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# Clinical Impact of High-Resolution Manometry and Impedance-pH Monitoring

## 2

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### 2.1 Introduction

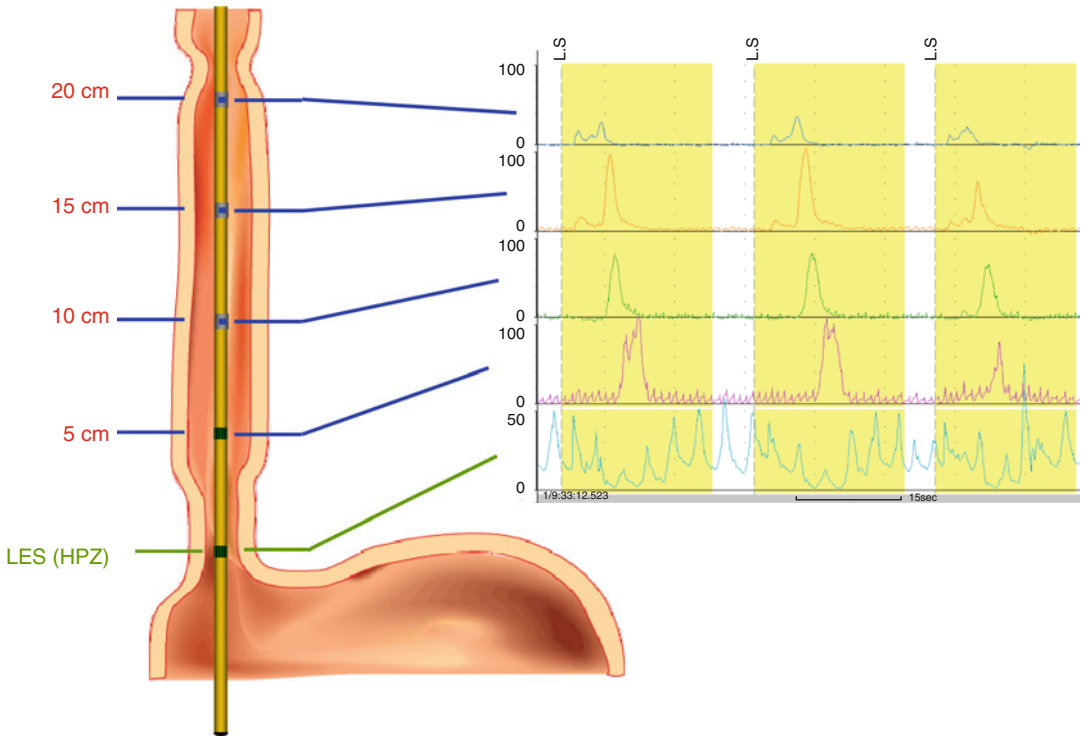
Deglutition is an important physiologic process ensuring our ability to ingest food and liquids. Under normal conditions we swallow approximately 600–2,500 times a day without effort or even noticing the complexity of this process [1]. After the oral and pharyngeal phase of deglutition the bolus is transported through the esophagus into the stomach by involuntary neuromuscular reflexes (i.e. the process of esophageal peristalsis) followed by the relaxation of the lower esophageal sphincter. Impaired swallowing (i.e. dysphagia) causes discomfort and limits our daily activity. Patients with esophageal symptoms (such as dysphagia and/or retrosternal chest pain) seeking medical attention should be referred to gastrointestinal (GI) specialists for detailed investigations. After having excluded structural lesions by endoscopy or barium esophagogram patients should undergo esophageal function testing to investigate esophageal motility abnormalities as a cause of their symptoms.

Heartburn and regurgitation are typical gastroesophageal reflux disease (GERD) symptoms. Current estimates suggest that approximate 40 % of the adult US population experience these symptoms at least once a week, making GERD the most common reason for outpatient GI consultation [2]. Patients with GERD symptoms are often treated with proton pump inhibitors (PPIs) by primary care physicians and those with severe, alarm or persistent symptoms are referred for further testing to GI specialists. An upper GI endoscopy performed to evaluate esophageal erosions as the hallmark of GERD finds erosive esophagitis in only 30 % of patients [3]. Patients with esophageal symptoms and normal esophageal mucosa are referred for esophageal reflux monitoring in order to assess esophageal acid exposure and the relationship between symptoms and gastroesophageal reflux episodes.

Over the years esophageal manometry and pH-monitoring have come to be regarded as the gold standards for testing esophageal motility and quantifying gastroesophageal reflux respectively. Recent technical and computational developments now make it possible to generate detailed pressure maps of the esophagus using high-resolution manometry and to detect gastroesophageal reflux independently of its acidic contents using impedance-pH monitoring. In the present chapter we review the use of high-resolution manometry (HRM) and multichannel intraluminal impedance-pH (MII-pH) monitoring in clinical practice.

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**Fig. 2.1** Conventional esophageal manometry using five pressure sensors located in the lower esophageal sphincter (LES) high pressure zone (HPZ) and esophageal body 5, 10, 15 and 20 cm above the LES

## 2.2 Esophageal Motility Testing Using High-Resolution Manometry

Esophageal manometry is used in clinical practice to quantify contraction amplitude, peristaltic velocity and sphincter relaxation (both upper and lower esophageal sphincter) during deglutition. Manometry systems consist of a catheter with built-in (solid state) or external pressure transducers (pressure being transmitted from esophagus to sensor by water-perfused catheter), a pressure recorder and a computer with dedicated software for analysis. Esophageal manometry is performed under local anesthesia after the patient has fasted for 4–6 h. The probe is inserted and after localizing the lower esophageal sphincter (LES) and measuring the resting pressure of the LES 10 swallows are performed 20–30 s apart in order to prevent deglutitive inhibition.

In conventional manometry it is possible to analyze the upper esophageal sphincter pressure, the contraction amplitude duration and contraction onset velocity. In conventional esophageal manometry normal peristalsis is achieved if the contraction amplitude exceeds 30 mmHg at two pressure sites in the distal esophagus spaced 5 cm apart and if the contraction onset velocity between these two sites is less than 8 cm/s. A contraction amplitude below 30 mmHg at one site is considered an ineffective contraction and a contraction onset velocity more than 8 cm/s between the (5-cm apart) sites is considered a simultaneous contraction if the contraction amplitude exceeds 30 mmHg. The normal value of the lower esophageal sphincter ranges from 10 to 45 mmHg with a residual pressure <8 mmHg in conventional esophageal manometry [4] (Fig. 2.1).

Since the introduction of esophageal manometry into clinical practice more than 50 years ago

important developments have taken place. Recent developments include high-resolution manometry and high-resolution impedance manometry. High-resolution manometry (HRM) uses closely spaced pressure sensors (usually 1–1.5 cm apart) with 20–36 channels per catheter. The software interpolates values between sensors providing a virtually continuous pressure map of the esophagus and its sphincters. The line plots are replaced by esophageal pressure topography graphs with time on the x-axis, distance of the pressure sensors from the nose on the y-axis and pressure magnitude on the z-axis, scaled by colour intensity. In the colour contour plot isobaric contours are displayed as a continuous line of a value of equal pressure on a pressure topography plot (Fig. 2.2). HRM visualizes the pressure topography of the esophagus in a way that is easy to understand (even for non-professionals after a short training), reliable and reproducible [5]. By the two-dimensional picturing of the measurements it is simple to identify normal contractions, primary vs. secondary peristalsis, hiatal hernias and the contractile features of the esophagus and even other high-pressure zones (i.e. an accentuated aortic arch or an augmented left atrium).

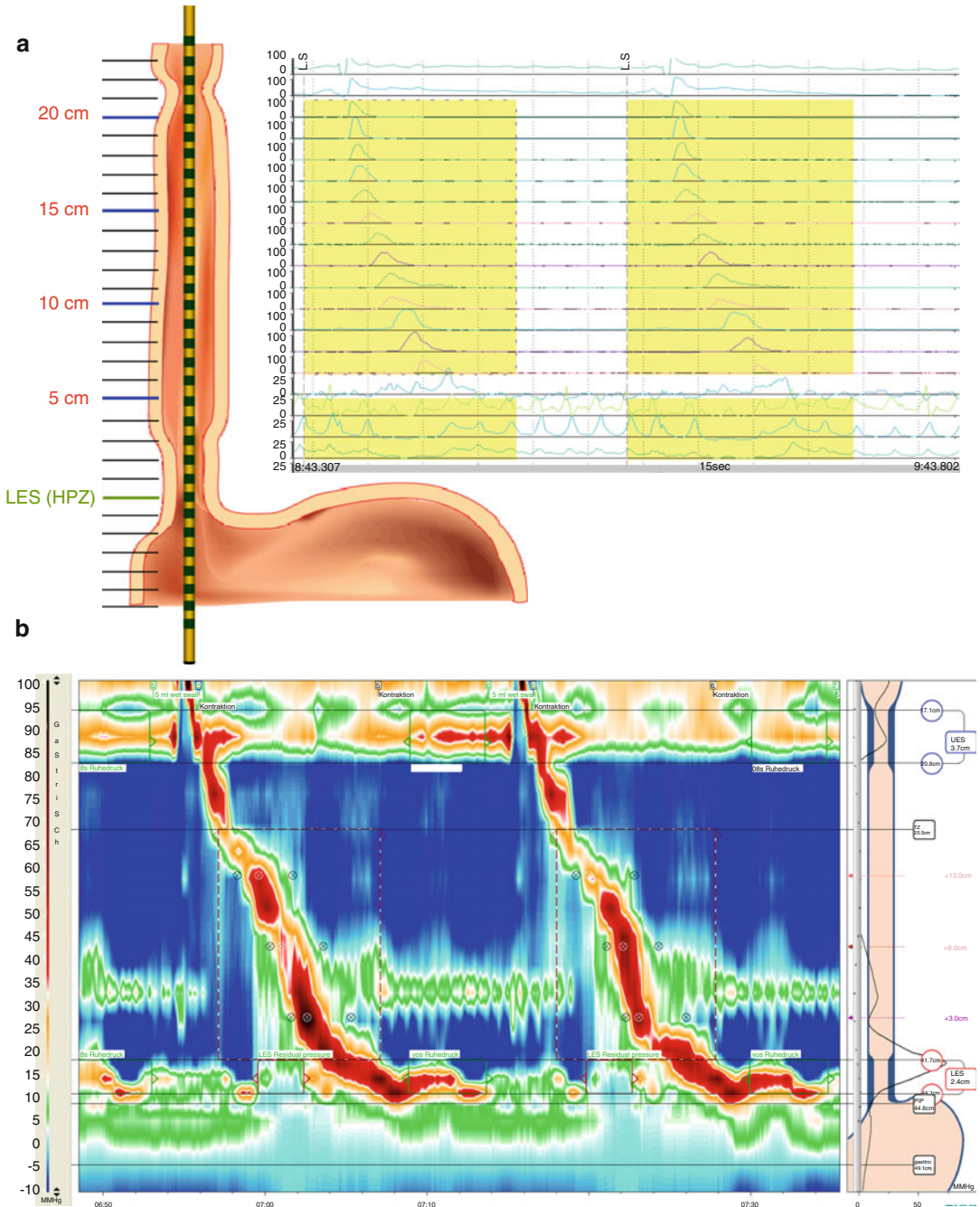
The field of applications ranges from the investigation of non-cardiac chest pain and of non-obstructive dysphagia, preoperative evaluations before bariatric or anti-reflux surgery and in advance of an impedance-pH manometry with a view to localizing the lower esophageal sphincter for the positioning of the MII-pH impedance probe.

In the characterization of the esophago-gastric junction (EGJ) pressure morphology it is important to remember the components of the pressure profile of the EGJ: the lower esophageal sphincter (LES) and the crural diaphragm (CD). In the case of a hiatal hernia with a double-pressure zone the fixed high-pressure zone independent of the respiration can be identified as the LES, and the high-pressure zone changing with respiration as the CD. The respiratory inversion point is the junction between intra-abdominal and intra-mediastinal pressure, defined as the position where the inspiratory EGJ pressure becomes less than the expiratory EGJ pressure. EGJ relaxation is mea-

sured in the deglutitive relaxation window, starting with the upper esophageal sphincter (UES) relaxation until the arrival of the peristaltic contraction arrives at the EGJ. Ghosh et al. proposed in 2007 after a quantitative analysis of 400 patients and 75 controls the integrated relaxation pressure computed over 4 s (IRP-4 s) as a parameter to quantify deglutitive LES relaxation [6].

The report of the analysis of the distal segment contractility contains the peristalsis, which is composed of a proximal segment with striated muscles and a distal segment with another three sub-segments with smooth muscles, interadjacent the transition zone. Contractile front velocity (CFV) measures the velocity of the conduction of the peristaltic contraction in cm/s at the isobaric contour of 30 mmHg beginning at the transition zone until the proximal EGJ with values up to 8.5 cm/s as the norm. The distal contractile integral (DCI) quantifies the esophageal peristalsis by integrating pressure, duration and distance of the peristaltic wave in the distal esophagus. Normal DCI values are below 5,000 mmHg · s · cm (Table 2.1).

The technical developments of HRM were followed by a revised classification of motility abnormalities. The Chicago classification integrates the morphology and pressure characteristics of the EGJ, EGJ relaxation, CFV and DCI of the peristalsis so making it possible to classify nutcracker esophagus subtypes, esophageal spasm and three subtypes of achalasia [7, 8] (Fig. 2.3). At first the EGJ has to be characterized by morphology and deglutitive EGJ relaxation (normal integrated relaxation pressure (IRP) < 15 mmHg). In the case of normal deglutitive EGJ relaxation an isobaric contour at 30 mmHg is drawn and the integrity of this isobaric area analyzed. In patients with continuous isobaric 30 mmHg contour the DCI is used to define hypertensive contractions (DCI > 5,000 mmHg · s · cm; nutcracker esophagus) or hypercontractile esophagus (DCI > 8,000 mmHg · s · cm; jackhammer esophagus) or normal esophageal peristalsis. In the case of breaks in the isobaric 30 mmHg contour Bulsiewicz et al. proposed a division into large (>2 cm) or small (<2 cm) breaks, the first



**Fig. 2.2** High-resolution esophageal manometry. Esophageal pressure data is collected from sensors placed at a distance of 1–1.5 cm from each other (a). In pressure topography the pharyngeal contraction, upper esophageal sphincter relaxation (normal 300–500 ms), proximal

esophageal peristalsis, transition zone (TZ), distal esophageal peristalsis and lower esophageal sphincter (LES) relaxation (normal 6–10 s) can be quantified for each swallow (b)

**Table 2.1** High resolution manometry (HRM) diagnostic criteria for esophageal motility abnormalities

Normal
Defect in the 30 mmHg isobaric contour in the distal segment of the esophagus <3 cm
CFV < 8 cm s <sup>-1</sup> in >90 % of swallows, IBP < 15 mmHg, DCI < 5,000 mmHg · s · cm
Normal EGJ relaxation (mean IRP < 15 mmHg) and normal IBP (10–35 mmHg)
Hypotensive peristalsis
>3 cm defects in the 30 mmHg isobaric contour of the distal esophageal segment
Intermittent: > 30 % swallows with hypotensive or absent peristalsis
Frequent: > 70 % swallows with hypotensive or absent peristalsis
Absent peristalsis
No propagating contractile wavefront in 100 % of swallows
No isobaric pressurization greater than 30 mmHg isobaric contour
Hypertensive peristalsis
Normal peristaltic velocity < 8 cm/s in >80 % of swallows
Mean DCI > 5,000 mmHg · s · cm or LES after contraction > 180 mmHg
Normal CVF
Spastic nutcracker
Normal CVF, Mean DCI > 8,000 mmHg · s · cm
Esophageal spasm (rapidly propagated contractile wavefront)
Spasm (CVF > 8 cm s <sup>-1</sup> ) in >20 % of swallows
<i>Segmental spasm</i> : Spasm limited to one of the three segments of the distal esophagus
<i>Diffuse spasm</i> : Spasm involving both upper segments of the distal esophagus
Peristaltic dysfunction
Mild: 3–6 swallows with failed peristalsis or a >2 cm defect in the 30 mmHg isobaric contour of the distal esophageal peristalsis
Severe: ≥7 swallows with either failed peristalsis or a >2 cm defect in the 30 mmHg isobaric contour of distal esophageal peristalsis
Aperistalsis: Contractile pressure < 30 mmHg throughout mid-distal esophagus in all swallows (Scleroderma pattern: aperistalsis with LES pressure < 10 mmHg)
Achalasia
Impaired deglutitive EGJ relaxation and/or opening
Elevation of intra-esophageal bolus pressure due to resistance to flow at EGJ
Classic: Mean IRP > 15 mmHg, aperistalsis
Achalasia with compression: Mean IRP > 15 mmHg, absent peristalsis with compartmentalized pressurizations
Spastic achalasia: Mean IRP > 15 mmHg, absent peristalsis and spasms (CFV > 8 cm s <sup>-1</sup> )
Abnormal LES tone
Hypotensive: 10 s mean < 10 mmHg with normal peristaltic function
Hypertensive: 10 s mean > 35 mmHg with normal peristaltic function and EGJ relaxation

Adapted from [8]

CFV contractile front velocity, DCI distal contractile index, EGJ esophago-gastric junction, IBP Intrabolus pressure, IRP integrated relaxation pressure, LES lower esophageal sphincter

ones being more often associated with impaired esophageal bolus clearance [9].

### 2.2.1 Achalasia

Achalasia is a primary esophageal motor disease caused by destruction of the inhibitory neurons in

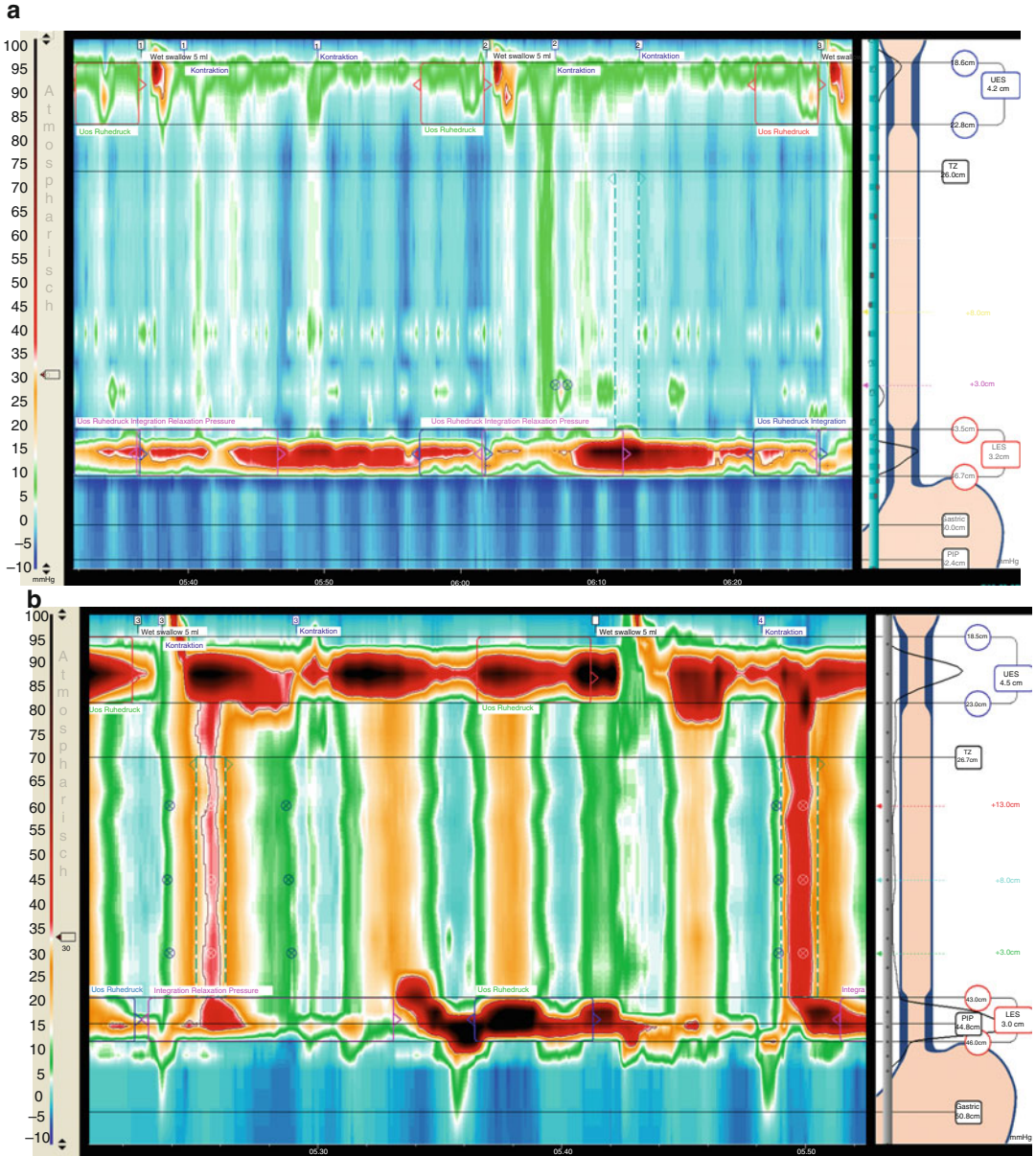
the Auerbach plexus of the entire esophagus resulting in a complete lack of peristalsis. It causes often dysphagia, regurgitation with risk of aspiration, chest pain and in the course of the disease weight loss. It is defined as aperistalsis and impaired deglutitive EGJ relaxation.

High-resolution manometry (HRM) brought novel criteria and sub-classification criteria for



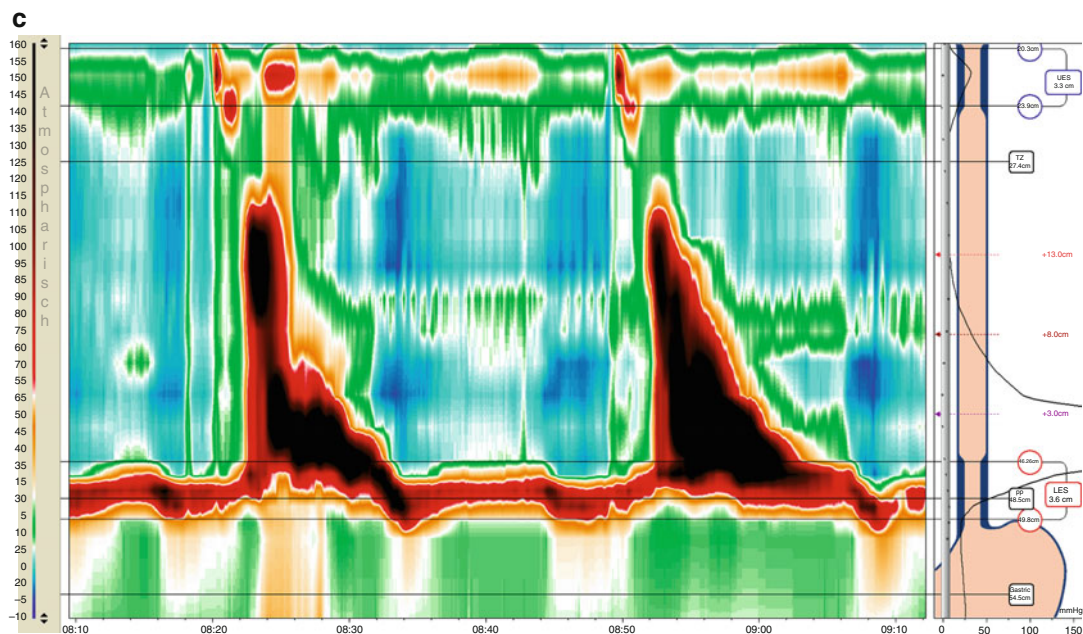
achalasia. Pandolfino et al. found that an elevated integrated residual pressure (IRP) > 15 mmHg in the absence of esophageal peristalsis had a sensitivity of 98 % and a specificity of 96 % to diagnose achalasia [10]. With HRM the sensitivity of the detection of achalasia improved, but more

important a new classification of clinically relevant subtypes of achalasia was developed. Classic type I achalasia includes patients with elevated IRP and absent esophageal peristalsis. Type II achalasia (with pressurization) is characterized by elevated IRP and absent peristalsis but elevated



**Fig. 2.3** Subtypes of achalasia: (a) type I classic achalasia – impaired EGJ relaxation and absent esophageal peristalsis; (b) type II with pressurization – impaired EGJ relaxation and non-peristaltic pressure changes exceeding

30 mmHg; (c) type III with spasms – more than 80 % spastic contractions in the distal esophagus with impaired EGJ relaxation. *EGJ* esophago-gastric junction



**Fig. 2.3** (continued)

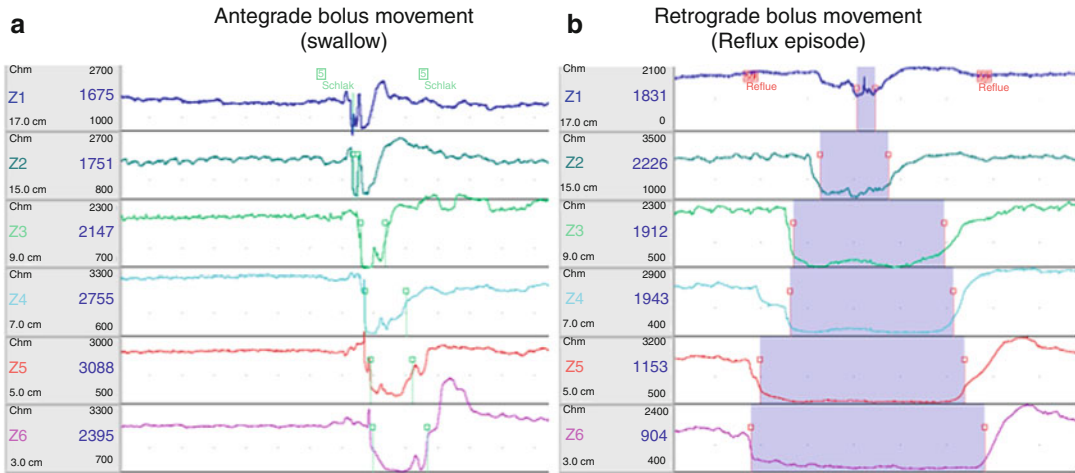
panesophageal pressure ( $>30$  mmHg). This corresponds to the previously defined *vigorous achalasia* by Spechler and Castell. Achalasia with compression shows a panesophageal pressurization and has the best outcome with a positive treatment response of 100 % to Heller myotomy and 91 % to pneumatic dilatation reported in 49 newly diagnosed type II achalasia patients. The least frequent pattern is type III achalasia (with spasms) characterized by impaired EGJ relaxation (i.e. elevated IRP) and with spastic contraction in the distal esophagus. Outcome studies suggest that this type (type III achalasia) has the overall poorest response to therapy (29 % overall).

### 2.3 Gastroesophageal Reflux Testing Using Combined Impedance-pH Monitoring

With each deglutition during meals we ingest small amounts of air that accumulate in the stomach and are subsequently released by belching. This process involves reflex, transient relaxations of the lower esophageal sphincter (tLESRs) induced by distension of gastric fundus. Depending

on the filling of the stomach, occasionally small amounts of liquids (or mixture of liquid and gas) can reflux back from the stomach into the esophagus. As long as these episodes (in normal individuals up to 50 episodes per day) are not associated with symptoms or do not cause esophageal lesions by prolonged esophageal acid exposure gastroesophageal reflux remains undetected. Increased body fat (i.e. increased body mass index), large/cal-rich meals and modern lifestyle (with large evening meals within a couple of hours of going to bed) are some of the factors that lead to an increased prevalence of gastroesophageal disease (GERD). In the mid 1980s potent acid suppressive therapy using proton pump inhibitors (PPIs) provided important relief for a disease that affects 15–20 % of the adult population. Therapy with PPIs had an important impact not only on the natural course and therapy of GERD but also brought new challenges for the diagnosis of GERD.

Esophageal pH-monitoring uses the low pH of the gastric content to identify gastroesophageal reflux episodes. This method, introduced in the early 1970s, soon became the gold standard to diagnose GERD, in particular in patients with



**Fig. 2.4** Bolus presence and movement detected by multichannel intraluminal impedance. Liquid bolus entry is identified by a rapid decline in impedance, bolus exit by the return of impedance to baseline. Serial impedance measurement segments on a catheter can be used to determine the direction of bolus movement: swallows (**a**) are

characterized by declines in impedance starting in the proximal and over time moving to the distal esophagus. Reflux episodes (**b**) are identified as declines in impedance starting distally and over time moving to the proximal esophagus

normal esophageal mucosa. Quantifying gastroesophageal reflux by monitoring intraluminal pH becomes difficult if gastric acidity is suppressed by PPIs, a common first-line scenario used for patients with typical (heartburn and/or regurgitation) and atypical (chest pain, hoarseness, epigastric burning, etc.) GERD symptoms. Among several methods attempting to overcome these limitations, multichannel intraluminal impedance monitoring has become the method of choice to identify gastroesophageal reflux episodes.

Multichannel intraluminal impedance (MII) measures resistance to an alternating electrical current between two electrodes placed within the esophageal lumen. In an empty esophagus the electrical circuit is closed by the esophageal mucosa that provides a resistance of 1,500–2,000  $\Omega$ . Liquids arriving in an impedance-measuring segment produce a rapid decline in impedance by improving electrical conductivity through the ionic charges within the bolus. Lining up multiple impedance measuring segments along a catheter allows to detect not only bolus presence but also the direction of bolus movement. Changes in intraluminal impedance progressing over time from proximal to distal indicate an aboral,

antegrade bolus movement as seen during swallowing while changes progressing over time from distal to proximal indicate an oral, retrograde bolus movement as seen during reflux episodes (Fig. 2.4). Furthermore impedance measurements provide information on the physical properties of the refluxate. Pure liquid reflux episodes produce only declines in impedance (progressing from distal to proximal) while pure gas reflux episodes (belching) produce only a rise in impedance. The combination of increases and declines in intraluminal impedance indicate mixed (liquid–gas or gas–liquid) content of the refluxate. Combining MII with pH (MII-pH) makes it possible to detect reflux episodes of all types and classify them into acid or non-acid by their nadir pH (acid – nadir pH < 4; non-acid – nadir pH > 4).

Over the years MII-pH data recorded in healthy volunteers have served as a basis for establishing normal values. Two multicenter studies from US-Belgian [11] and French-Belgian [12] groups provided normal values for the total number of reflux episodes (acid and non-acid, liquid and mixed), proximal extent of reflux episodes and esophageal acid exposure off PPI therapy. A subsequent study by Zentilin et al. provided



**Table 2.2** Normal values for combined MII-pH monitoring based on 95th percentile data in healthy volunteers not taking acid suppressive therapy

		Impedance-pH monitoring		
		US-Belgian	French-Belgian	Italian
		(N=60)	(N=72)	(N=25)
Esophageal pH data				
% time pH <4	Total	6.7	5.0	4.0
	Upright	9.7	6.2	5.0
	Recumbent	2.1	5.3	3.0
Esophageal MII data				
# reflux episodes	Total	73	75	61
	Acid	55	50	51
	Weakly acid	26	33	38
	Weakly alkaline	1	15	18

MI I multichannel intraluminal impedance

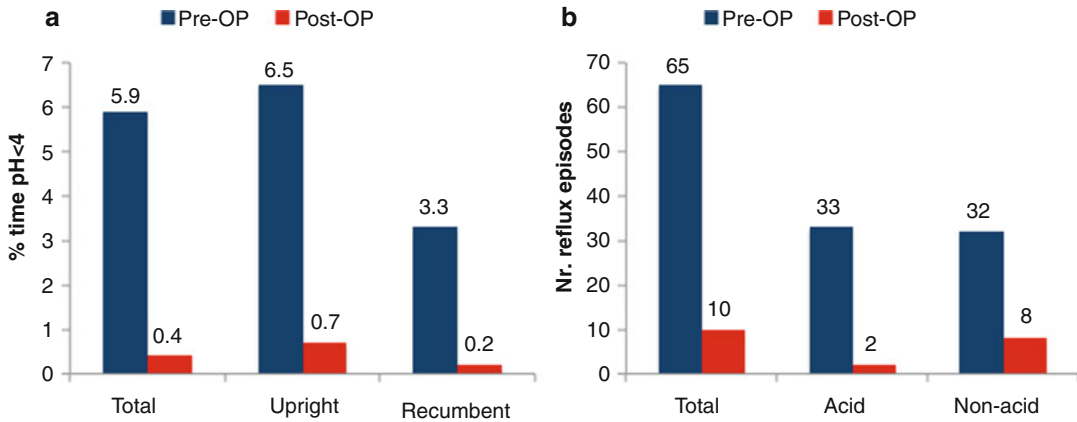
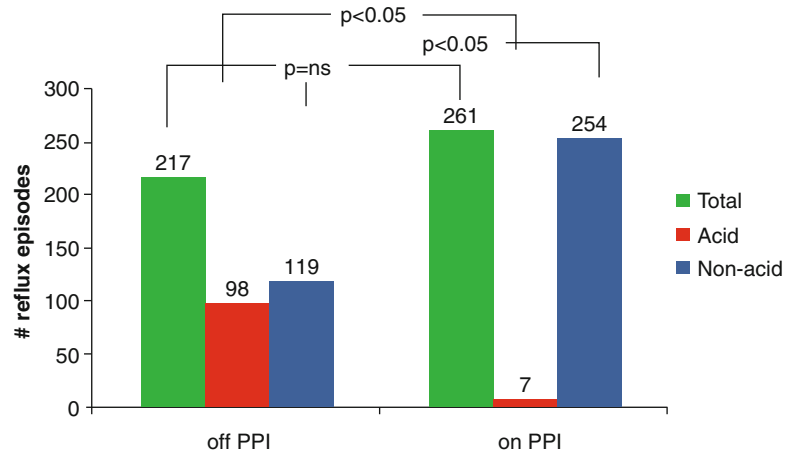
normal values for an Italian population on a Mediterranean diet [13]. Currently available normal values are summarized in Table 2.2. Using these normal values and the ability to detect non-acid reflux Savarino et al. [21] compared MII-pH findings from 150 patients with typical symptoms and normal upper GI endoscopy (nonerosive reflux disease (NERD)) with those from 48 healthy volunteers. The authors found that NERD patients had more reflux episodes (median [25th–75th percentile]) compared with healthy volunteers (total: 46 [26–65] vs. 32 [18–43],  $P<0.05$ ; acid: 29 [14–43] vs. 17 [8.5–31.0],  $P<0.05$ ; and nonacid: 20 [15–27] vs. 18 [13.5–26.0],  $P=NS$ ). Among 87 patients with normal distal esophageal acid exposure 22 (15 %) had a positive symptom association probability (SAP) for acid, 19 (12 %) for nonacid reflux, and 7 (5 %) for both. Classifying patients with symptomatic nonacid reflux as having a hypersensitive esophagus reduced the number of patients with functional heartburn from 65 (43 %) to 39 (26 %). These data suggest that monitoring for nonacid reflux in NERD patients reduces the proportion of patients misclassified as having *functional heartburn*.

Early data on post-prandial reflux documented that acid suppressive therapy with PPI reduces distal esophageal acid exposure and number of acid reflux episodes but does not reduce the total number of reflux episodes (Fig. 2.5) [14]. Subsequent studies evaluating acid and non-acid reflux episodes over 24 h noted the same phenomenon that

PPI therapy increases the pH of the gastroesophageal refluxate without affecting the total number of reflux episodes. Evaluating 30 patients with typical reflux symptoms on and off PPI therapy Hemmink et al. found fewer acid reflux episodes ( $49\pm34$  off PPI vs.  $20\pm25$  on PPI) but a higher number of weakly acidic reflux episodes ( $24\pm17$  off PPI vs.  $48\pm31$  on PPI) in patients on versus off acid suppressive therapy [15]. By documenting ongoing gastroesophageal reflux in patients on acid suppressive therapy these studies provided a sound rationale for looking for non-acid reflux as a cause of persistent symptoms in the presence of acid suppressive therapy.

Multicenter studies from the US and France documented that in 30–45 % of patients on therapy with PPI twice daily esophageal symptoms during 24-h combined MII-pH monitoring were associated with ongoing gastroesophageal reflux [16, 17]. These studies underscore that non-acid reflux episodes are the main type of reflux associated with persistent symptoms on PPI therapy. In a step-wise increase of acid suppressive therapy in GERD patients Becker et al. [18] evaluated patients with ongoing symptoms while on once daily PPI and found abnormal MII-pH findings (abnormal distal esophageal acid exposure or abnormal number of MII-detected reflux episodes) in 39 % of patients. Patients were followed up for at least 3 months after the dose of PPI was increased. Following increased acid suppression the authors found significantly better symptomatic relief in

**Fig. 2.5** Postprandial gastroesophageal reflux episodes on and off PPI therapy. Acid suppressive therapy reduces the number of acid reflux episodes but does not reduce the total number of reflux episodes. This leads to an apparent paradoxical increase in the number of non-acid (weakly acidic/weakly alkaline) reflux episodes. *PPI* proton pump inhibitor, (Adapted from [14])



**Fig. 2.6** Gastroesophageal reflux before and after Nissen-Rossetti fundoplication. The surgical procedure reduces distal esophageal acid exposure expressed as % time

pH<4 (a) and all types of gastroesophageal reflux episodes, both acid and non-acid (b) (Adapted from [19])

patients with abnormal MII-pH results (90 %) compared to patients with normal MII-pH findings (43 %), concluding that combined MII-pH monitoring facilitates a more focused therapeutic approach to patients with PPI-resistant GERD possibly avoiding PPI overuse.

In contrast to acid suppressive therapy, anti-reflux surgery reduces both acid and non-acid gastroesophageal reflux episodes (Fig. 2.6). Del Genio et al. performed MII-pH monitoring in 15 consecutive patients who underwent laparoscopic Nissen-Rossetti fundoplication before and 7 months after the operation [19]. The authors found not only a significant improvement in

esophageal symptoms (heartburn pre-op  $2.3 \pm 0.8$  vs. post-op  $0.2 \pm 0.2$ ; regurgitation pre-op  $1.8 \pm 0.9$  vs. post-op  $0.3 \pm 0.2$ ;  $p < 0.05$ ) and distal esophageal acid exposure (total % time pH<4 pre-op  $5.9 \pm 2.9$  vs. post-op  $0.4 \pm 0.3$ ;  $p < 0.05$ ) but also a significant reduction in the total number of reflux episodes (pre-op  $65.2 \pm 45.4$  vs. post-op  $10.0 \pm 10.0$ ;  $p < 0.01$ ) both acid (pre-op  $33.4 \pm 21.4$  vs. post-op  $1.3 \pm 3.6$ ;  $p < 0.01$ ) and non-acid (pre-op  $31.8 \pm 34.1$  vs. post-op  $8.3 \pm 9.3$ ;  $p < 0.05$ ). Frazzoni et al. documented the superiority of laparoscopic fundoplication over maximal acid suppressive therapy in controlling reflux episodes in patients with persistent symptoms

on acid suppressive therapy [20]. Evaluating 71 patients with persistent heartburn/regurgitation on PPI twice daily the authors identified 40 patients with either an increased number of reflux episodes or positive symptom association on acid suppressive therapy prepared to undergo fundoplication. Anti-reflux surgery further reduced distal esophageal acid exposure (% time pH < 4 on PPI 1.0 (0.3–2.4) vs. post-surgery 0.1 (0.0–0.3);  $p < 0.01$ ), and decreased the number of acid (on PPI 14 (6–25) vs. post-surgery 2 (1–6);  $p < 0.01$ ) and non-acid reflux episodes (on PPI 69 (50–94) vs. post-surgery 21 (12–31);  $p < 0.01$ ).

While these recent data suggest that laparoscopic fundoplication improved gastroesophageal reflux control, other therapeutic options are warranted for patients with persistent symptoms on acid suppressive therapy who are not prepared (or not candidate) for anti-reflux surgery.

## 2.4 Summary

High resolution esophageal manometry is an important development in esophageal function testing. The increased sensor density and novel topographic representation of pressure changes during swallowing provide a comprehensive evaluation of pharyngeal contraction, upper esophageal sphincter relaxation, esophageal body peristalsis and lower esophageal sphincter relaxation for each swallow. Interpretation of esophageal manometry becomes more intuitive, limiting pitfalls due to LES misplacement, double swallowing or secondary peristalsis. The recent sub-classification of achalasia using HRM makes it possible to predict therapeutic outcome.

Impedance-pH monitoring represents a shift in the reflux monitoring paradigm where reflux episodes are detected as the presence of liquid in the esophagus and sub-classified into acid vs. non-acid (or weakly acidic) based on the pH data. Combined MII-pH monitoring has revealed that non-acid reflux is the main culprit preceding symptoms in patients with persistent symptoms on acid suppressive therapy. This information should allow a better selection of patients who would benefit from therapies aimed

at correcting the gastroesophageal barrier such as laparoscopic fundoplication or endoscopic anti-reflux procedures.

### Key Points

- Esophageal manometry is indicated in patients with esophageal symptoms and no structural abnormalities (i.e. normal upper GI endoscopy and/or barium esophagogram)
- High resolution manometry provides a detailed description of the upper/lower esophageal sphincter relaxation and esophageal peristalsis during swallowing
- Sub-classification of achalasia allows to predict outcome following pneumatic dilatation or laparoscopic myotomy
- Impedance-pH monitoring detects gastroesophageal reflux episodes independently of their acid content, allowing detection of acid and non-acid reflux
- Non-acid reflux is responsible for ongoing symptoms in up to 40 % of patients under therapy with proton pump inhibitors
- Acid suppressive therapy reduces the acidity of the gastroesophageal refluxate but does not change the total number of gastroesophageal reflux episodes
- Intervention augmenting the gastroesophageal barrier (i.e. laparoscopic fundoplication) is superior to acid suppressive therapy in controlling acid and non-acid reflux

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