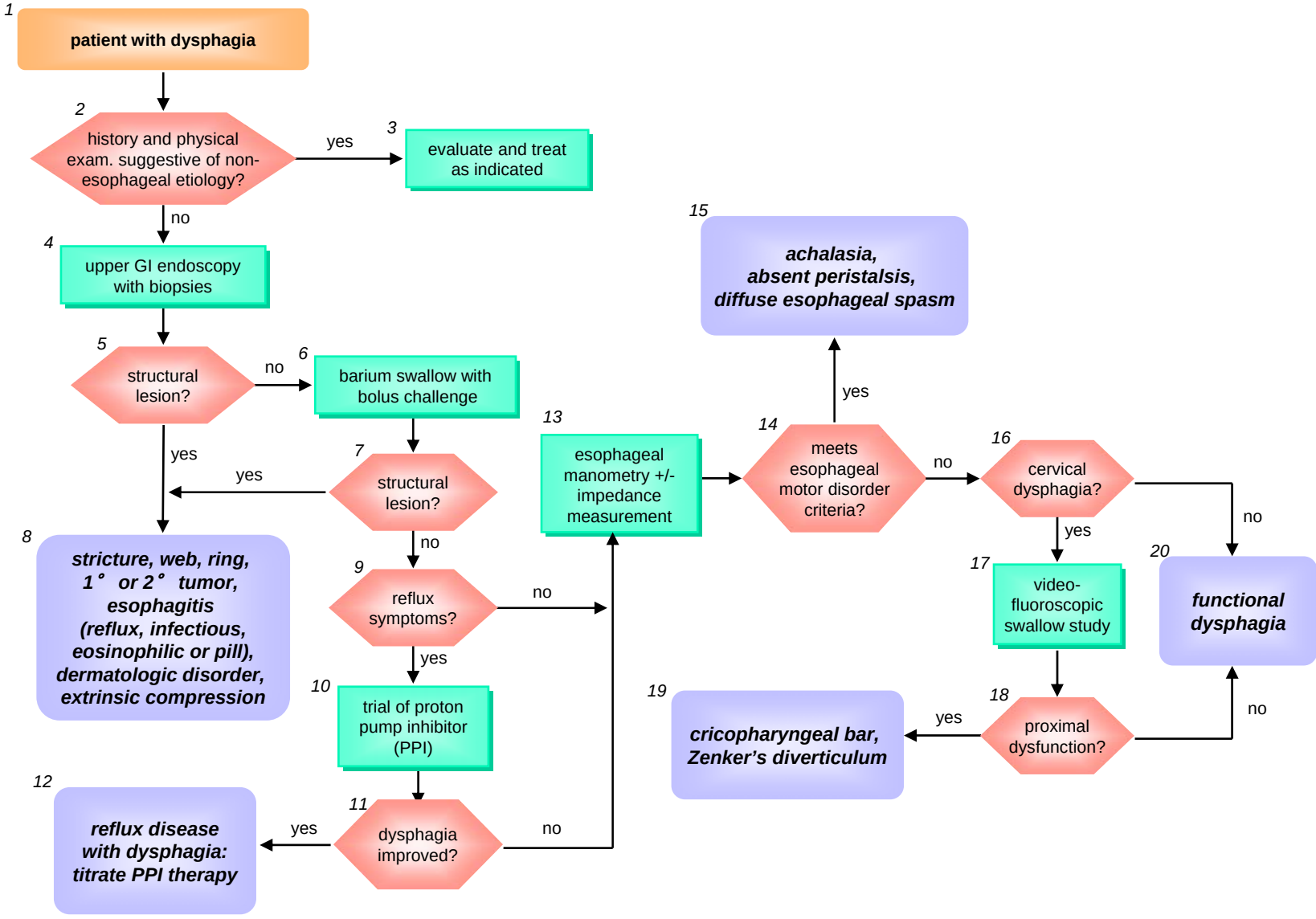
Figure 3: Dysphagia



Dysphagia

Case history

A 44-year bus driver is referred to a gastroenterologist by his primary care physician because of dysphagia for solid food and liquids (Box 1, Fig 3). The symptoms had begun about a year ago. They were intermittent and mild initially, but for the past few weeks bolus hold-up at the midthoracic level is perceived with almost all meals. He has no chest pain or odynophagia. There are no features of oropharyngeal dysphagia, and physical examination for non-esophageal causes of dysphagia is negative (Box 2). His weight has remained constant at 92 kg. The patient experiences heartburn once a week on average. A brief therapeutic trial with an H_2 receptor antagonist, initiated by his primary care physician, eliminated his heartburn but had not resulted in improvement of the dysphagia. His medical history is otherwise unremarkable, and he does not take any drugs. There is no family history of gastrointestinal disease.

Upper GI endoscopy is performed (Box 4) and excludes macroscopic esophagitis or any organic lesion causing esophageal obstruction (Box 5), and microscopic examination of biopsies taken from the distal as well as the proximal esophagus shows that there is no evidence of eosinophilic esophagitis or other histologic abnormality (Box 8). A barium swallow with marshmallow bolus challenge (Box 6) reveals no structural lesion and no impairment of transit through the esophagus (Box 7). Because of the presence of the mild reflux symptoms (Box 9), a trial of PPI, omeprazole 40mg twice daily, is initiated (Box 10). This does not result in improvement of the patient's dysphagia (Box 11).

The gastroenterologist then arranges a manometric study of the esophagus (Box 13). This shows normal esophageal peristalsis, normal LES pressure and normal LES relaxation upon swallowing (Box 14). Concomitant impedance monitoring confirms complete bolus transit with nine of ten swallows, findings within the range of normal. The patient again denies any cervical symptoms (Box 16). A diagnosis of ***functional dysphagia*** is made (Box 20).

Figure legend

1. Dysphagia should be characterized as occurring with only solid food, suggesting a structural abnormality, or both solids and liquids, suggesting a motility abnormality, and whether localized as proximal or distal. The associated symptom of odynophagia is also important as this is highly suggestive of esophageal ulceration.

2. History and physical examination should explore for non-esophageal causes of dysphagia: neck masses, goiter, or neurological findings supportive of oropharyngeal dysphagia.

3. Detection of a non-esophageal condition associated with dysphagia would conclude the evaluation for functional dysphagia.

4. Biopsies should be obtained at the time of endoscopy regardless of visual abnormalities to evaluate for eosinophilic esophagitis; 5 mucosal biopsies should be obtained (12-13). Although histological criteria for esophagitis may also be detected (basal cell hyperplasia, rete pegs extending toward surface) these findings lack specificity for GERD (14).

5. Detection of a structural lesion would conclude the evaluation for functional dysphagia.

6. Barium swallow with solid bolus challenge (barium tablet or barium impregnated marshmallow) is useful in detecting obstructive lesions such as a subtle distal esophageal ring. In this application, this exam has superior sensitivity to upper GI endoscopy (30).

7. Detection of a structural lesion would conclude the evaluation for functional dysphagia.

8. Dysphagia is a common symptom of a multitude of inflammatory and structural esophageal disorders, the detection of which would exclude functional dysphagia (31-33).

9. Concomitant symptoms of heartburn or regurgitation suggest that GERD may be the cause of dysphagia.

10. When used as a therapeutic trial in dysphagia PPIs are usually dosed in a twice daily regimen for at least two weeks (34).

11. Resolution of dysphagia with PPI therapy would imply that the dysphagia was a manifestation of reflux disease and exclude a diagnosis of functional dysphagia.

12. As with all patients, once a satisfactory treatment response has been established, the dose of PPI should then be reduced to the minimal dose still associated with a satisfactory treatment response.

13. If no structural abnormality is found, manometry is indicated. It may be preferable to obtain a high-resolution manometry (esophageal pressure topography) study if available because of a greater sensitivity in the diagnosis of achalasia and other motility disorders (35-36).). If available, concurrent impedance-based assessment of esophageal transit may provide additional information regarding the completeness of bolus transit in the esophagus.

14. The Rome III criteria stipulate that histopathology-based disorders, DES and achalasia preclude the diagnosis of functional dysphagia. Achalasia is defined by absent peristalsis and impaired deglutitive LES relaxation; DES by ≥20% of test swallows exhibiting simultaneous or spastic contractions in the distal esophagus (25).

15. We propose that in addition to DES and achalasia, absent or severely disrupted peristalsis should also lead to exclusion of the diagnosis of functional dysphagia.

16. High (cervical, oropharyngeal) dysphagia is reported by about 30% of individuals with distal disease. However, once distal disease has been adequately excluded the suggestion of any evidence for cervical dysphagia should prompt evaluation for proximal esophageal dysfunction.

17. A videofluoroscopic swallowing study allows for the detailed examination of the swallow mechanism including the opening characteristics of the upper esophageal sphincter which is often a blind spot at

endoscopy or barium swallow examination because of lack of adequate distention (37). Note that if the initial barium study (Box 6) included videofluoroscopy as is done in some institutions, this step can be omitted.

18. The finding of proximal esophageal dysfunction would end the evaluation for functional dysphagia.

19. A cricopharyngeal bar is caused by fibrous degeneration at the upper esophageal sphincter with resultant restricted opening and can be accepted as the cause of dysphagia once other pathology is excluded (38). This can also lead to the formation of Zenker's diverticulum, the mouth of which is located just proximal to the cricopharyngeus.

20. **Rome III diagnostic criteria for functional dysphagia are: 1) sense of solid and/or liquid foods sticking, lodging, or passing abnormally through the esophagus, and 2) absence of evidence that gastroesophageal reflux is the cause of the symptom, and 3) absence of histopathology-based esophageal motility disorders, and 4) criteria fulfilled for the last 3 months with symptom onset at least 6 months prior to diagnosis (26).**