Prevalence and Clinical Significance of Esophagogastric Outflow Obstruction (EGJOO) in Patients with Gastroesophageal Reflux Disease (GERD)

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Background: Gastroesophageal Reflux Disease (GERD) is defined by typical and/or atypical reflux symptoms with abnormal acid exposure, Barrett's esophagus, or erosive esophagitis. Esophagogastric junction outflow obstruction (EGJOO) describes manometrically a lower esophageal sphincter (LES) dysfunction diagnosed during high resolution esophageal manometry (HREM). Chicago Criteria defines EGJOO as the LES's inability to relax appropriately based on Integrated Relaxation Pressure (IRP). Pressure > 15 mmHg indicates dysfunction. Although considered gold standard for diagnosing esophageal motility disorders, Chicago Classification does not consider factors affecting LES function. Clinical significance of EGJOO in patients with GERD is unclear. Methods: Prospectively collected data from 364 patients seen in tertiary referral center were analyzed. GERD-HQRL and RSI questionnaires, endoscopy, CINEesophagram, pH testing and endoscopic functional luminal imaging were performed as clinically indicated, and compared with the final diagnosis of EGJOO after considering anatomic changes and upright swallow measurements during HREM to determine if swallows in the upright position corrected IRP. Patient characteristics were further analyzed. Results: Of the 364 patients evaluated, 147 had confirmed GERD and HREM. Of those, 64 had abnormal HREM (Figure 1). 31/64 (48%) patients had abnormal HREM suggesting EGJOO. In these 31 patients (Table 1), review of the HREM showed that 21 performed upright swallows; 14(67%) had IRP corrected suggesting HREM artifact (Figure 1). The other 7 patients (33%) with upright swallows had unchanged IRP confirming true EGJOO. 4/7 (57%) were classified as "mechanical" EGJOO (prior Nissen fundoplication, hiatal hernia) and 3(44%) were true functional EGJOO. Conclusions: EGJOO is a manometric abnormality diagnosed in confirmed GERD patients evaluated with HREM. Upright swallows normalized the abnormal IRP in two thirds of EGJOO. Based upon these data, pH testing and routine upright swallows during HREM may minimize the misdiagnosis of EGJOO in the setting of confirmed GERD. Prospective validation of this approach is needed.

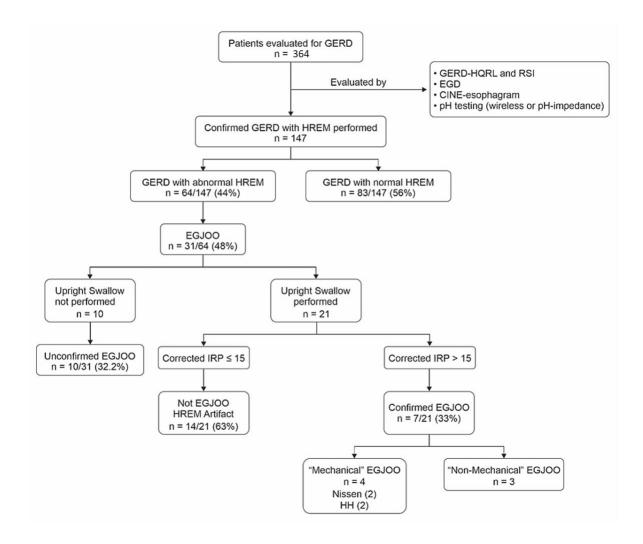


Figure 1: flow diagram of GERD patients diagnosed with EGJOO.

Table 1: Patient Characteristics					
	EGJOO (n=31)				
Age, mean ± SD	55 ± 12.1				
Gender, n (%)					
Male	13 (41.9%)				
Female	18 (58.1%)				
BMI, mean ± SD	28.4 ± 7.4				
IRP basal, median (IQR)	20.6 (6.4)				
On opiates, n (%)	4 (12.9%)				
Hiatal hernia length (cm), n (%)					
1 cm	6 (19.3%)				
2 cm	6 (19.3%)				
>3 cm	2 (6.4%)				
Prior Nissen fundoplication, n (%)	5 (16%)				
HREM findings, n (%)					
EGJOO	20 (64.6%)				
EGJOO + IEM	1 (3.2%)				
EGJOO + Jackhammer esophagus	1 (3.2%)				
EGJOO + HH	9 (29%)				

Table 1: Patient Characteristics

TITLE: PATIENTS WITH INEFFECTIVE ESOPHAGEAL MOTILITY UNDERGOING MAGNETIC SPHINCTER AUGMENTATION DEMONSTRATE OUTCOMES SIMILAR TO PATIENTS WITH NORMAL ESOPHAGEAL MOTILITY

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Introduction: Many patients with GERD have associated dysmotility such as ineffective esophageal motility (IEM) that may affect outcomes of Magnetic sphincter augmentation (MSA). We aimed to evaluate outcomes of MSA in patients with IEM.

Methods: This is a multi-institutional, retrospective study, of patients with IEM, defined by Chicago classification 3, undergoing MSA from 2012-2017. Cases were matched to non-IEM patients on sex, BMI, presence of Barrett's, hiatal hernia size and method of hiatal closure. Dysphagia was defined as a score of >3 on the difficulty swallowing item on the GERD-HRQL survey.

Results: A total of 106 patients with IEM underwent MSA. The IEM group GERD-HRQL score improved from 22 to 4 (p<0.001) and DeMeester score improved from 33 to 8 (p<0.001) at 1 year, similar to the non-IEM group (Table 1).

At 1 year, in the IEM group, 14/17 (82%) patients had resolution of dysphagia; 3/17 (18%) had persistent dysphagia; 9/68 (13%) had new onset dysphagia and 59/68 (87%) never had dysphagia. A similar trend was observed in the non-IEM group (Figure 1).

From 2012-2017, 23 (22%) IEM patients were dilated after MSA, compared to 27 (27%) non-IEM patients (p = 0.33). Seven (7%) IEM vs 6 (6%) non-IEM patients had their device explanted. Compared to the explanted non-IEM patients, the explanted IEM patients had lower preop DCI of 195.5, lower preop dysphagia rates (14% vs 50%), higher preop DeMeester scores 46.3 vs 35.5 and a shorter duration of symptoms, 95 vs 157 months.

Conclusion: MSA in IEM patients demonstrates comparable rates of symptomatic and objective GERD improvement. Resolution of baseline dysphagia and rates of developing new onset

dysphagia were similar. The need for postoperative dilation and explantation were similar. IEM, therefore, should not preclude use of MSA in patients with GERD.

Word count: 292/300 words

	IEM	Non-IEM	p-value
Age, mean (SD)	49.4 (15.4)	53.4 (14.6)	0.06
BMI, mean (SD)	26.5 (4.1)	27 (4.2)	0.39
Duration of symptoms, mean (SD)	120.9 (105.1)	144.9 (113.6)	0.16
Male, n (%)	63 (59.4%)	55 (56.1%)	0.63
Female, n (%)	43 (40.6%)	43 (43.9%)	0.63
Dysphagia, n (%)	18 (18.9%)	23 (24%)	0.39
Preoperative GERD-HRQL score, median (IQR)	22 (14 - 34)	25 (16 - 36)	0.69
Postoperative GERD-HRQL score at 1 year, median (IQR)	4 (2 - 11)	5 (2 - 9)	0.75
Preoperative total DeMeester score, median (IQR)	33 (21.4 - 47.6)	28.65 (18 - 45.9)	0.22
Postoperative total DeMeester score at 1 year, median (IQR)	8.2 (3.2 – 25.2)	7.5 (2.8 – 20.6)	0.86

Table 1. Patient demographics, with pH testing and quality of life outcomes

SD = standard deviation, IQR = Interquartile range

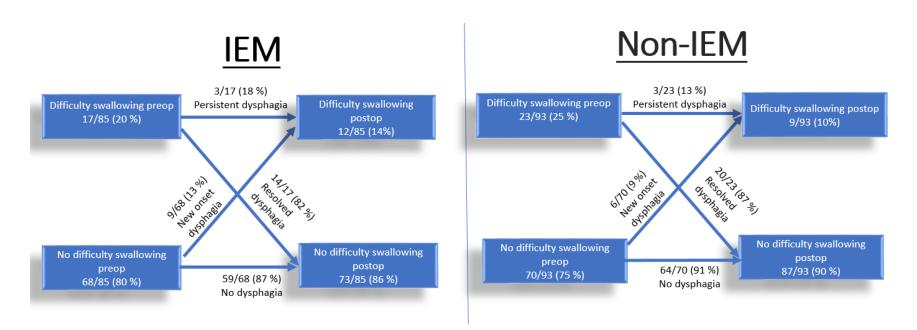


Figure 1: Evolution of dysphagia symptoms preoperatively and postoperatively as assessed by the GERD-HRQL. Dysphagia is considered present where there is a score of >3 for the difficulty swallowing question on the survey.

ADVANCED IMPEDANCE METRICS ON IMPEDANCE-PH TESTING PREDICT LUNG FUNCTION DECLINE AT 1 YEAR IN IDIOPATHIC PULMONARY FIBROSIS PATIENTS

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Background: Gastro-esophageal reflux (GER) has been associated with idiopathic pulmonary fibrosis (IPF). Mean nocturnal baseline impedance (MNBI) is a marker of esophageal mucosal integrity, while post-reflux swallow-induced peristaltic wave (PSPW) index reflects the efficacy of esophageal refluxate clearance. Both metrics offer novel ways to assess reflux burden, but their role in evaluating extraesophageal GER remains unclear.

<u>Aim:</u> To assess the relationship between MNBI and PSPW index on multi-channel intraluminal impedance-pH testing (MII-pH) and lung function decline over 1 year in IPF patients.

Methods: Adults with IPF undergoing pre-lung transplant MII-pH off acid suppression at a tertiary center were enrolled. Pulmonary function test (PFT) data was collected at the time of MII-pH and at 1-year follow-up. MNBI was calculated by averaging baseline impedance at three 10-minute time intervals (1AM, 2AM, 3AM), and classified as proximal (channels 15-17 cm above LES) and distal (channels 3-9 cm above LES). PSPW was defined as a peristaltic swallow propagating through the esophagus within 30 seconds of a reflux event. PSPW index was calculated by dividing total PSPW events by total reflux episodes. Data were analyzed using student's t-test/Pearson's correlation for univariate and linear regression for multivariate analyses.

<u>**Results:</u>** 125 subjects (mean age=61.7 years, 62% male) were included. Significantly greater decline in FEV1 and FVC at 12 months were seen in subjects with lower distal MNBI, proximal MNBI, and PSPW index (Table 1). On multivariate analyses controlling for gender, age and baseline lung function, distal MNBI, proximal MNBI, and PSPW index all remained independently associated with greater FEV1 and FVC decline (Table 2).</u>

<u>Conclusion</u>: Low distal MNBI, proximal MNBI, and PSPW index were independent predictors of more severe lung function decline over 1 year in IPF patients. These advanced impedance metrics may have prognostic value and supports a role for reflux in IPF pathogenesis.

Table 1: Univariate analyses of impedance metrics on MII-pH and change in PFT findings in 12 months. **(A)** Pearson's correlations between impedance metrics and PFT changes showed that distal MNBI, proximal MNBI, and PSPW index all significantly correlated with FEV1 change, percent FEV1 change, FVC change, and percent FVC change in 12 months. **(B)** When impedance metrics are dichotomized per previously published cutoffs (Frazzoni 2014), low distal MNBI (<2292 Ω), low proximal MNBI (<2292 Ω), and low PSPW index (<50%) correlated with more significant decline in PFT findings in 12 months.

Α	FEV1 Change	FEV1 % Change	FVC Change	FVC % Change
	in 12 months	in 12 months	in 12 months	in 12 months
Distal MNBI	R=0.573,	R=0.565,	R=0.550,	R=0.506,
	<i>p</i> =0.0066	<i>p</i> =0.0076	<i>p</i> =0.010	<i>p</i> =0.019
Proximal MNBI	R=0.471,	R=0.532,	R=0.510,	R=0.562,
	<i>p</i> =0.010	<i>p</i> =0.013	<i>p</i> =0.018	<i>p</i> =0.008
PSPW Index	R=0.596,	R=0.626,	R=0.544,	R=0.554,
	<i>p</i> =0.007	<i>p</i> =0.004	<i>p</i> =0.016	<i>p</i> =0.014
В	FEV1 Change	FEV1 % Change	FVC Change	FVC % Change
	in 12 months	in 12 months	in 12 months	in 12 months
Low Distal MNBI	0.19 L/min vs	7.429% vs	0.20 L/min vs	5.14% vs
(<2292 Ω)	-0.095 L/min	-1.786%	-0.11 L/min	-0.929%
vs Normal	<i>p</i> =0.028	<i>p</i> =0.016	<i>p</i> =0.059	<i>p</i> =0.091
Low Proximal	0.22 L/min vs	8.50% vs	0.23 L/min vs	6.50% vs
MNBI (<2292 Ω)	-0.085 L/min	-1.60%	-0.098 L/min	-1.07%
vs Normal	<i>p</i> =0.029	<i>p</i> =0.011	<i>p</i> =0.050	<i>p</i> =0.039
Low PSPW Index	0.34 L/min vs	12.0% vs	0.33 L/min vs	7.67% vs
(<50%)	-0.074 L/min	-0.94	-0.084 L/min	-0.375%
vs Normal	<i>p</i> =0.025	p=0.015	<i>p</i> =0.063	<i>p</i> =0.064

Table 2: Multivariate linear regression models of impedance metrics on MII-pH and change in PFT findings in 12 months. Separate models were constructed for each impedance metric to avoid co-linearity.

Α	FEV1 % Change in 12 months								
Covariates	β-coeff	<i>p</i> -value	β-coeff	<i>p</i> -value	β-coeff	<i>p</i> -value			
Low Distal MNBI	-11.76	0.016	-	-	-	-			
Low Proximal MNBI	-	-	-10.02	0.021	-	-			
Low PSPW	-	-	-	-	-15.03	0.019			
Age	3.34	0.438	5.64	0.202	-0.65	0.888			
Male	5.72	0.244	-2.23	0.589	2.77	0.542			

Smoking	-4.35	0.278	-3.25	0.430	-7.03	0.101
Baseline FEV1 % Predicted	0.020	0.846	-0.069	0.518	-0.02	0.848
В		F۱	/C % Change	in 12 mont	hs	
Covariates	β-coeff	<i>p</i> -value	β-coeff	<i>p</i> -value	β-coeff	<i>p</i> -value
Low Distal MNBI	-8.91	0.034	-	-	-	-
Low Proximal MNBI	-	-	-7.39	0.047	-	-
Low PSPW	-	-	-	-	-10.40	0.078
Age	4.90	0.193	6.36	0.102	2.24	0.600
Male	6.28	0.163	0.017	0.996	3.70	0.402
Smoking	-5.26	0.152	-4.43	0.243	-7.07	0.09
Baseline FVC % Predicted	-0.031	0.741	-0.93	0.356	-0.063	0.596

Hiatal Hernia Recurrence Following Magnetic Sphincter Augmentation and Cruroplasty: Long-Term Outcomes

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BACKGROUND: Magnetic sphincter augmentation (MSA) is an effective treatment option for gastroesophageal reflux disease (GERD). Initially, MSA was relatively contraindicated in patients with concomitant hiatal hernias > 3 cm. Short term data suggest that MSA with hiatal hernia repair may be equally effective. Our study aimed to evaluate long term MSA outcomes, with special attention to hiatal hernia recurrence.

METHODS: We performed a retrospective cohort study of all patients with GERD and intraoperative hiatal hernias between 2009 and 2015, who underwent MSA, hiatal hernia repair without mesh, and cruroplasty. All patients underwent videoesophagram and/or upper endoscopy at one year postoperatively, then annually for up to five years. Hiatal hernias > 2 cm were deemed clinically significant. The gastroesophageal reflux disease health-related quality of life (GERD-HRQL) survey was conducted at 3 months, 6 months, and annually.

RESULTS: There were 79 patients (37 male, 42 female). The mean age was 63.8. The mean follow-up was 2.3 years. At the time of latest follow-up, the mean GERD HRQL score was 5, from 20 preoperatively (p<0.05, Wilcoxon signed rank test). There were two detected hiatal hernia recurrences >2 cm (2.531%). At their last follow-up, 3 (4%) of patients had resumed proton pump inhibitors.

Preoperative Characteristics	
Body mass index (mean, SD)	26.32, 6.9300
GERD-HRQL (mean, SD)	20.29, 8.726
Preop DeMeester score (mean, SD)	50.04, 30.51
Intraoperative Characteristics	
Hiatal Hernia Size (cm), (mean, SD)	4.82, 1.23
Average OR duration in hours (mean, SD)	1.2849, 0.524
Post-operative Outcomes	
Endoscopic dilation, n (%)	16/79 (20.25%)
Postop DeMeester Score (mean, SD)	14.01, 17.51

Table 1: SD=Standard Deviation

CONCLUSIONS: Initial concerns about MSA with hiatal hernia repair are not supported. Our study demonstrates that MSA with cruroplasty is safe and results in excellent long-term outcomes.

IS THERE A NON-BARRETT'S PATHWAY TO ESOPHAGEAL ADENOCARCINOMA? IMPLICATIONS FOR SCREENING AND MANAGEMENT

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Background/Aims:

Barrett's esophagus (BE), defined by the presence of intestinal metaplasia (IM), is the only identifiable precursor lesion for esophageal adenocarcinoma (EAC). Recent studies have suggested the possibility of an alternate, non-IM pathway to EAC that is associated with a more aggressive EAC with worse survival.

Methods:

This was a retrospective cohort study of patients with EAC evaluated at a tertiary care center from 2009-2019. Cases were categorized according to the presence or absence of IM. We compared demographic characteristics, clinical stage, and therapy between the two groups. We used Cox proportional hazards regression to determine the association of IM with overall survival, adjusting for sex, age, proton pump inhibitor use, tumor location, and stage.

Results:

A total of 478 patients were included and 105 (22%) had no evidence of IM (**Table 1**). The non-IM-EAC group had a greater proportion of patients diagnosed with advanced disease (41 vs. 2% for stage 4, p<0.001) and gastroesophageal junction tumors (76 vs. 47%, p<0.001). As a result, they were less likely to undergo endoscopic therapy alone (0.95% vs. 30.56%, p<0.001) or surgery alone (0.95% vs. 9.65%, p=0.003). Although on univariate analysis, IM-EAC patients had improved overall survival compared to non-IM-EAC (HR 0.45, 95%CI 0.33-0.62) (**Figure 1**), the presence of IM-EAC was no longer significant on multivariable analysis (HR 0.89, 95%CI 0.62-1.26). Additional factors associated with survival were age at diagnosis, PPI use, and increasing stage of diagnosis (**Table 2**).

Conclusions:

Patients in the non-IM-EAC cohort are younger, and present with more advanced disease compared to IM-EAC patients. However, the absence of IM was not associated with overall survival and this was largely driven by stage at presentation. Future prospective studies with detailed molecular sequencing are required to clarify if a non-IM-EAC exists, which would have significant implications for screening and management strategies.

Table 1. Characteristics of individuals with (IM-EAC) and without (Non-IM-EAC) intestinal metaplasia

Characteristic	Total	IM-EAC	Non-IM-EAC	P value∗
Survival dava modion (IOD)	N=478	N=373 (78.03%)	N= 105 (21.97%)	-0.001
Survival, days, median (IQR)	607 (267-1224)	660 (295-1330)	424 (219-862)	< 0.001
Age at diagnosis, mean ± SD	64.79 ± 10.78	65.48 ± 10.46	62.36 ± 11.57	0.0086
BMI, kg/m ₂ , mean \pm SD,	$\textbf{27.89} \pm \textbf{5.44}$	28.36 ± 5.50	26.39 ± 4.95	0.937
	N (%)	N (%)	N (%)	P value**
Gender				0.900
Male	425 (88.91)	332 (89.01)	93 (88.57)	
Female	53 (11.09)	41 (10.99)	12 (11.43)	
Race				0.732
White	424 (88.7)	329 (88.20)	95 (90.48)	
Black	3 (0.63)	3 (0.80)	0 (0)	
Asian	1 (0.21)	1 (0.27)	0 (0)	
Unknown	50 (10.46)	40 (10.72)	10 (9.52)	
Smoker				0.353
Non-smoker	162 (33.89)	131 (35.12)	31 (29.52)	
Former/Current	301 (62.97)	229 (61.39)	72 (68.57)	
Unknown	15 (3.14)	13 (3.49)	2 (1.90)	
Ethanol use				0.674
None	393 (82.22)	309 (82.84)	84 (80.00)	
Former/Current	65 (13.60)	48 (12.87)	17 (16.19)	
Unknown	20 (4.18)	16 (4.29)	4 (3.81)	
Family history EAC	20 (4.33)	15 (4.18)	5 (4.85)	0.944
Charlson Deyo Score		, <i>i</i>		0.161
0	315 (65.90)	237 (63.54)	78 (74.29)	
1	96 (20.08) [´]	78 (20.91) [´]	18 (17.14)́	
2	29 (6.07)	26 (6.97)	3 (2.86)	
3+	38 (7.95)	32 (8.58)	6 (5.71)	
PPI Use	271 (58.28)	234 (64.29)	37 (36.63)	< 0.001
Cancer Location				< 0.001
Esophageal	204 (42.68)	183 (49.06)	21 (20.00)	
GEJ	253 (52.93)	173 (46.38)	80 (76.19)	
Unknown	21 (4.39)	17 (4.56)	4 (3.81)	
Clinical Stage			. (0.0.)	<0.001
0	5 (1.05)	5 (1.34)	0 (0)	
1	125 (26.15)	124 (33.24)	1 (0.95)	
2A	6 (1.26)	6 (1.61)	0 (0)	
2B	25 (5.23)	24 (6.43)	1 (0.95)	
3	149 (31.17)	105 (28.15)	44 (41.90)	
4A	46 (9.62)	30 (8.04)	16 (15.24)	
4B	80 (16.74)	44 (11.80)	36 (34.29)	
Unknown	42 (8.79)	35 (9.38)	7 (6.67)	
Endoscopic Eradication	115 (24.06)	114 (30.56)	1 (0.95)	< 0.001
Therapy alone	110 (24.00)	(30.30)	1 (0.33)	\0.001
Surgery alone	37 (7 74)	36 (0.65)	1 (0.05)	0.003
<u> </u>	37 (7.74)	36 (9.65)	1 (0.95)	
Neoadjuvant + surgery	151 (31.59)	110 (29.49)	41 (39.05)	0.063
Chemotherapy +/- radiation	111 (23.22)	66 (17.69)	45 (42.86)	< 0.001
No treatment	2 (0.42)	0 (0)	2 (1.90)	0.008

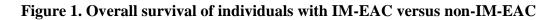
*ANOVA test was used to assess differences among age groups for continuous variables

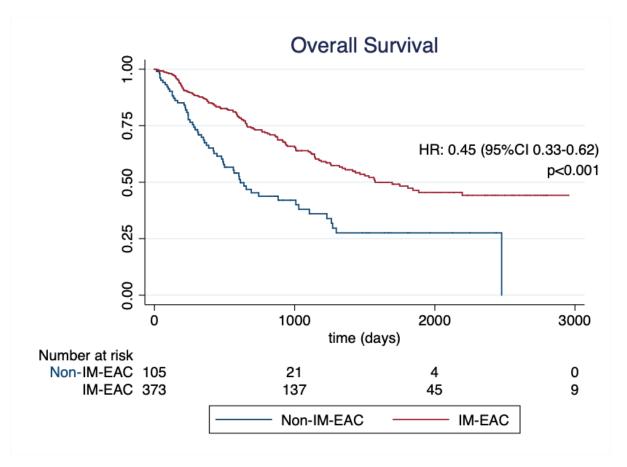
**Chi-square test was used to assess differences among age groups for categorical variables

Table 2. Univariate and Multivariate Survival Analysis

	Univariate Analys	Multivariable Analysis		
Characteristic	Hazard Ratio [95% CI]	P value	Adjusted Hazard Ratio [95% CI]	
Smoker (ref: non)				
Former/ Current	1.03 (0.76-1.40)	0.846		
Unknown	0.82 (0.37-1.80)	0.618		
Ethanol use (ref: none)				
Former/ Current	1.59 (1.05-2.40)	0.27		
Unknown	1.22 (0.66-2.26)	0.530		
Family hx esophageal cancer				
Yes	1.12 (0.59-2.12)	0.732		
Unknown	1.09 (0.41-2.95)	0.860		
Race (ref: white)				
Black	1.49 (0.37-6.02)	0.575		
Asian	5.63 (0.78-40.55)	0.086		
Unknown	1.32 (0.82-2.16)	0.258		
Charlson Deyo Score (ref: 0)				
1	1.37 (0.95-1.97)	0.089		
2	2.03 (1.24-3.34)	0.005		
3+	1.43 (0.86-2.39)	0.167		
Endoscopic Therapy alone	0.17 (0.10-0.29)	< 0.001		
Surgery alone	0.32 (0.15-0.68)	0.003		
Neoadjuvant + surgery	0.79 (0.58-1.08)	0.133		
Chemo +/- radiation	4.37 (3.24-5.90)	< 0.001		
No treatment	121.55 (25.05-589.64)	< 0.001		
Presence of BE/IM	0.45 (0.33-0.62)	< 0.001	0.89 (0.62-1.26)	
Age at diagnosis	1.00 (0.99-1.01)	0.867	1.02 (1.00-1.03)	
Male Sex	1.51 (0.92-2.49)	0.104	1.17 (0.70-1.94)	
PPI Use (ref: no)	0.49 (0.37-0.66)	< 0.001	0.73 (0.53-0.997)	
Location (ref: esophageal)			1.33 (0.95-1.87)	
Gastroesophageal junction	1.82 (1.34-2.48)	< 0.001		
Unknown	1.35 (0.67-2.73)	0.402		
Clinical Stage (ref: 1)	, , , , , , , , , , , , , , , , , , ,			
2A	4.99 (1.48-16.82)	0.009	3.94 (1.07-14.55)	
2B	2.75 (1.25-6.04)	0.012	2.26 (0.97-5.26)	
3	3.70 (2.29-6.15)	< 0.001	3.31 (1.91-5.76)	
4A	6.77 (3.75-12.22)	< 0.001	6.26 (3.38-11.76)	
4B	11.01 (6.52-1860)	<0.001	9.82 (5.55-17.38)	
Unknown	2.18 (1.10-4.31)	0.026	9.82 (5.55-17.38)	

Multivariable analysis included





HIGH RESOLUTION MANOMETRY IN A FUNCTIONING FUNDOPLICATION

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BACKGROUND

The Chicago classification was devised to provide guidance in interpreting High Resolution Manometry (HRM) in patients with esophageal dysphagia. However, it is unclear whether those values are applicable after fundoplication as the literature is sparse. The aim of this study was to provide a full HRM data set in patients with a normal functioning fundoplication.

METHODS

Patients who underwent primary laparoscopic fundoplication were identified from a prospectively collected database. All patients had preoperative pH testing and HRM. Postoperatively, a normal functioning fundoplication was defined by 1) normal acid exposure determined by esophageal pH-testing 2) no dysphagia reported on the standard questionnaire on the day of the postoperative HRM. Fifty patients met criteria and were included in the analysis. Paired t test was performed for analysis of pre- and postoperative changes, unpaired t test was performed for comparison of Nissen and Toupet fundoplication.

RESULTS

	PRE-OPERATIVE VALUES MEDIAN 95 th PERC.		POST-OPERA	DVALUE	
LES	MEDIAN	95 ¹¹¹ PERC.	MEDIAN	95 th PERC.	P VALUE
Total length, cm	3.3	4.8	3.95	4.4	0.0098
Resting pressure, mmHg	10.25	48.37	19.5	41.99	0.0263
IRP, mmHg	6	16	14	25.65	0.0001
EGJ CI, mmHg*cm	9.2	97.76	10.6	61.86	0.7032
ESOPHAGEAL BODY	0.1	00	2010	02.00	0.7001
DCI, mmHg*cm*sec	696.85	2437.55	683.5	3383.4	0.7021
DECA, mmHg	64	157.65	63	131.7	0.6274
CFV, cm/sec	3	5.96	3	13.3	0.3176
DL, sec	7.6	10.27	8.29	11.09	0.0015
Peristaltic swallows, %	85	100	90	100	0.1367
Simultaneous swallows, %	0	20	0	30	0.7282
Failed swallows, %	20	65.5	20	95.5	0.2658
UES					
Resting pressure, mmHg	106.2	218.73	84.5	195.12	0.1526

Postoperative testing was obtained at a median follow up of 12 months.

Table 1: HRM parameters after laparoscopic fundoplication (n = 50)
Image: second s

Table 2 shows comparative values for Nissen and Toupet fundoplication, as well as a comparison of preoperative and postoperative values in those two subgroups. Median composite score for postoperative pH testing was 0.9. No significant difference between Nissen und Toupet fundoplication was found (p=0.1118).

	N	IISSEN (n = 33)		Т	OUPET (n = 17)		
	MEDIAN (95 th) PRE	MEDIAN (95 th) POST	P VALUE PRE/POST	MEDIAN (95 th) PRE	MEDIAN (95 th) POST	P VALUE PRE/POST	P VALUE N/T
LES							
Total length, cm	3.4 (5.24)	4 (4.4)	0.0971	3.2 (4.14)	3.7 (4.44)	0.0387	0.2963
Resting pressure,	13 (45.8)	20 (39.2)	0.1543	8 (46.6)	18 (46.18)	0.0841	0.7174
mmHg							
IRP, mmHg	6 (16)	15 (29.4)	0.0001	5 (16.06)	12 (23.2)	0.0003	0.3667
EGJ CI, mmHg*cm	12.09 (98)	12.91 (80.63)	0.6429	7.36 (85.26)	8.93 (61.47)	0.8778	0.2918
ESOPHAGEAL BODY							
DCI, mmHg*cm*sec	1016 (3203.8)	817 (3698.2)	0.4016	304 (1601.2)	348 (2353)	0.1072	0.0864
DECA, mmHg	75 (164.8)	66 (133.8)	0.3589	29 (101.6)	39 (113)	0.2914	0.0436
CFV, cm/sec	3 (6.1)	2.95 (11.5)	0.5413	3 (5.2)	3.6 (9.7)	0.3903	0.8656
DL, sec	7 (9.26)	8.4 (11.4)	0.0060	8.3 (14.88)	8 (10.16)	0.1622	0.5062
Peristaltic	90 (100)	90 (100)	0.0503	70 (100)	70 (100)	1	0.3324
swallows, %							
Simultaneous	0 (20)	0 (42)	0.2719	0 (28)	0 (6)	0.1771	0.2492
swallows, %							
Failed swallows, %	20 (54)	10 (54)	0.7807	30 (74)	40 (100)	0.2624	0.0060
UES							
Resting pressure, mmHg	124 (224.2)	81 (183.4)	0.0134	94.1 (189.58)	89.2 (218.4)	0.3497	0.2292

Table 2: HRM values after Nissen or Toupet fundoplication

CONCLUSION

This data provides useful guidance for using HRM in clinical management of dysphagia after fundoplication. As expected, the addition of a fundoplication significantly increases the LES measurements (total length, resting pressure, IRP). However, the previously accepted upper limit defining esophageal outflow obstruction (IRP > 15mmHg) is not clinically applicable after fundoplication as the majority of patients in this dysphagia-free cohort exceeded this value. Interestingly, there does not appear to be a difference in HRM LES values between Nissen and Toupet fundoplication.

The Impact of Body Mass Index on Recurrence Rates Following Laparoscopic Paraesophageal Hernia Repair

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Introduction:

Laparoscopic paraesophageal hernia repair (LPEHR) in obese patients is controversial, as obesity is a known risk factor for other types of hernia repairs. There is little debate that combining the operation with a concurrent weight loss procedure is optimal for patients with class III obesity. However, many patients who present with symptomatic paraesophageal hernias (PEH) are disinterested in, do not qualify for, or do not have insurance coverage for bariatric surgery. The aim of this study is to review the impact of body mass index (BMI) on hernia recurrence rates following LPEHR.

Methods:

All patients who underwent LPEHR between 2006-2012 were identified from a prospectively collected database. Inclusion criteria included elective repairs that had both non-permanent mesh reinforcement of the crural closure and fundoplication. Redo operations, Collis gastroplasty, and relaxing incisions were excluded. Concurrent bariatric surgery was routinely offered to patients with BMI \geq 35. Recurrence was defined on post-operative esophagram or endoscopy as >2cm of intrathoracic stomach. Chi-squared and Fischer's exact test were used to compare outcomes based on BMI stratification.

Results:

Three hundred and fifty patients met clinical criteria and 236 completed follow up evaluations. Radiographic (n=157) and/or endoscopic (n=132) follow-up was performed at a mean of 34 months (IQR 16-46 months). Hernia recurrence was significantly less frequent for normal weight individuals compared to the rest of the group (p=0.039) but there is no significant difference between BMI<35 and \geq 35 (p=0.882). Redo LPEHR was performed in 41% of recurrences (9.7% of total group).

	All Patients n=236	Normal weight (BMI 18.5-24.9)	Overweight (BMI 25-29.9)	Class I Obesity (BMI 30-34.9)	Class II Obesity (BMI 35-39.9)	Class III Obesity (BMI \ge 40)	p-value
		n=37	n=84	n=67	n=37	n=11	
BMI mean	30.46	22.61	27.60	32.15	37.25	43.49	
Recurrence	23.7%	10.8%	30.9%	23.9%	24.3%	18.1%	0.197
Rate	(56/236)	(4/37)	(26/84)	(16/67)	(9/37)	(2/11)	
Operative	11.4%	8.1%	10.7%	11.9%	10.8%	27.2%	0.528
Complications	(27/236)	(3/37)	(9/84)	(8/67)	(4/37)	(3/11)	

Table1.

Conclusion:

Hernia recurrence rates after LPEHR are significantly improved for normal weight individuals. However, many patients requiring LPEHR do not meet bariatric surgery criteria, or do not want weight loss surgery. BMI alone should not be an exclusion criterion for patients with symptomatic PEH, but a preoperative weight loss program should be considered when clinically reasonable.