Of the inflammatory conditions affecting the head and neck, the most commonly observed on MRI is sinusitis. The respective sagittal and axial T1 and T2WI of Figs. 56.1A,B demonstrate a band of low to moderate and high SI, respectively, surrounding the periphery of the maxillary sinuses, bilaterally. This finding correlates with mucosal thickening, which alone does not indicate the presence of a sinus infection. In this case, however, the (B) bilateral dependent areas of high SI fluid with resulting air-fluid levels and associated mucosal thickening clinch the diagnosis of sinusitis. Sagittal images, as in Fig. 56.1A, can be used to confirm that the fluid is dependant, aiding in differentiation from a retention cyst. The variance in MRI SI characteristics of sinusoidal fluid is described in Chapter 20. Contrast administration helps to distinguish the enhancing, inflamed mucoperiosteum in acute sinusitis from the nonenhancing contents of a retention cyst, mucocele, or retained secretions. Mucoceles—most commonly occurring in the frontal sinuses—are benign, slow-growing, cystic, expansile masses that develop secondary to obstruction of the sinus ostium. The SI of mucoceles also vary directly with protein content such that especially proteinaceous lesions mimic the high SI appearance of hemorrhage (more frequently seen in coagulopathy or the setting of trauma) on T1WI. Desiccation over time results in a lower SI on both T1WI and T2WI. The SI evolution of sinusoidal blood products is similar to that described for intraparenchymal blood in Chapter 8, although a delay in course may be observed due to overall poor oxygenation. Sinusitis associated with Wegener's granulomatosis is
identifiable by osseous destruction out of proportion to mucosal involvement. In an immunocompromised patient or one in which a sinus infection has not responded to antibiotic therapy, a superimposed fungal infection must be considered. The signal intensity of secretions within the involved sinus is variable, although fungal elements may lead to low SI on T2WI. These organisms also frequently invade sinusoidal emissary veins to extend intracranially or into the cavernous sinus via the cortical veins or dural venous sinuses, respectively. Enhancement or loss of the normal signal void within the dural venous sinus is an ominous finding. Orbital extension may also occur, initially visualized as edematous SI changes within the eyelid. Progression of orbital cellulitis results in chemosis, formation of subperiosteal phlegmon, then abscess, and finally infiltration of the peri- and retroorbital fat. Idiopathic orbital inflammatory disease (pseudotumor) is a similarly appearing inflammatory condition, marked by a poorly margined enhancing soft tissue mass within any area of the orbit, and is a diagnosis of exclusion.

Other infections within the head and neck take a multitude of forms. Although seldom obtained for acute otitis media or mastoiditis, MRI readily demonstrates fluid within the middle ear or mastoid air cells. Likewise cholesteatomas—associated with chronic middle ear infections—are most commonly evaluated by CT, although MRI allows differentiation of the nonenhancing lesion from any surrounding enhancing, inflammatory granulation tissue. Fat within a cholesterol granuloma, meanwhile, lends it a high SI on T1WI, distinguishing it from the two previous entities. Within the external auditory canal, malignant otitis externa appears as high SI on T2WI, potentially eroding the undersurface of the temporal bone and extending intracranially. Subsequent parenchymal or dural involvement is best visualized on postcontrast scans. On the other hand, osteomyelitis—which more commonly occurs in the mandible secondary to infected teeth—is more sensitively detected as low SI on precontrast T1WI. Infections may spread to the parapharyngeal space via the petrous bone or tonsils. A peritonsillar abscess is demonstrated, respectively, on the FS T2 and contrast-enhanced FS T1WI of Figs. 56.1C,D. On the former, enlarged, inflamed lymph nodes and tonsillar tissue appear distinct from muscle as high SI. Encasement of the internal carotid artery—appearing as a dark flow void—constitutes a surgical emergency, whereas compromise and leftward shift of the nasopharyngeal airway are also present. The illustrated abscess also characteristically demonstrates a nonenhancing, necrotic center encased by a large region of peripheral enhancement (Fig. 56.1D). As opposed to postcontrast scans, precontrast T1WI are best obtained without FS to allow for identification of normal fat planes and their destruction. Abscesses within the salivary glands appear similar, mimicking malignancy in this area; symmetric, bilateral gland enlargement suggests inflammation related to autoimmunity. Ductal calculi—a common cause of sialadenitis—may be seen as signal voids (due to dense calcification), but are more reliably imaged by CT or sialography. MR sialography may, however, eventually replace the latter for evaluation of chronic sialadenitis, allowing noninvasive assessment of the ductal system and gland parenchyma. Finally, causes of inflammatory thyroiditis are not reliably distinguished on MRI, although periglandular extension of hyperintensity on T2WI distinguishes an infection from autoimmune entities like Graves disease. The latter may appear concomitant with extracocular myositis, which demonstrates diffuse muscular enlargement and occasional optic nerve compression on MRI. The fibrosis of Reidel thyroiditis correlates with low SI on T1WI and T2WI, while Hashimoto thyroiditis appears as prominent areas of high SI separated by fibrotic areas of low SI on T2WI.