Abstract: The prevalence of heterotopic gastric mucosa (HGM) in the cervical esophagus is frequently underestimated. Tiny microscopic foci have to be distinguished from a macroscopically visible patch, also called "inlet patch." Symptoms as well as morphologic changes associated with HGM are regarded as a result of the damaging effect of acid, produced by parietal cells in the mostly fundic type of HGM. We herein review the literature and propose a new clinicopathologic classification of esophageal HGM: Most of the carriers of esophageal HGM are asymptomatic (HGM I). Some individuals with HGM in the esophagus complain of dysphagia, odynophagia, or "extraesophageal manifestations" (hoarseness and coughing), without further morphologic findings (HGM II). Still fewer patients are symptomatic due to morphologic changes, i.e., esophageal strictures, webs, or esophagotracheal fistula (HGM III). Malignant transformation via dysplasia (intraepithelial neoplasia, HGM IV) to cervical esophageal adenocarcinoma (HGM V) is exceedingly rare (only 24 reported cases). In contrast to Barrett's esophagus, HGM should not be regarded as a precancerous lesion. Symptoms are more likely to occur in patients with inlet patch, whereas malignant transformation and adenocarcinogenesis can also occur in microscopic HGM foci. Asymptomatic HGM requires neither specific therapy nor endoscopic
surveillance. Only in symptomatic cases treatment, i.e., dilatation for (benign) strictures or acid suppression for reflux symptoms, can be recommended. Patients with low-grade dysplasia in HGM might be candidates for surveillance strategies, whereas in cases of high-grade dysplasia and invasive adenocarcinoma oncological treatment strategies must be employed.