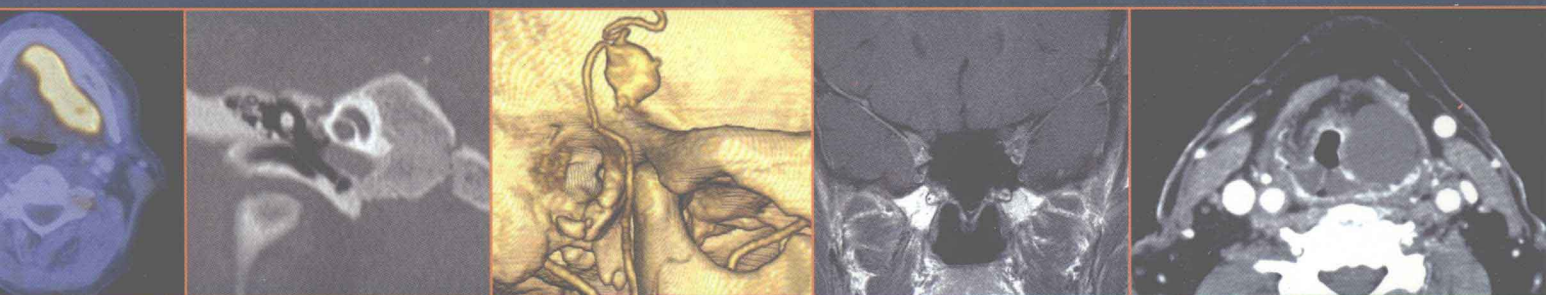


Peter M. Som • Hugh D. Curtin

# HEAD and NECK IMAGING

*Fifth Edition*



**Volume 1**

MIDFACE AND SINONASAL CAVITIES

ORBIT AND VISUAL PATHWAYS

CENTRAL SKULL BASE

TEMPORAL BONE

JAWS AND TEMPOROMANDIBULAR JOINTS

**FIFTH EDITION**

# HEAD and NECK IMAGING

**Volume 1**

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## **To Judy and Carole, again**

*Each new edition of our book seems to take more effort and consume more time to complete than any of the previous editions. Through the years that we have invested in this project, you have given us love, support, and inspiration while showing continued exceptional patience.*

*For just being there, we dedicate this fifth edition of Head and Neck Imaging to the both of you with our love and thanks.*

**PMS and HDC**

# Preface

Since the fourth edition of our book, technology has continued to advance, leading to better lesion conspicuity and diagnosis. In addition, new insights have been gained into embryology, physiology, and pathology that better allow us to understand the diseases we encounter. Taking all of this new information into account, we have attempted to make this fifth edition as complete a reference on the topics of head and neck imaging as possible. The new edition is a thorough updating of the prior edition. It has been reorganized, new topics have been added, and the book is now in full color.

Specifically, the embryology, physiology, and anatomy sections have been expanded. These are now complemented by numerous color drawings to illustrate specific points. We are pleased to have been able to include drawings from Frank Netter, *Sobotta's Anatomy*, *Gray's Anatomy*, and pictures from other sources along with numerous original works. These beautiful drawings help clarify the anatomy and complement the rest of the text, and we thank Elsevier for helping make their inclusion in the book possible. We also want to specifically thank the Netter family for helping us include Frank Netter's beautiful drawings.

The imaging atlases were also greatly expanded with more detail and anatomic variants being addressed in each area. Some chapters were greatly expanded and updated. This can be seen in the treatment of PET/CT imaging. New chapters were also added, such as Nonsurgical Treatment of Head and

Neck Cancer and Imaging-Based Intervention: Biopsy and Treatment. However, due to concerns about the overall size of this edition, almost all plain radiographs have been removed. Since these films are rarely used today in everyday practice, we thought the space would be better used to cover more fully the current topics of head and neck imaging. The fourth edition will remain available for those who want analysis of plain radiographs of the paranasal sinuses, for example.

As always, any multi-authored text is only as good as the contributions from the contributors, and we again thank all of them for the thorough treatments of their topics, especially in this current era of limited academic time. We also want to thank the dedicated people at Elsevier who worked so hard with us to produce this edition. Our readers have said that they use our text in several distinct ways. They use it as an anatomic reference to aid their interpretation skills, as a resource to look up a particular entity with which they are not familiar, and as a pictorial reference to compare to a case they are currently reading. In response to these uses, we have made this fifth edition as comprehensive as possible, and we hope that our readership will find this edition to be the complete resource that they expect and that they will use it to learn and review head and neck imaging, as well as an aid in the daily diagnosis of current cases. We also hope that they enjoy the field of head and neck imaging as much we do.

Peter M. Som  
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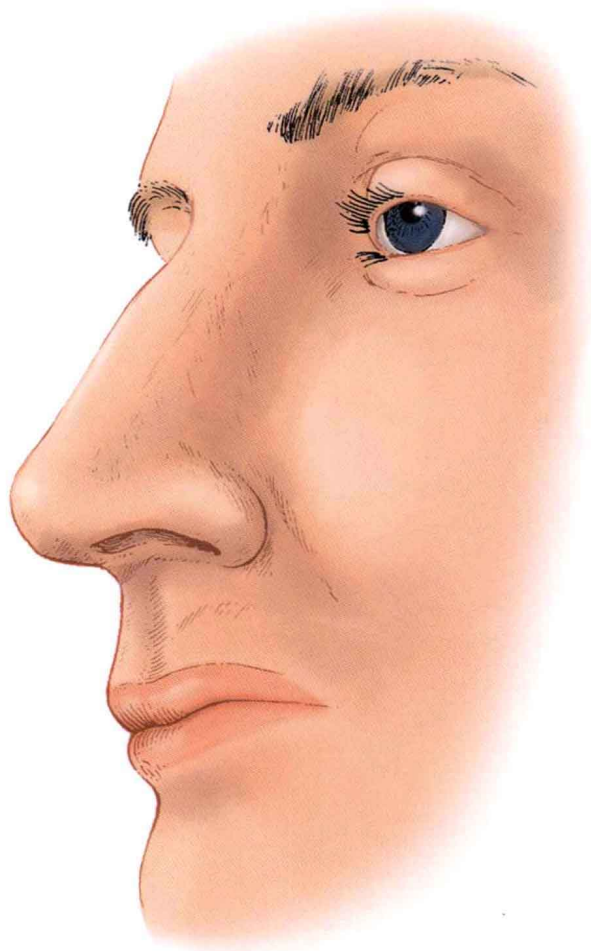
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## SECTION I

# MIDFACE AND SINONASAL CAVITIES







# Embryology and Congenital Lesions of the Midface

Thomas P. Naidich, Susan I. Blaser, Ruby J. Lien, David G. McLone,  
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The face and brain form by a series of cell migrations, proliferations and differentiations that are highly orchestrated through cascades of cell-signaling systems. Systematic variations in cell signaling determine the wide spectrum of craniofacial malformations, ranging from *hypo*expansion of the midline (as in holoprosencephaly) to *hyper*expansion of the midline (as in frontonasal dysplasia and craniofacial duplication).<sup>1</sup> Conservation of the signaling systems across organs explains the concurrence of craniofacial, oropalatal and limb malformations in the same patient. Conservation of signaling systems across species explains the utility of experimental animal data for understanding human disease.

The craniofacial structures are phylogenetically new. Ancestral forms evolved from filter feeding toward active predation by developing a nerve plexus to detect and capture prey, a cartilaginous and skeletal housing to provide fixed spatial orientation for the sensory apparatus, muscularized jaws to secure the prey, and muscularized gill arches to improve efficiency of predation.<sup>2</sup> Modification of migrating neural crest pigment cells already present in ancestral forms and shifts in the domains of signaling molecules like fibroblast growth factor 8 (FGF8) and the hedgehog signaling system led to novel features in a wholly new anterior body section (the “new head”).<sup>2</sup> Understanding the new head and its malformations requires knowledge of the *molecular morphology* of normal craniofacial embryogenesis and the derangements in molecular signaling that lead to malformation.<sup>1-26</sup>

Traditionally, congenital malformations have been defined by their effect on gross anatomy and classified by phenotypic similarities.<sup>27</sup> Clinically valid constellations of pathology have been called syndromes and named for the authors who reported them. However, phenotypically similar diseases may result from different molecular mechanisms, limiting the value of syndromic classifications. For that reason, this chapter reviews both the classical features of selected midface malformations and significant aspects of their molecular pathogenesis. To the fullest extent possible, the molecular pathways have been condensed to their essential components. Understanding of these pathways will prove to be the foundation for all future advances in appreciating these diseases.

## MOLECULAR MORPHOLOGY

### Nomenclature

By convention, *genes* are designated by their initials *in italics*. The gene product is designated by its initials without italics. Human genes and human gene products are written in uppercase letters, while the corresponding animal genes and gene products are designated by lowercase letters. Thus, the human gene sonic hedgehog is designated *SHH*, while the same mouse gene is designated *shh*. The human gene product is written SHH (sometimes Shh) and the mouse product shh. Genes and gene products that act earlier in the cascade are designated “upstream.” Those that result from upstream signals are considered to lie “downstream” in the cascade. Genes and gene products that increase the activity of other genes/gene products are designated activators, whereas those that reduce their activity are designated repressors. The gene products are proteins that may be spliced together in differing isoforms, each isoform having a different level of activity or specificity of action. Differing isoforms of the same product may serve as activators or repressors, even in the same cascade, creating positive and negative autoregulatory signaling loops. Mutations that increase signaling activity are designated “gain-of-function” mutations. Those that reduce signaling activity are designated “loss-of-function” mutations. Loss-of-function mutations in one of the two paired chromosomes may reduce the total output of gene product below a critical threshold, leading to mutations by *haploinsufficiency* of gene product.

### Transmembrane Signaling

In utero, signal transduction pathways normally relay information from outside the cell, through the plasma membrane and cytoplasm, into the nucleus to regulate and coordinate the expression of target genes (Fig. 1-1). From the nucleus, related information then passes outward to alter cytoplasmic structures, to modulate the cell response to incoming signals, and to coordinate activities of other cells, nearby or distant.<sup>3</sup> The signals employed often take the form of *ligands*, which may be diffusible (e.g., growth factors) or stationary (e.g., extracellular