1 SINONASAL TRACT

American Joint Cancer Committee Staging Criteria (6th ed.) Sinonasal Malignancies

Primary tumor (T)	
Maxillary sinus	
TX	Primary tumor cannot be assessed
T0	No evidence of primary tumor
Tis	Carcinoma in situ
T1	Tumor limited to the maxillary sinus mucosa with no erosion or destruction of bone
T2	Tumor causing bony erosion or destruction including extension into hard palate and/or middle nasal meatus, excluding extension to posterior maxillary wall and pterygoid plates
T3	Tumor invades bone of posterior maxillary wall, subcutaneous tissues, orbital floor, medial orbit, pterygoid fossa, and/or ethmoids
T4a	Tumor invades anterior orbit, skin of cheek, pterygoid plates, infratemporal fossa, cribriform plate, sphenoid, or frontal sinuses
T4b	Tumor invades orbital apex, dura, brain, middle cranial fossa, cranial nerves other than maxillary division of trigeminal nerve, nasopharynx, or clivus
Nasal cavity and ethmoid sinuses	
Tx	Primary tumor cannot be assessed
T0	No evidence of primary tumor
Tis	Carcinoma in-situ
T1	Tumor restricted to any one subsite, with or without bony invasion
T2	Tumor invades two subsites in a single region or extends into an adjacent region within the nasoethmoidal complex, with or without bony invasion
T3	Tumor invades medial orbit, orbital floor, maxillary sinus, palate, or cribriform plate
T4a	Tumor invades anterior orbital contents, skin of nose or cheek, minimal anterior cranial fossa extension, pterygoid plates, sphenoid, or frontal sinuses
T4b	Tumor invades orbital apex, dura, brain, middle cranial fossa, cranial nerves other than maxillary division of trigeminal nerve, nasopharynx, or clivus
Regional lymph nodes (N)	
Nx	Cannot be assessed
N0	No regional lymph node metastasis
N1	Metastasis in a single ipsilateral lymph node ⩽3 cm
N2a	Metastasis in a single ipsilateral lymph node >3 cm but ⩽6 cm
N2b	Metastasis in multiple ipsilateral lymph nodes ⩽6 cm
N3	Metastasis in a lymph node >6 cm

Distant metastasis (M)	
Mx	Cannot be assessed
M0	No distant metastasis
M1	Distant metastasis

Stage I	T1	N0	
Stage II	T2	N0	
Stage III	T3 T1 T2 T3	N0 N1 	
Stage IVA	T4a T1 T2 T3 T4a	N0 N1 N2 	M0
Stage IVB	T4b Any T	Any N N3	
Stage IVC	Any T	Any N	M1

ANATOMIC DEFINITIONS FOR THE SINONASAL TRACT
Nasal vestibule
Anterior boundary: nares
Posterior boundary: perpendicular line dropped from the frontonasal suture through the anterior aspect of the inferior turbinate
Nasal cavities
Anterior boundary: continuous with the vestibule
Posterior boundary: posterior choanae
Superior boundary: cribriform plate
Inferior boundary: hard palate
Medial boundary: nasal septum
Lateral boundary: lateral nasal wall with maxillary and ethmoid ostia and turbinates
Turbinates
Scrolllike projections of bone and vascular soft tissue
The superior turbinate is smallest, and the inferior turbinate is largest
Attaches to the lateral nasal wall anteriorly, the free edge, is posterior
Schneiderian mucosa
Pseudostratified columnar ciliated epithelium with goblet cells
The lamina propria is loose and well vascularized, with serous and mucinous glands

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Olfactory mucosa (OM)
Bipolar olfactory nerve fibers cross through the cribriform plate and terminate in the OM forming olfactory cilia
Bowman's glands, or olfactory glands, which appear similar to serous minor salivary glands
The frontal sinus
Paired sinuses between the internal and external cranial tables
Ethmoid complex
Paired complex of sinuses contains three to eighteen cells, grouped as anterior, middle, or posterior, according to the location of their ostia
Medial boundary: upper nasal fossa
Lateral boundary: lamina papyracea of the orbit
Superior boundary: fovea ethmoidalis which is the medial extension of the orbital plate of the frontal bone
Sphenoid sinus
Situated posterior to the ethmoid sinuses
Superior boundary: floor of the anterior cranial fossa
Posterior boundary: optic chiasm and the sella turcica
Lateral boundary: orbital apex, the optic canal, the optic nerve, and cavernous sinus
Inferior boundary: nasopharynx
Anterior boundary: nasal fossa
Maxillary sinus
Medial boundary: lateral wall of the nasal cavity (''party wall'')
The curved posterolateral wall separates the sinus from the infratemporal fossa.
The anterior sinus wall is the facial surface of the maxilla.
Inferior boundary: hard palate
Superior boundary: orbital rim and orbital apex

Nonneoplastic Lesions

Sinonasal Polyps

Sinonasal polyps result from expansion of the Schneiderian mucosa lamina propria by fluids and proteins. This can be caused by chronic allergy, vasomotor rhinitis, infectious rhinosinusitis, diabetes mellitus, cystic fibrosis, aspirin intolerance, and nickel exposure.

Between 10 and 20 percent of children with cystic fibrosis have nasal polyps. Generally, nasal polyps in children are uncommon, and 29 percent of such polyps in children are associated with cystic fibrosis.

Sampter's triad refers to the syndrome of nasal polyps, aspirin intolerance, and bronchial asthma. About 20 percent of patients with nasal polyps have asthma, and conversely about 30 percent of asthmatic patients have polyps.

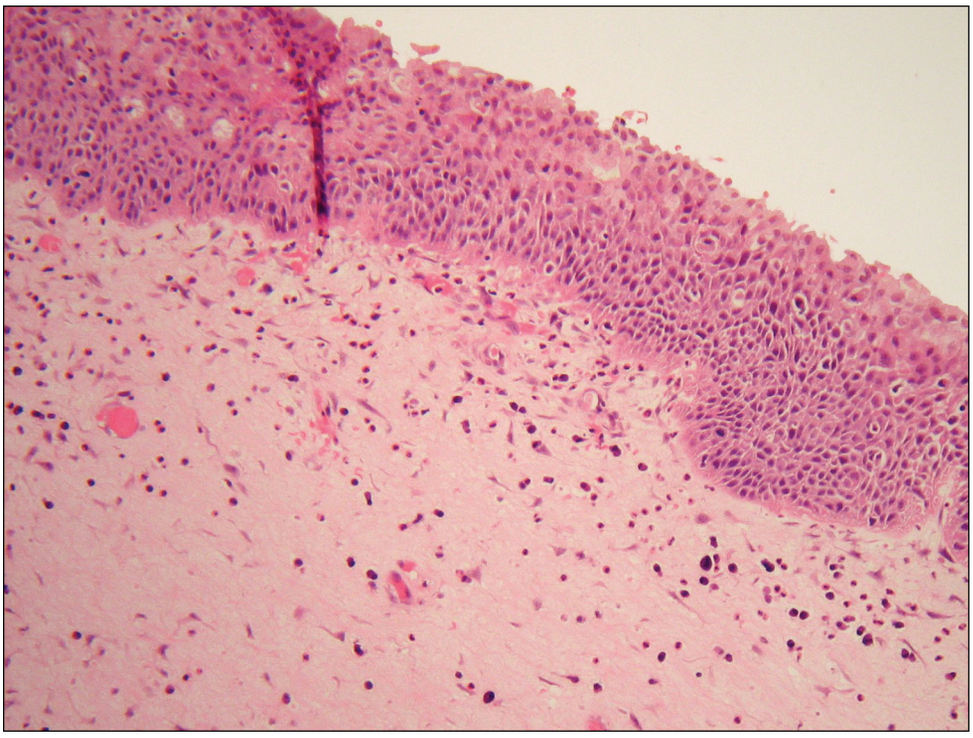


Figure 1.1 The lamina propria of a Schneiderian polyp is distended by proteinaceous exudate with variable inflammation. The underlying seromucinous glands may be present or absent. The basement membrane is thicker than normal.

Gross Examination:

- Single or multiple bilateral lesions; grossly translucent and soft.

Histopathology:

- Thickened basement membrane (Figure 1.1).
- Mucosal surface: respiratory epithelium or squamous metaplasia, ±hyperplasia (Figure 1.2).
- If diffuse squamous metaplasia and hyperplasia are present, then consider inverted papilloma.
- Allergic polyp – Inflamed Schneiderian polyp with minimal to marked infiltrate of eosinophils (Figures 1.3 and 1.4).
- If the surface epithelium has a "pierced" microglandular cribriform pattern, then consider oncocytic Schneiderian papilloma, respiratory epithelial adenomatoid hamartoma, or sinonasal serous hamartoma (Figures 1.5 and 1.6).

Antrochoanal Polyp

Antrochoanal polyps represent 4–6 percent of all sinonasal polyps; they arise in the maxillary antrum and prolapse through a sinus ostium. The polyp can extend posteriorly, further prolapsing through the posterior nasal choanae into the nasopharynx. Thus, a long stalk is characteristic. Stalk torsion can give rise to bizarre reactive fibroblasts within the lamina propria. Clinically, antrochoanal polyps can become quite large, mimicking a tumor.

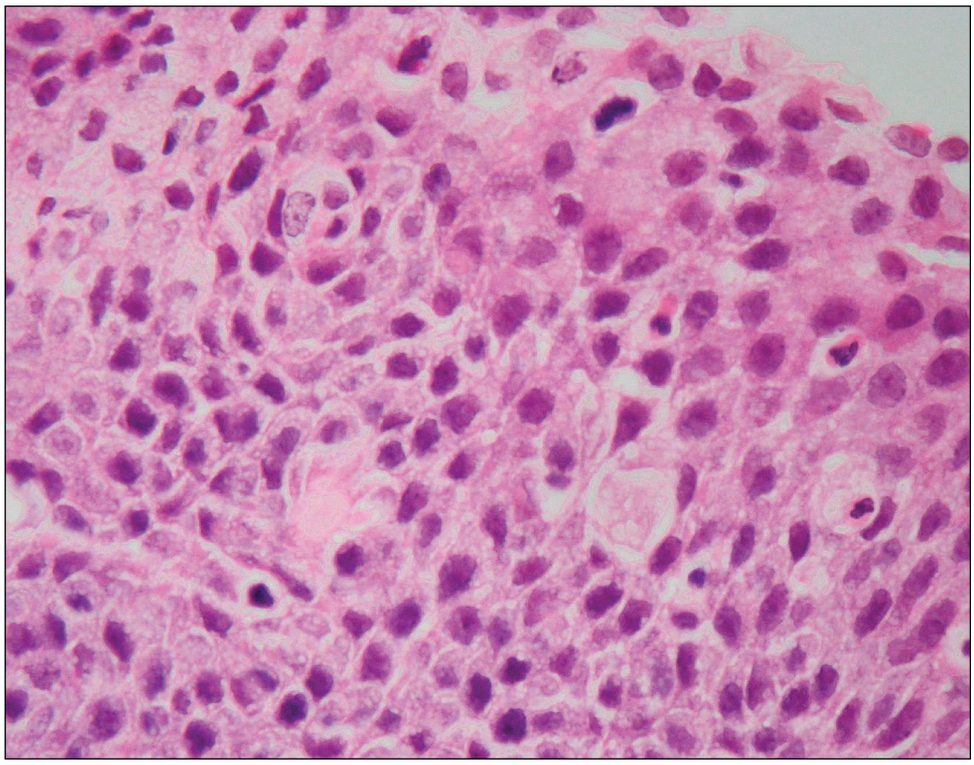


Figure 1.2 Squamous metaplasia can be common in polyps. Here we see reactive atypia due to inflammation.

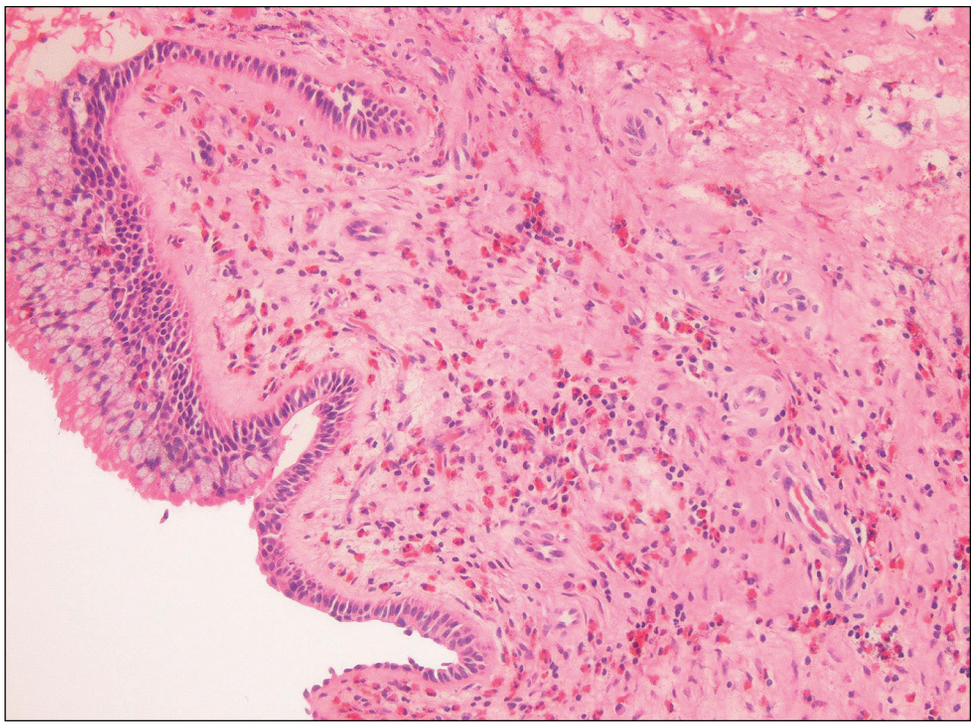


Figure 1.3 Eosinophils are seen in allergic polyps; only a scattering of eosinophils need be seen to deem the process as allergic. By contrast, nonallergic sinusitis or polyps contain a lymphoplasmatic infiltrate.

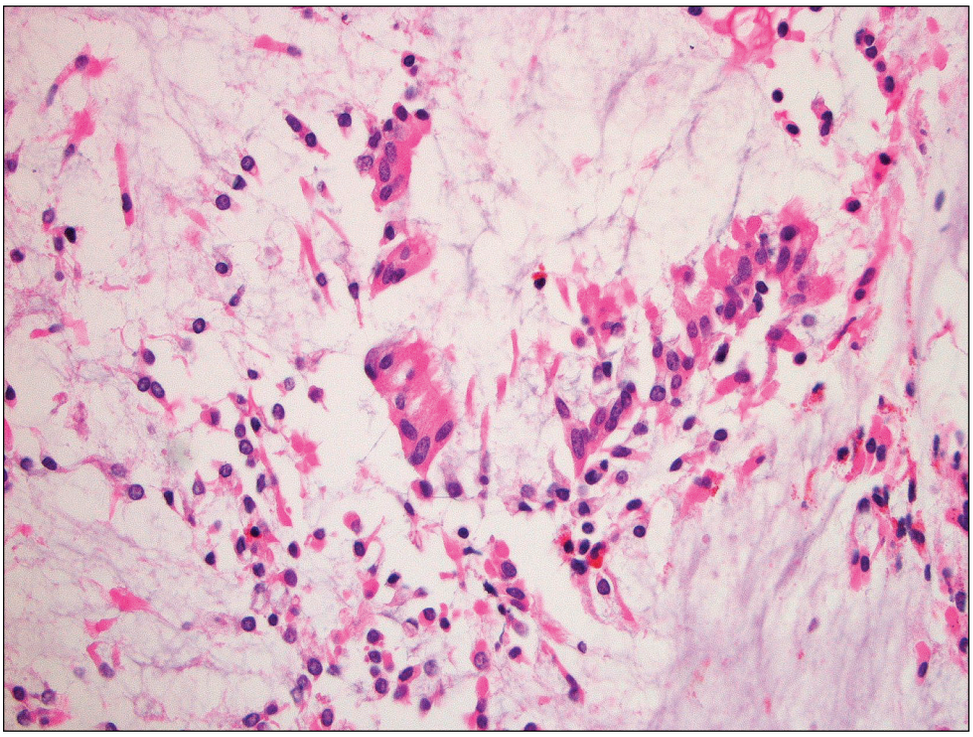


Figure 1.4 Extravasated mucin of an allergic polyp can contain shed respiratory columnar cells, goblets cells, and occasional eosinophils, as seen here. This differs from allergic mucin, which demonstrates eosinophil degranulation and Charcot–Leyden crystals (see **Figure 1.13**).

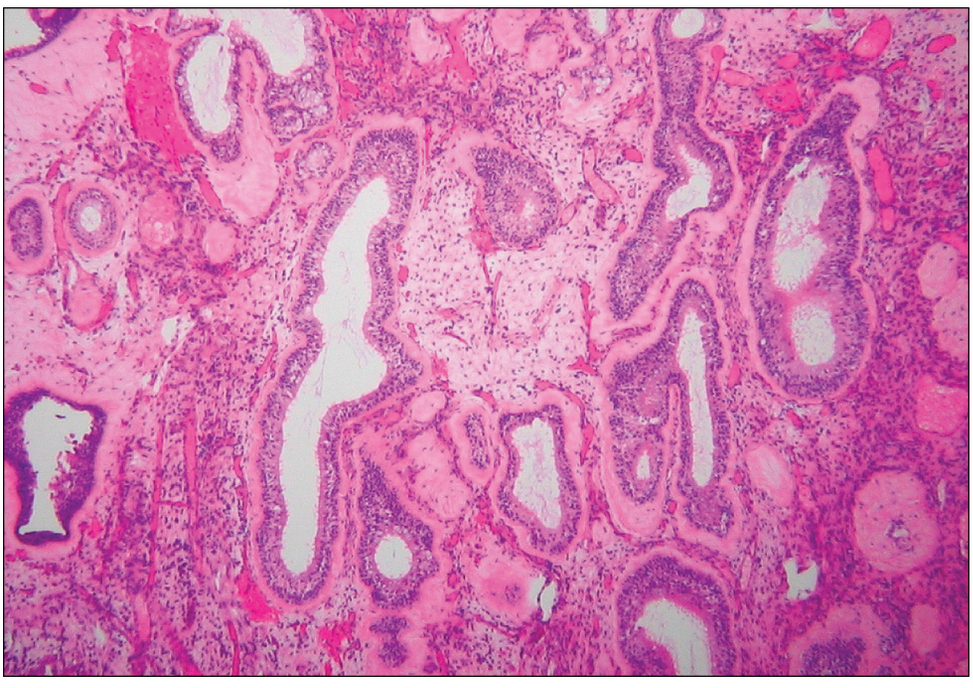


Figure 1.5 Long-standing respiratory polyp: The hyperplastic glands are crowded. The lamina propria is filled with reactive fibroblasts and inflammation. The differential diagnosis of hyperplastic polyps includes respiratory epithelial adenomatoid hamartoma (REAH) and low-grade intestinal-type adenocarcinoma (ITAC).

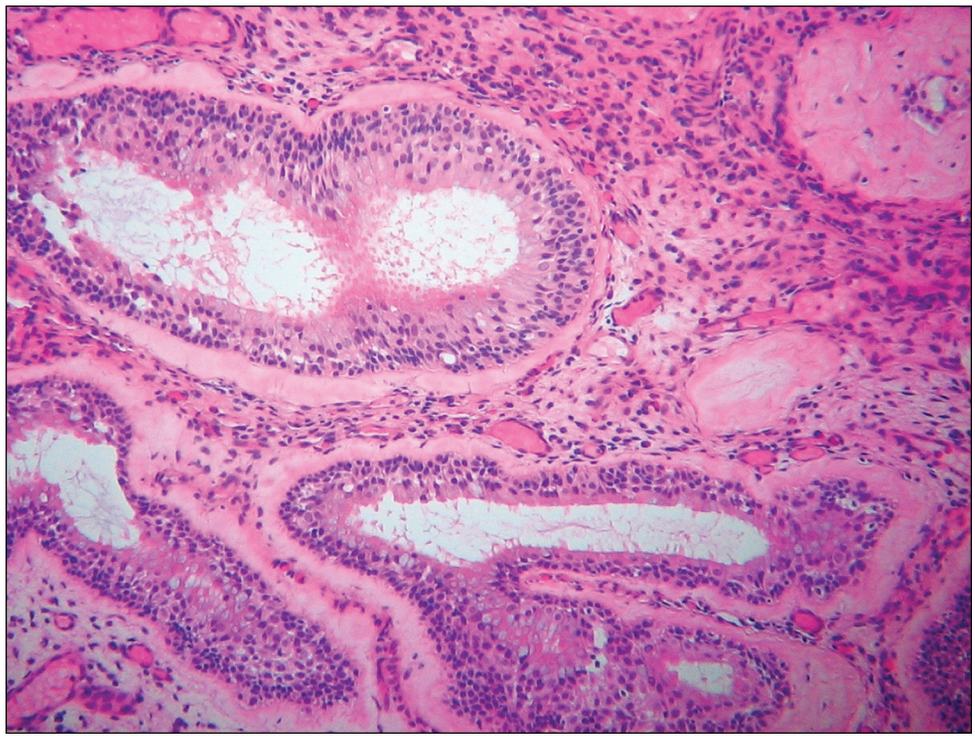


Figure 1.6 The basement membrane around these hyperplastic glands is thickened.

Antrochoanal polyp continued
Histopathology:

- The lamina propria may have few or no seromucinous glands (Figure 1.7).
- Atypical fibroblasts may mimic a malignant process (Figures 1.8 and 1.9).
- The reactive fibroblasts do not become confluent or hypercellular.
- Neovascularization, chronic inflammation, and Russell bodies support the diagnosis of antrochoanal polyp.

Allergic Fungal Sinusitis

Katzenstein first described allergic fungal sinusitis (AFS) in a group of patients with allergic symptoms; the sinonasal specimens were histologically similar to those of allergic bronchopulmonary aspergillosis. AFS represents 6–9 percent of all chronic sinus disease requiring surgery (Figure 1.10). Importantly, the role of fungi in AFS is actually still speculative. Many cases are associated with *A. fumigatus*, *A. flavus*, or dematiaceous fungi; but given the environmental ubiquity of these fungi, they may be incidental to the process.

The fungal hypothesis holds that susceptible individuals develop extreme eosinophil-driven hypersensitivity reactions to environmental fungi, and that an immune T_H2-like lymphocyte-mediated response to fungal antigens is responsible for the process. T_H2 cells regulate IgE production and allergic inflammatory response. Although fungal hyphae, by definition, are present within AFS, their role in initiating or promoting this disease remains circumstantial.

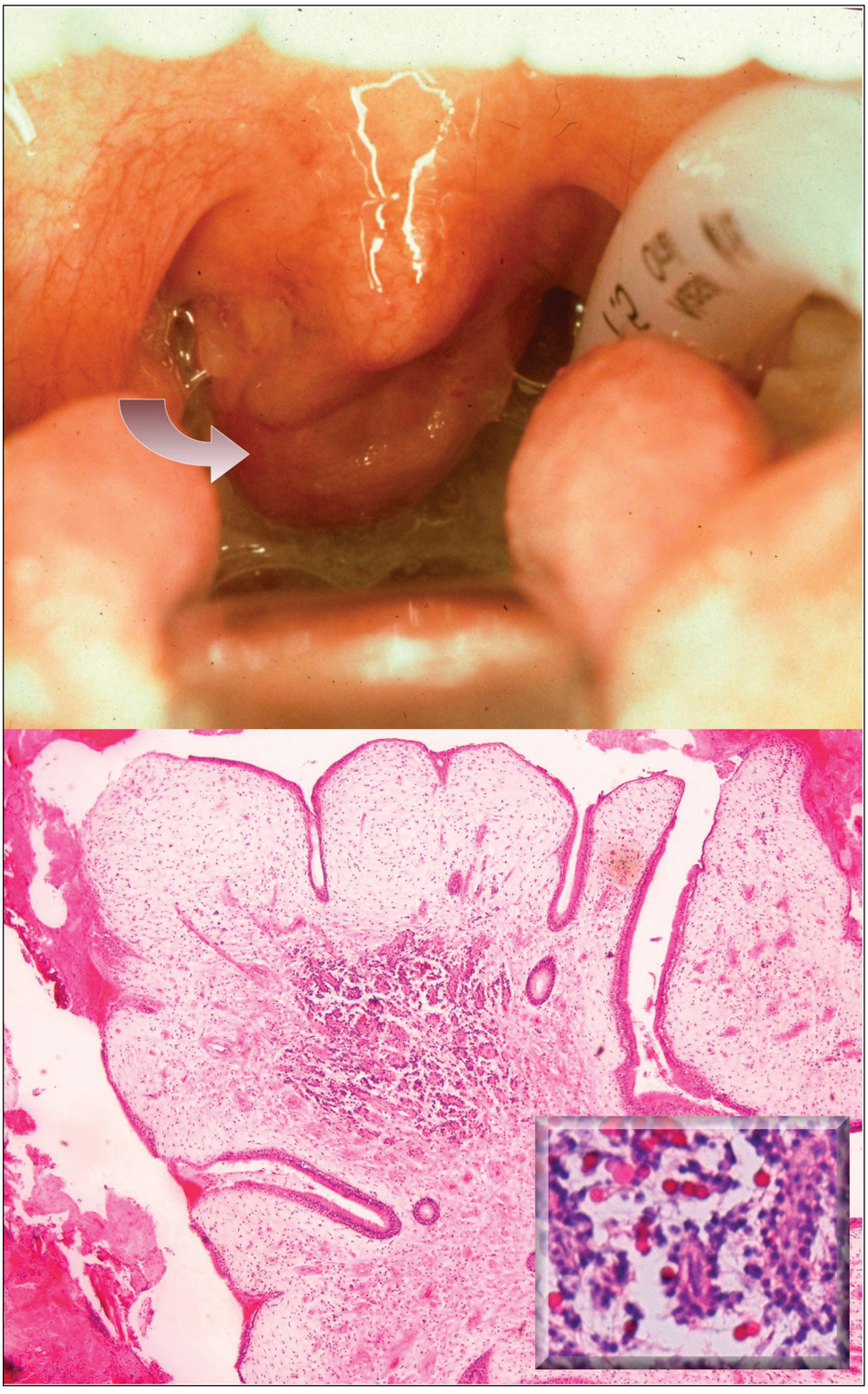
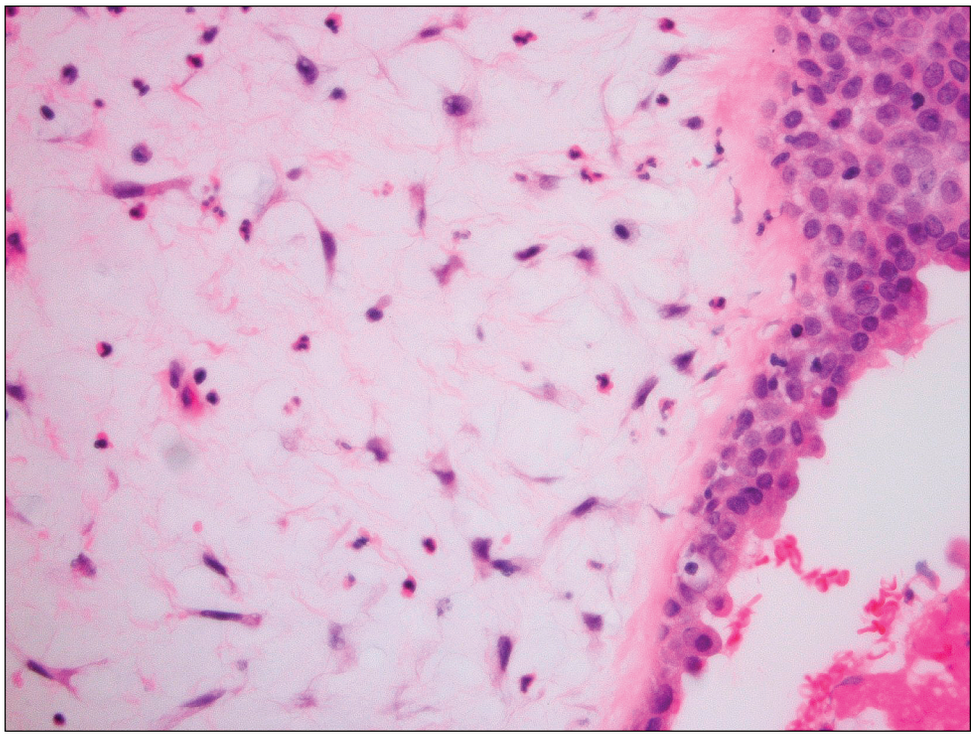
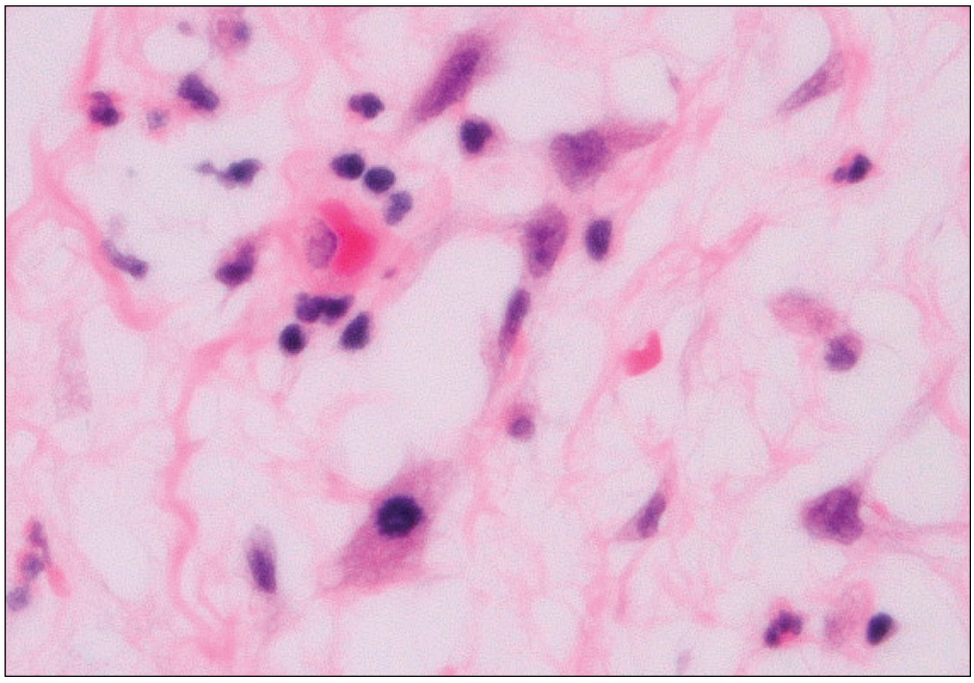


Figure 1.7 Antrochoanal polyps may become quite large, clinically mimicking neoplasia. Top: this antrochoanal polyp is seen behind the uvula (curved arrow) (*Courtesy of Dr. Richard V. Smith*). Bottom: at low power we see a polypoid growth with neovascularization and a lymphoplasmacytic infiltrate with Russell bodies (inset). Seromucinous glands are not present.



Figures 1.8 Antrochoanal polyp: Scattered fibroblasts are present.



Figures 1.9 Some of these fibroblasts appear atypical and mitotically active.