a large calyx-type ending by one or two large-diameter afferent neurons (Figure 1-25). Type II hair cells are cylindrical in shape and contacted by bouton-shaped endings from both the afferent and efferent systems. These afferent neurons are myelinated but small compared with those that innervate type I hair cells. The crista is a ridge of neuroepithelial cells that traverse the ampullated end of each membranous duct. The ampulla contains the sense organ for the detection of angular acceleration and deceleration[25,26] (see Figure 1-23). The hair cells in the crista ampullaris are arranged with the type I hair cells near the crest of the crista and type II hair cells near the slopes.[24] It is noteworthy that the hair cells in cristae of the semicircular ducts are oriented in a single direction based on the alignment of their ciliary bundle.[24] The ciliary bundle of each vestibular hair cell consists of 100–150 stereocilia and a single kinocilium, which is located to one side of the stereocilia (see Figure 1-25). In the case of the horizontal duct, the kinocilium of each hair cell is located on the utricular side of the crista. In the vertical cristae, the kinocilia are located toward the nonampullated ends or away from the utricle (Figure 1-26). Sitting on top of the crista and reaching to the roof of the ampullary wall, even attached to it, is a gelatinous partition called the cupula, composed of mucopolysaccharides possessing an equal density to the endolymph, which surrounds it (see Figure 1-23). It is the displacement of the cupula that initiates through the hair cells an action potential in the neurons contacting the hair cells.[27] When the deflection of the cupula is toward the kinocilium, there is a depolarization of the hair cell neuron unit with an increase in the resting vestibular nerve action potential, whereas a deflection away from the kinocilium produces hyperpolarization or a decrease in the resting discharge of the vestibular neuron.[10] The semicircular ducts in each labyrinth are oriented to each of three planes in space, with the lateral ducts recording angular acceleration in a horizontal plane, whereas the