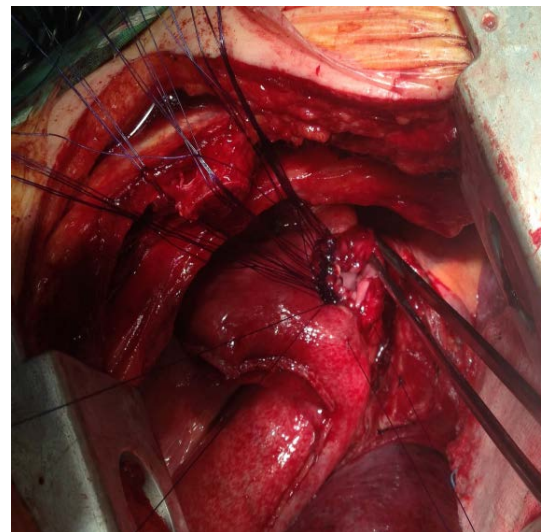
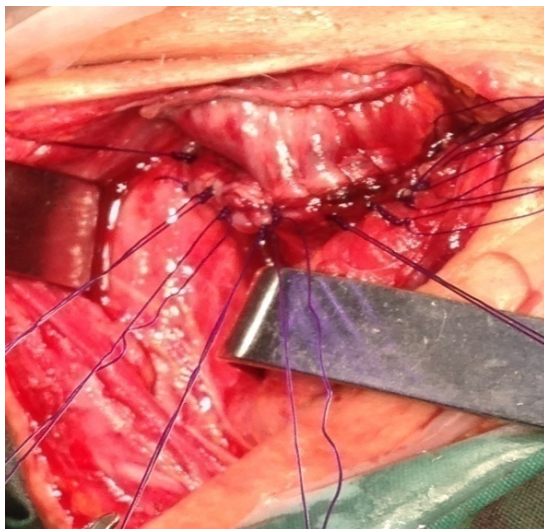




ΠΑΘΗΣΕΙΣ ΟΙΣΟΦΑΓΟΥ



ΔΗΜΗΤΡΙΟΣ Α. ΣΧΙΖΑΣ

*ΕΠΙΚΟΥΡΟΣ ΚΑΘΗΓΗΤΗΣ
Α΄ ΧΕΙΡΟΥΡΓΙΚΗ ΚΛΙΝΙΚΗ ΕΚΠΑ
ΛΑΪΚΟ ΝΟΣΟΚΟΜΕΙΟ*



ΑΝΑΤΟΜΙΑ/ΙΣΤΟΛΟΓΙΑ ΟΙΣΟΦΑΓΟΥ



ΑΝΑΤΟΜΙΑ ΟΙΣΟΦΑΓΟΥ

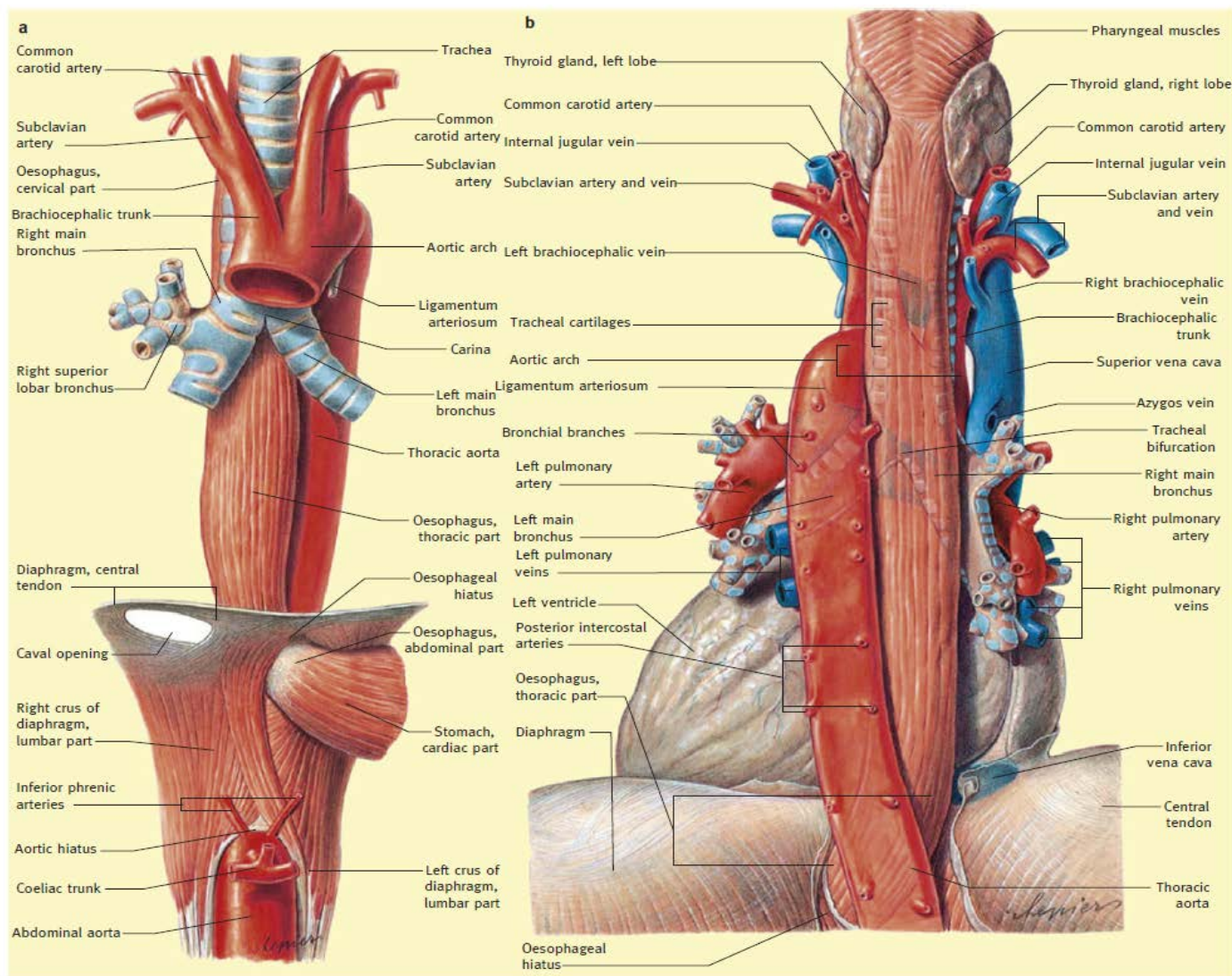
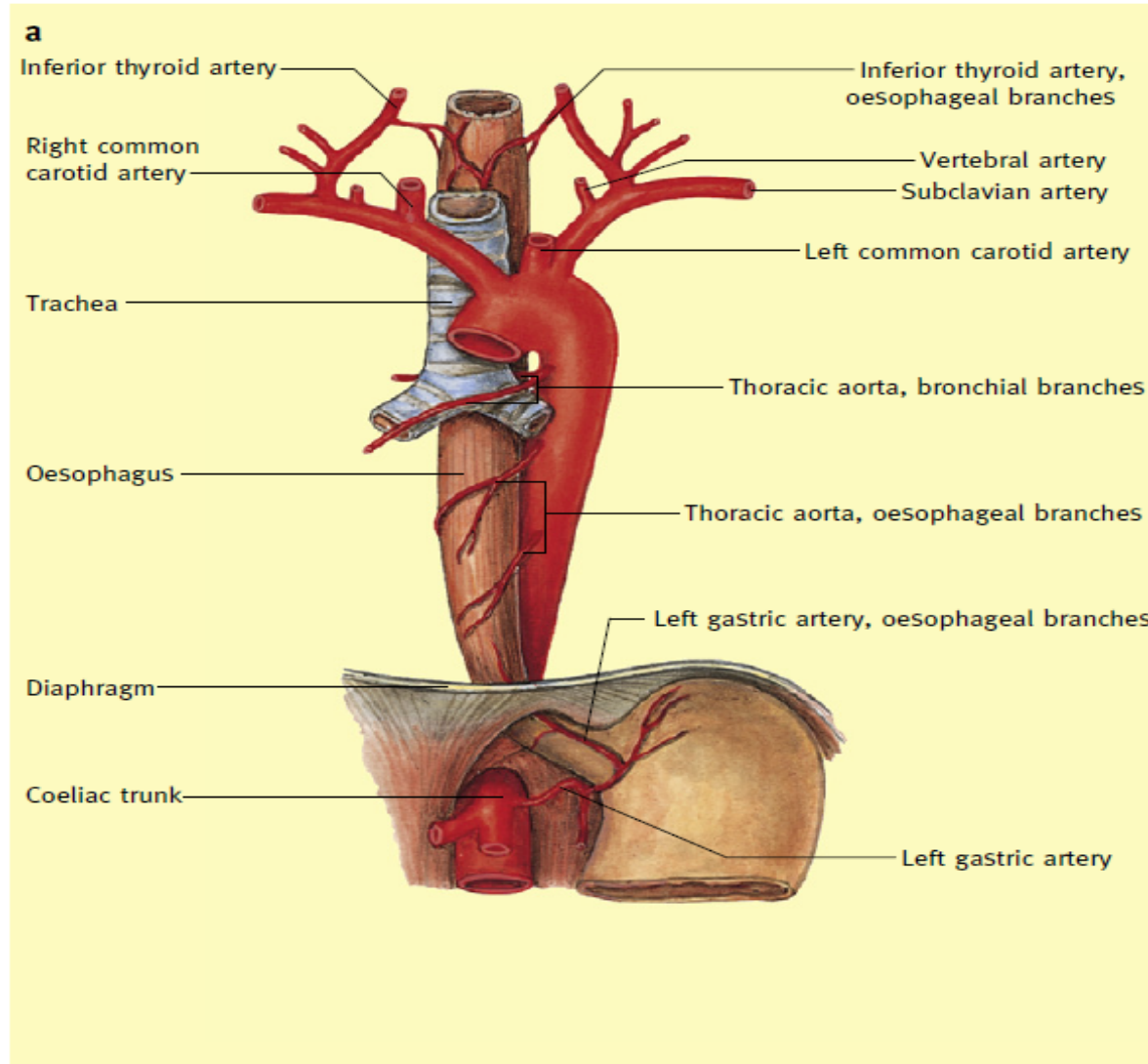


Figure 1 The oesophagus: anterior view (a), posterior view (b). (From Gray's Anatomy 40th edition).

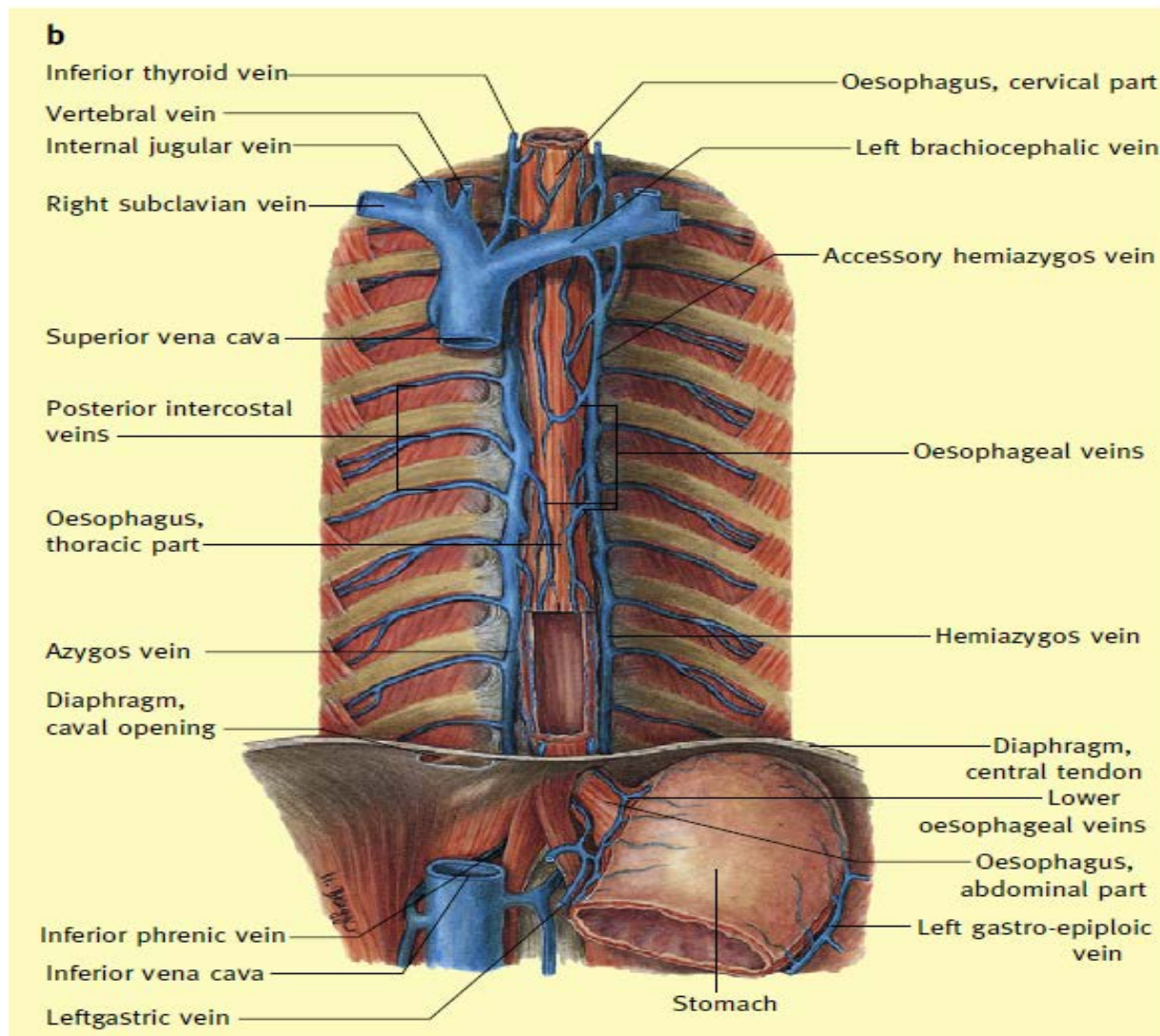


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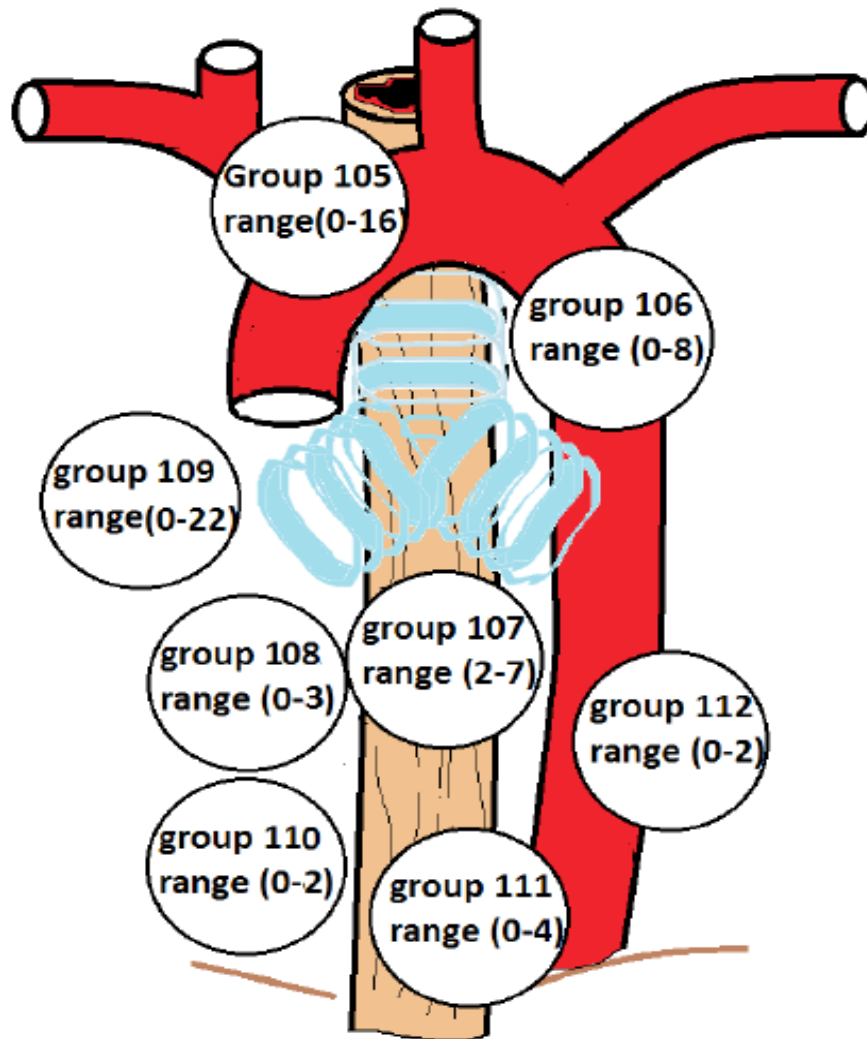


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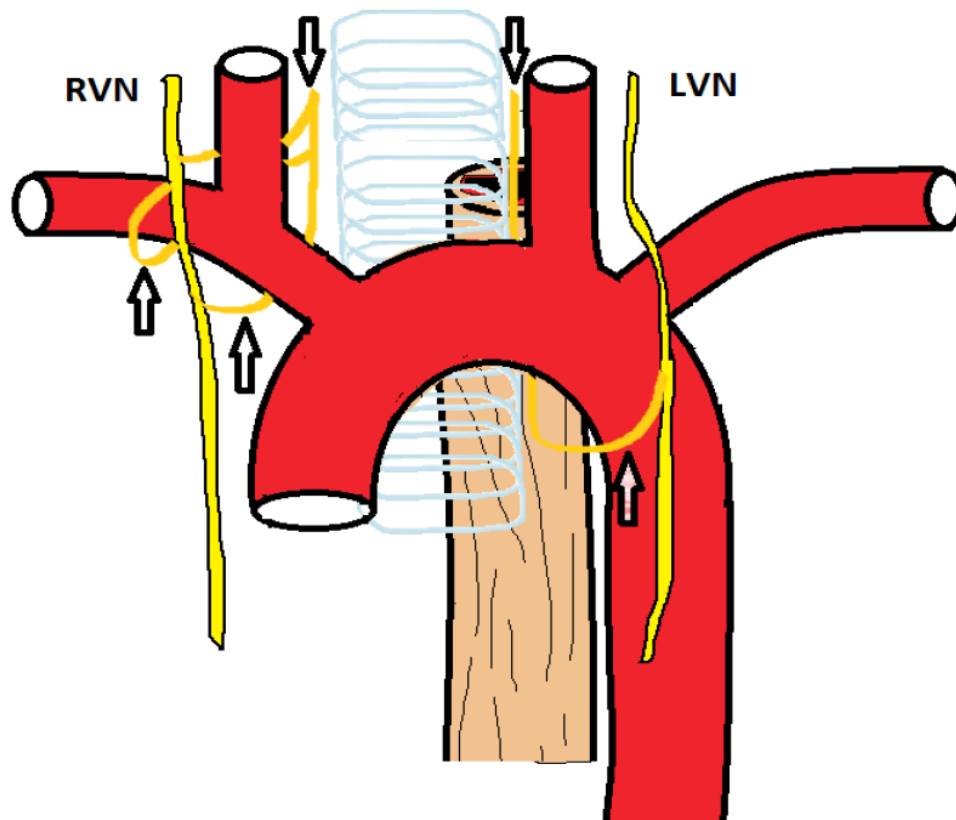


ΛΕΜΦΙΚΗ ΑΠΟΧΕΤΕΥΣΗ





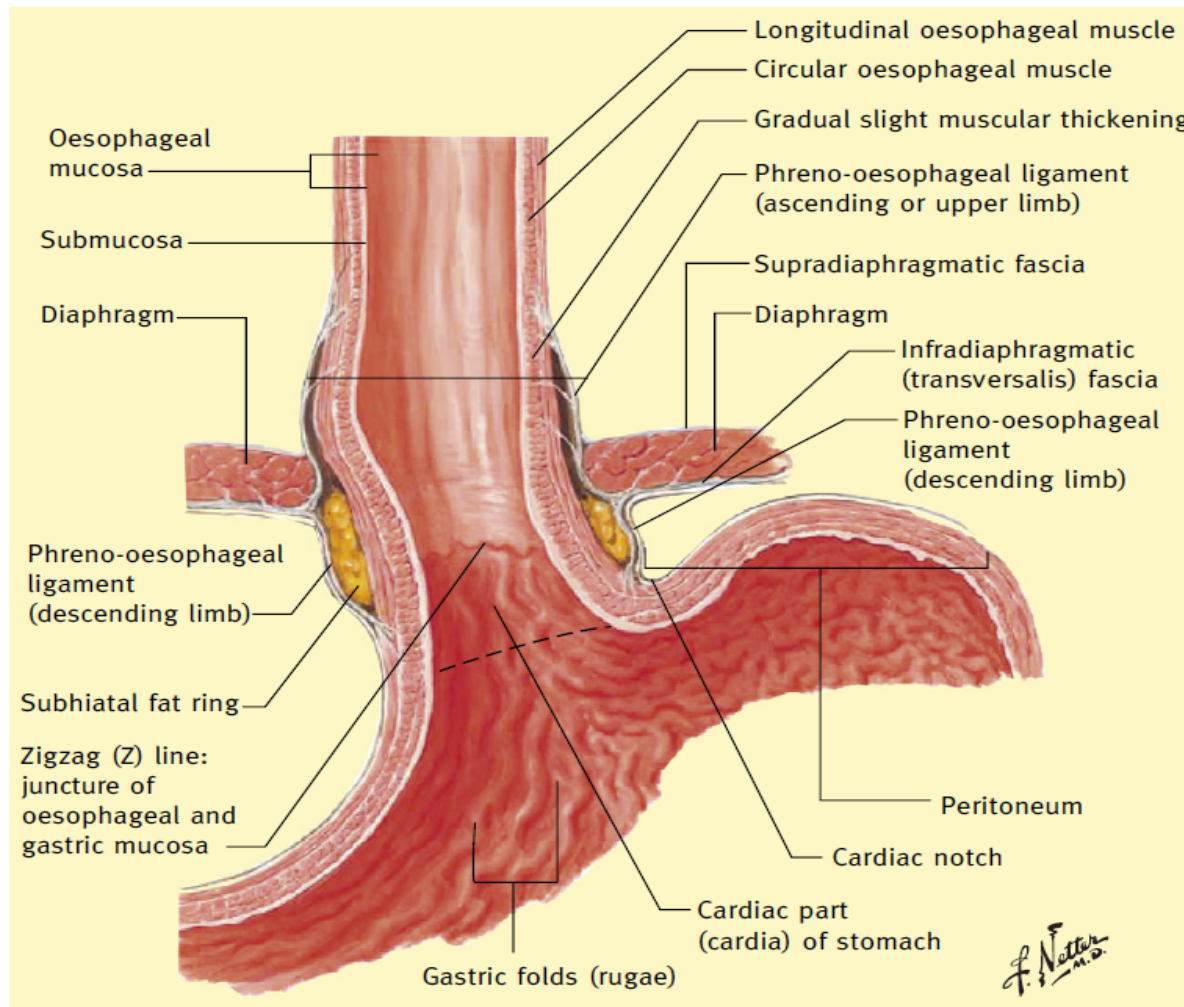
ΠΑΛΙΝΔΡΟΜΑ ΛΑΡΥΓΓΙΚΑ ΝΕΥΡΑ



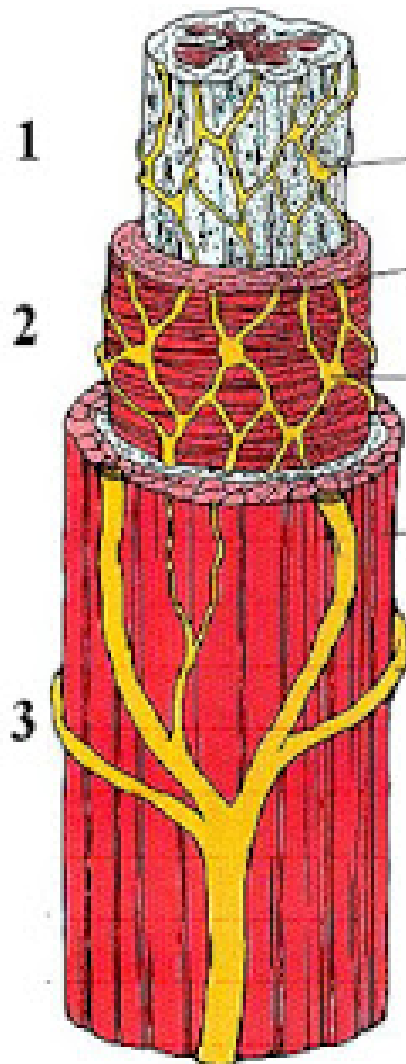
RVN: right vagus nerve. LVN: left vagus nerve. Arrow: recurrent laryngeal nerves



ΑΝΑΤΟΜΙΑ ΚΑΡΔΙΟΟΙΣΟΦΑΓΙΚΗΣ ΣΥΜΒΟΛΗΣ



The lower end of the oesophagus and oesophago-gastric junction. (From Gray's Anatomy 40th edition).



MUCOSA and SUBMUCOSA

CIRCULAR MUSCLE: contraction causes an increase in luminal pressure

LONGITUDINAL MUSCLE: contraction causes shortening

INNERVATION = (1) MEISSNER/SUBMUCOSAL PLEXUS; (2) AUERBACH/MYENTERIC PLEXUS; (3) VAGUS NERVE

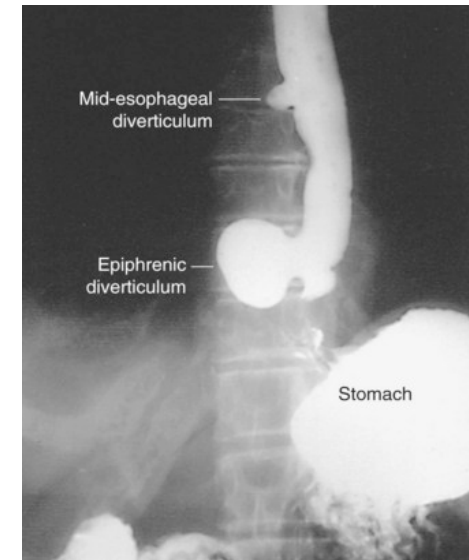


ΕΚΚΟΛΠΩΜΑΤΑ ΟΙΣΟΦΑΓΟΥ



ΘΕΣΗ / ΜΗΧΑΝΙΣΜΟΣ

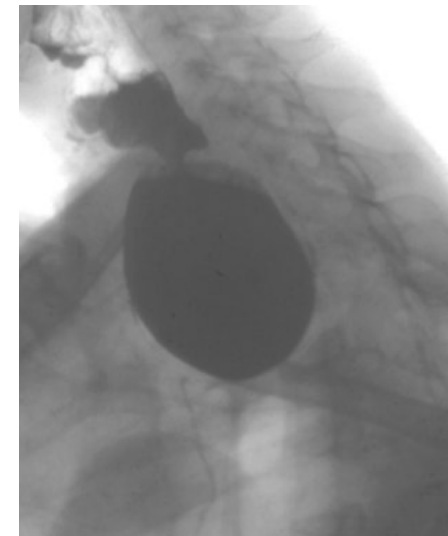
Zenker diverticulum	Immediately above the <u>upper esophageal sphincter</u>
Traction diverticulum	Near the <u>midpoint of the esophagus</u>
Epiphrenic diverticulum	Immediately above the <u>lower esophageal sphincter</u>





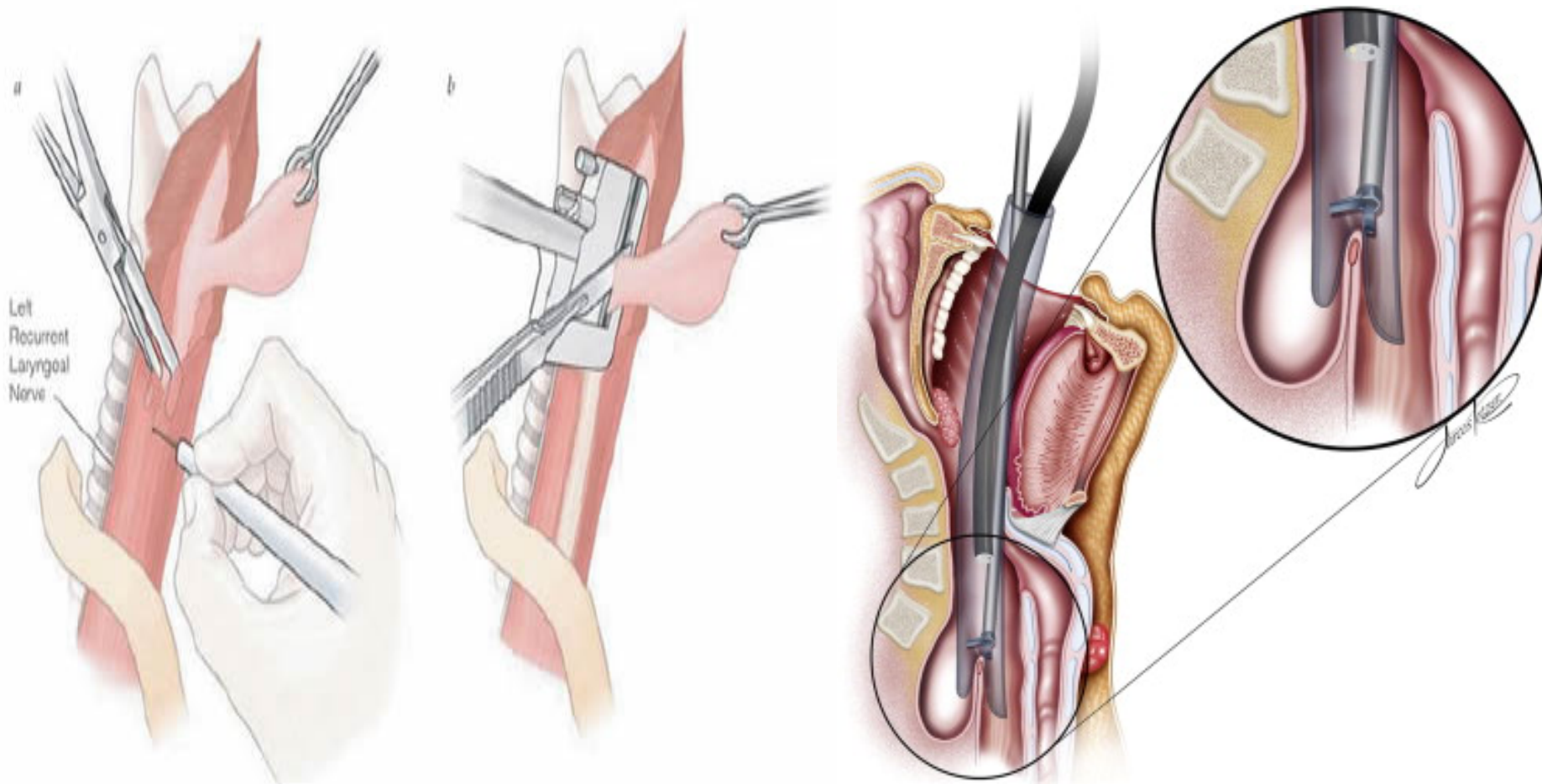
ΕΚΚΟΛΠΩΜΑ ZENKER

- Ψευδές εκκόλπωμα
- Ασυνέργεια κρικοφαρυγγικού μυός κατά την κατάποση
- Δυσκαταποσία, αναγωγές τροφών ημερών, πνιγμονή, κακοσμία στόματος
- Σπάνια επιπλοκές (διάτρηση, αιμορραγία)
- Διόγκωση αρ. πλάγιας τραχηλικής χώρας
- Διάγνωση (γαστροσκόπηση, **βαριούχο γεύμα**)





ΕΚΚΟΛΠΩΜΑ ΖΕΝΚΕΡ – ΑΝΤΙΜΕΤΩΠΙΣΗ





ΚΙΝΗΤΙΚΕΣ ΔΙΑΤΑΡΑΧΕΣ ΟΙΣΟΦΑΓΟΥ



ΤΑΞΙΝΟΜΗΣΗ ΚΙΝΗΤΙΚΩΝ ΔΙΑΤΑΡΑΧΩΝ

Classification of esophageal motility disorders

- **Inadequate LES relaxation**

Classic achalasia

Atypical disorders of LES relaxation

- **Uncoordinated contraction**

Diffuse esophageal spasm

- **Hypercontraction**

Nutcracker esophagus

Isolated hypertensive LES

- **Hypocontraction**

Ineffective esophageal motility



ΑΧΑΛΑΣΙΑ ΟΙΣΟΦΑΓΟΥ

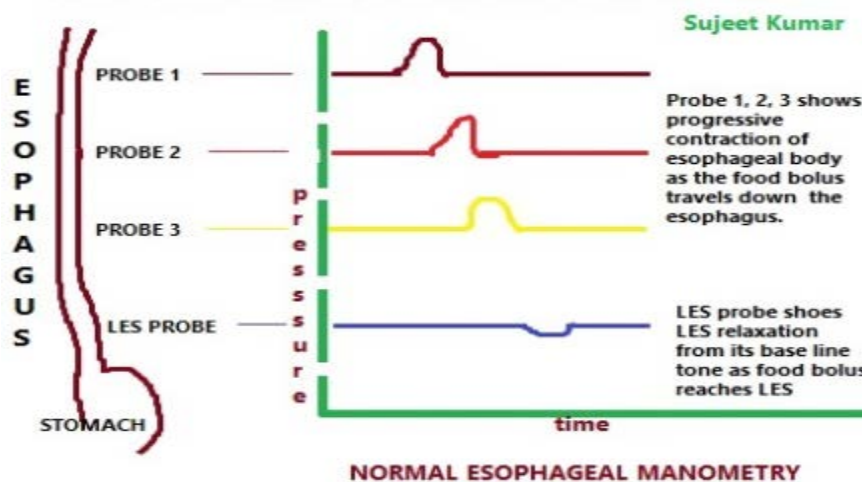
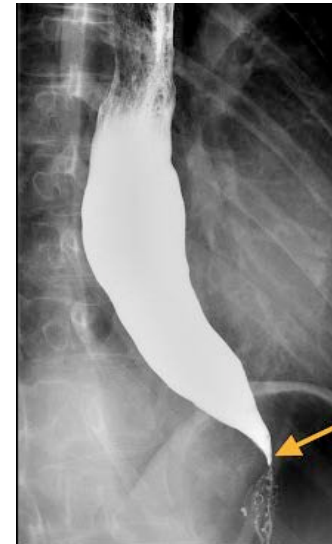


ΚΛΙΝΙΚΗ ΕΙΚΟΝΑ

- Δυσφαγία (παράδοξη δυσφαγία ?)
- Αναγωγές
- Θωρακικό άλγος
- Αίσθημα καύσου
- Μικροεισροφήσεις
- **Μεγάλη χρονική απόσταση από την έναρξη των συμπτωμάτων μέχρι τη διάγνωση**

ΔΙΑΓΝΩΣΤΙΚΕΣ ΕΞΕΤΑΣΕΙΣ

- Ενδοσκόπηση
- Βαριούχο Γεύμα
- Αξονική Τομογραφία
- 24ωρη Ρημετρία
- Μανομετρία





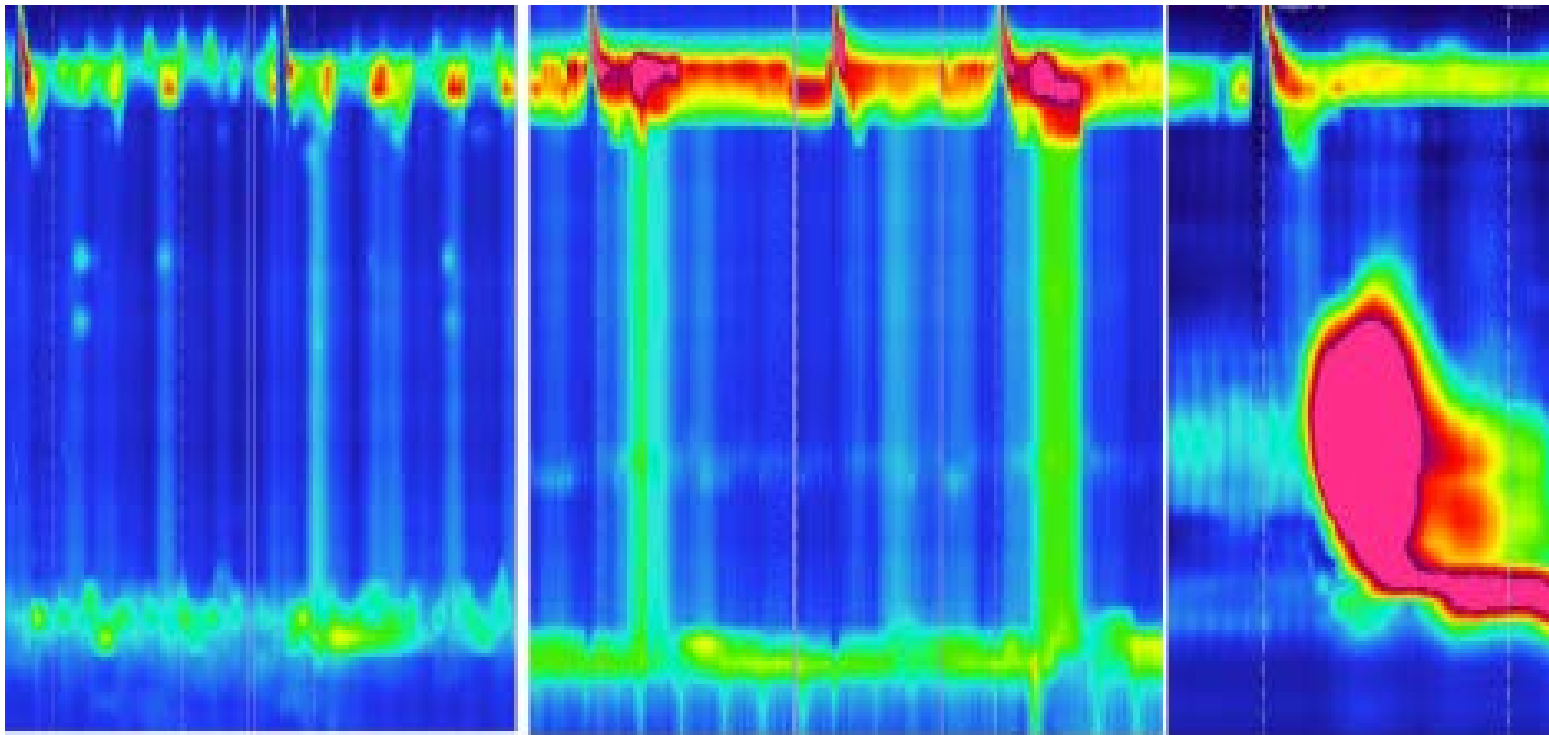
CHICAGO CLASSIFICATION

Table 1. Clinical achalasia syndromes within and beyond Chicago Classification v3.0

Syndrome	Median IRP	Esophageal contractility	Qualifications/notes
Type I achalasia	> 15 mmHg	Absent contractility	
Type II achalasia	> 15 mmHg	Absent peristalsis Pan-esophageal pressurization with $\geq 20\%$ of swallows	
Type III achalasia	> 15 mmHg	Absent peristalsis Premature contractions with $\geq 20\%$ of swallows	
EGJ outflow obstruction	> 15 mmHg	Sufficient peristalsis to exclude types I, II or III achalasia	Heterogeneous group Can be early or incomplete achalasia (12–40%) Can resolve spontaneously Technical issues
Absent contractility	≤ 15 mmHg (CART analysis suggested ≤ 10 mmHg)	Absent contractility	Heterogeneous group Abnormal FLIP distensibility index or esophageal pressurization with swallows or MRS supports achalasia
Distal esophageal spasm	Normal or increased	$\geq 20\%$ premature contractions (DL < 4.5 s)	May be evolving type III achalasia
Jackhammer	Normal or increased	$\geq 20\%$ of swallows with DCI > 8000 mmHg s cm	May be evolving type III achalasia if DL < 4.5 s with $\geq 20\%$ swallows
Opioid effect	> 15 mmHg	Normal, hypercontractile, or premature	Can mimic EGJ outflow obstruction, type III achalasia, DES, or jackhammer
Mechanical obstruction	Normal or increased	Absent, normal, or hypercontractile	EUS or CT imaging of the EGJ may clarify the cause



CHICAGO CLASSIFICATION



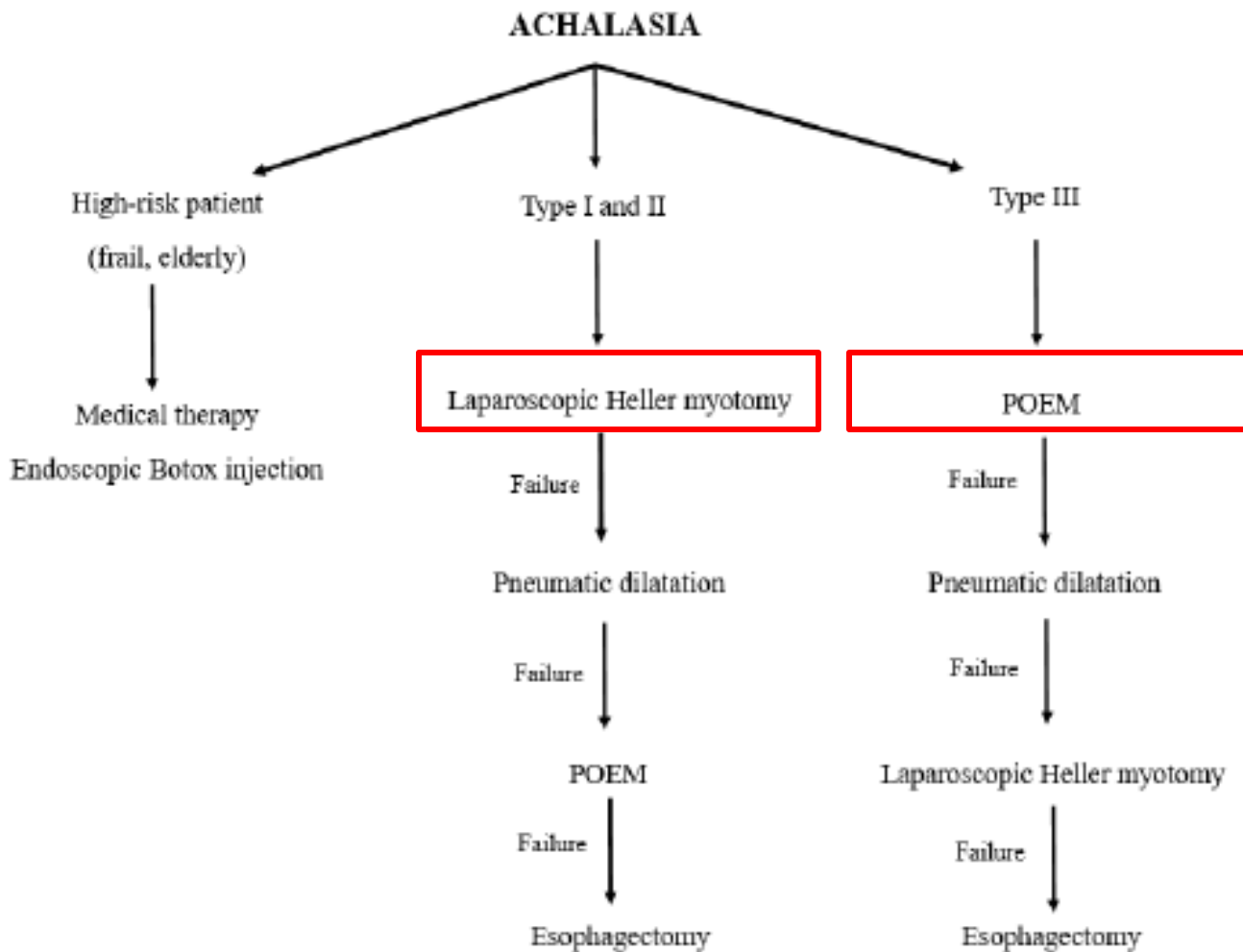
Type I

Type II

Type III

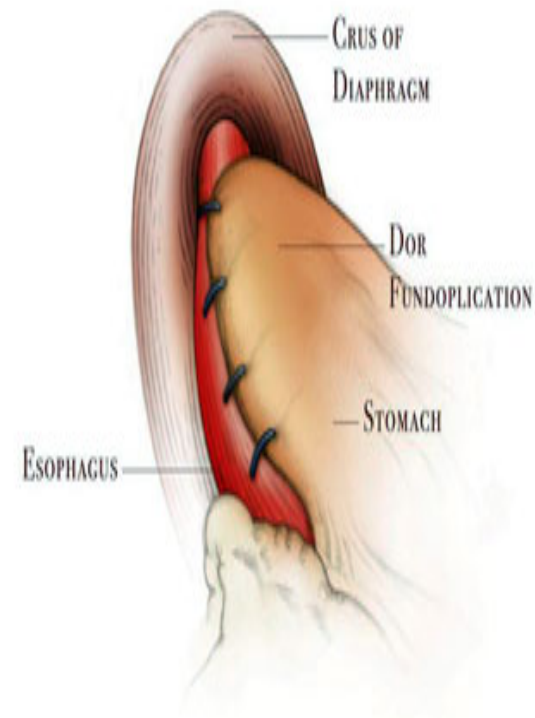
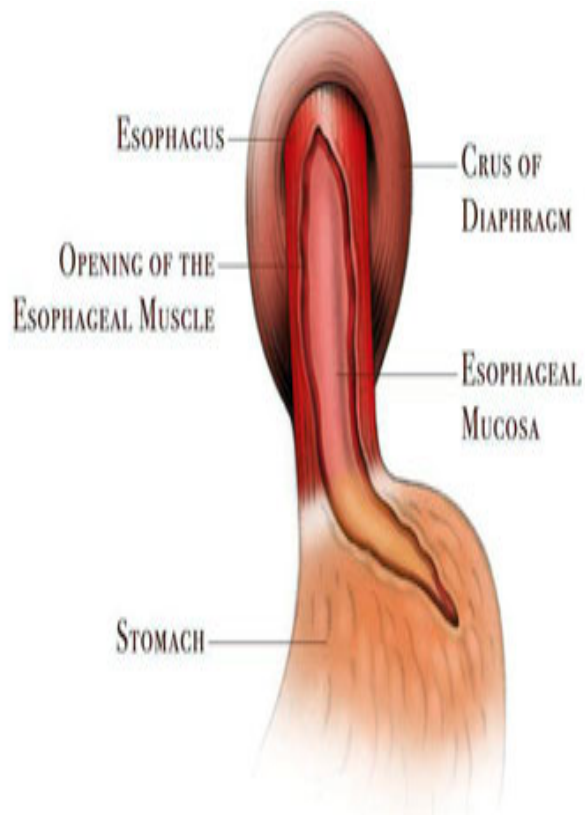
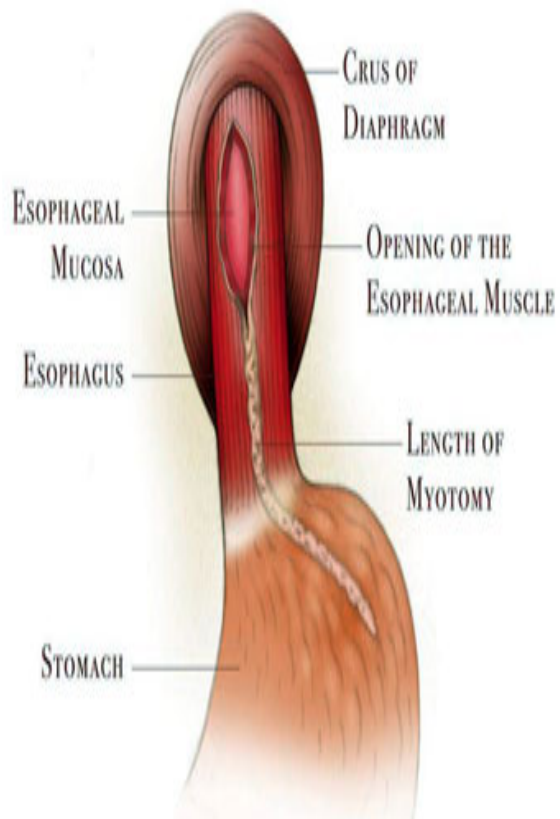


ΘΕΡΑΠΕΥΤΙΚΟΣ ΑΛΓΟΡΙΘΜΟΣ





HELLER MYOTOMY





ΔΙΑΓΝΩΣΤΙΚΗ ΔΙΕΡΕΥΝΗΣΗ

- Ιστορικό, κλινική εξέταση (αποσαφήνιση συμπτωμάτων)
- Ιδιαίτερη σημασία η ανταπόκριση στη λήψη PPI's
- Βαριούχο γεύμα για αποσαφήνιση της ανατομίας (π.χ. συνυπάρχουσα διαφραγματοκήλη, στένωση οισοφάγου κ.α.)
- **Ενδοσκόπηση (Barrett's, οισοφαγίτιδα, γαστρίτιδα, πεπτικό έλκος, καρκίνος)**
- Μανομετρία (αποκλεισμός αχαλασίας, συνύπαρξη κάποιας κινητικής διαταραχής)
- **ΡΗμετρία (το gold-standard για τη διάγνωση)**



ΕΝΔΕΙΞΕΙΣ ΧΕΙΡΟΥΡΓΙΚΗΣ ΑΝΤΙΜΕΤΩΠΙΣΗΣ

- **Αντικειμενική διάγνωση ΓΟΠΝ και καλή ανταπόκριση στα PPI's**
- **Επιθυμία ασθενούς να διακόψει τα PPI's (νέοι ασθενείς, κόστος, επιπλοκές κ.α.)**
- Αναγωγές και καύσος που δεν ελέγχονται πολύ καλά φαρμακευτικά
- Σοβαρή υποψία για εξωγαστρεντερικές εκδηλώσεις ΓΟΠΝ (αναπνευστικές επιπλοκές κ.α.)

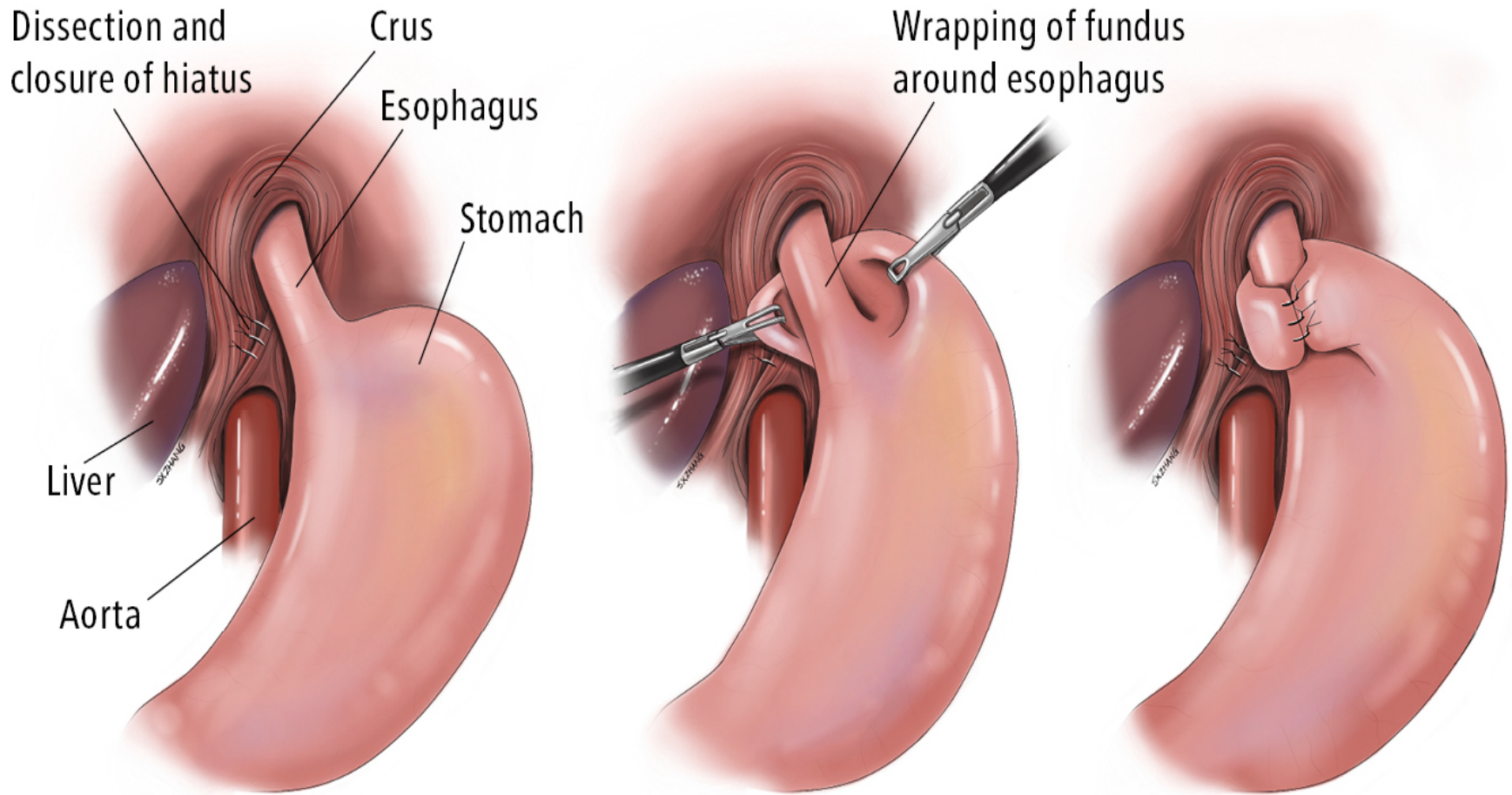


ΤΙ ΕΙΔΟΥΣ ΘΟΛΟΠΛΑΣΤΙΚΗ

- Σήμερα αποτελεί **gold-standard** η **πλήρης (360°) θολοπλαστική κατά Nissen** σε όλους τους ασθενείς εκτός από αυτούς με **αχαλασία ή σκληρόδερμα**
- Η θολοπλαστική κατά Nissen προσφέρει καλύτερο έλεγχο της παλινδρόμησης από τις μερικές θολοπλαστικές, με τα ίδια ποσοστά μετεγχειρητικής δυσφαγίας
- Ειδικά σε παχύσαρκους με ΓΟΠΝ, ιδανική επέμβαση είναι το Roux-en Y Gastric Bypass



ΘΟΛΟΠΛΑΣΤΙΚΗ ΚΑΤΑ NISSEN

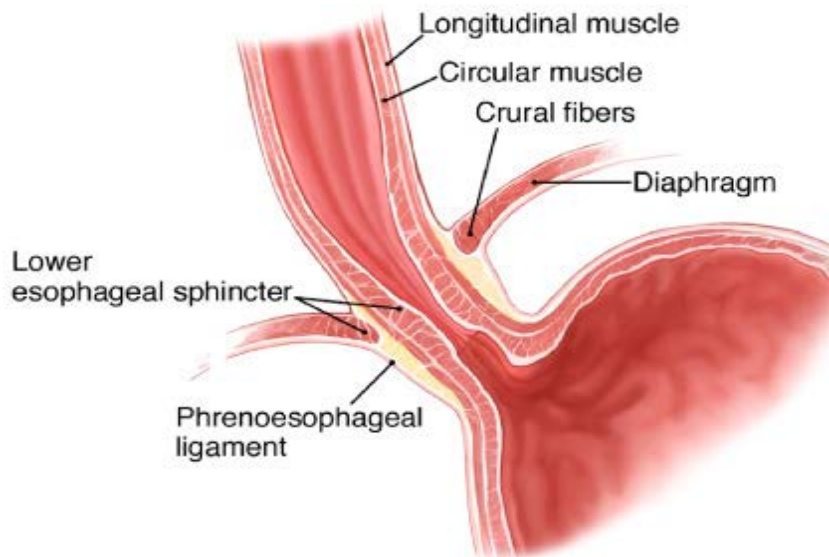




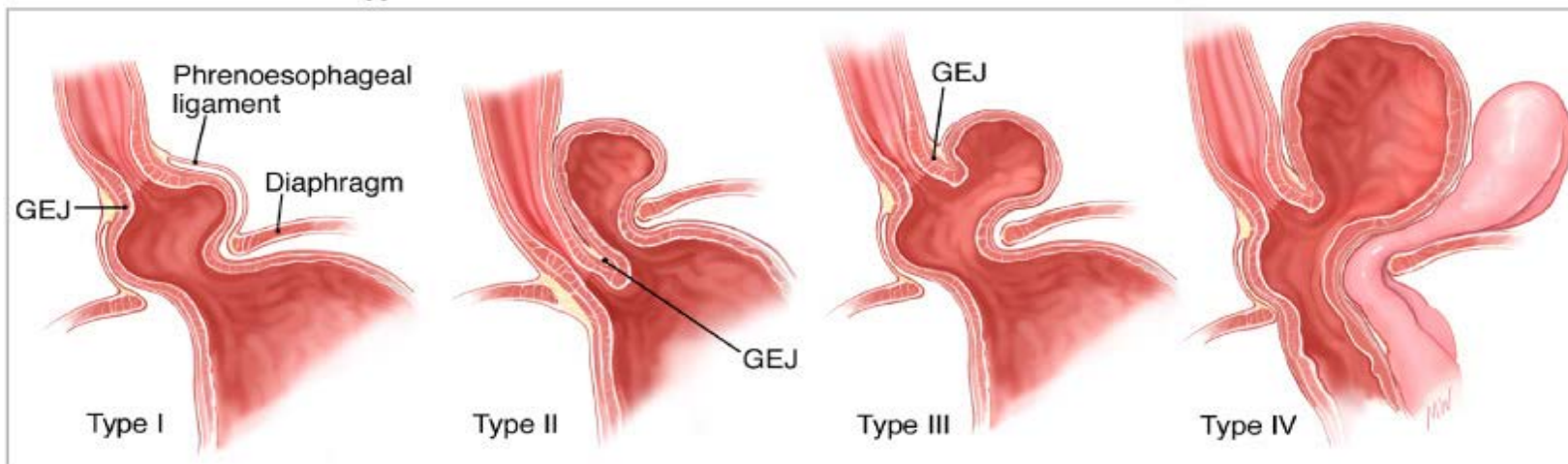
ΔΙΑΦΡΑΓΜΑΤΟΚΗΛΕΣ



ΤΥΠΟΙ ΔΙΑΦΡΑΓΜΑΤΟΚΗΛΗΣ



A



B



ΕΝΔΕΙΞΕΙΣ ΧΕΙΡΟΥΡΓΙΚΗΣ ΑΝΤΙΜΕΤΩΠΙΣΗΣ

- Η αποκατάσταση κατ'επολίσθησιν διαφραγματοκήλης (Τύπου I) απουσία ΓΟΠΝ δεν ενδείκνυται
- Όλες οι συμπτωματικές παραοισοφαγικές διαφραγματοκήλης (Τύπου II,III,IV) πρέπει να χειρουργούνται
- Στις ασυμπτωματικές παραοισοφαγικές διαφραγματοκήλες (Τύπου II,III,IV) η θεραπευτική προσέγγιση πρέπει να εξατομικεύεται ανάλογα με την ηλικία και τις συννοσηρότητες του ασθενούς



ΤΕΧΝΙΚΑ ΣΤΟΙΧΕΙΑ

- **Η λαπαροσκοπική αποκατάσταση** προσφέρει μικρότερη νοσηρότητα και διάρκεια νοσηλείας σε σχέση με την ανοιχτή αποκατάσταση και αποτελεί το **gold standard**
- **Ο σάκος της διαφραγματοκήλης πρέπει να ανατάσσεται** από το μεσοθωράκιο και ιδανικά να εκτέμνεται
- Η χρήση πλέγματος οδηγεί σε **μείωση των βραχυπρόθεσμων ποσοστών** υποτροπής της διαφραγματοκήλης
- Η καρδιοοισοφαγική συμβολή πρέπει οπωσδήποτε να βρεθεί σε **υποδιαφραγματική θέση (2-3 cm)** μετά την αποκατάσταση της διαφραγματοκήλης



ΚΑΡΚΙΝΟΣ ΟΙΣΟΦΑΓΟΥ

ΕΠΙΔΗΜΙΟΛΟΓΙΑ



ΠΑΓΚΟΣΜΙΑ ΕΠΙΠΤΩΣΗ - ΑΝΔΡΕΣ

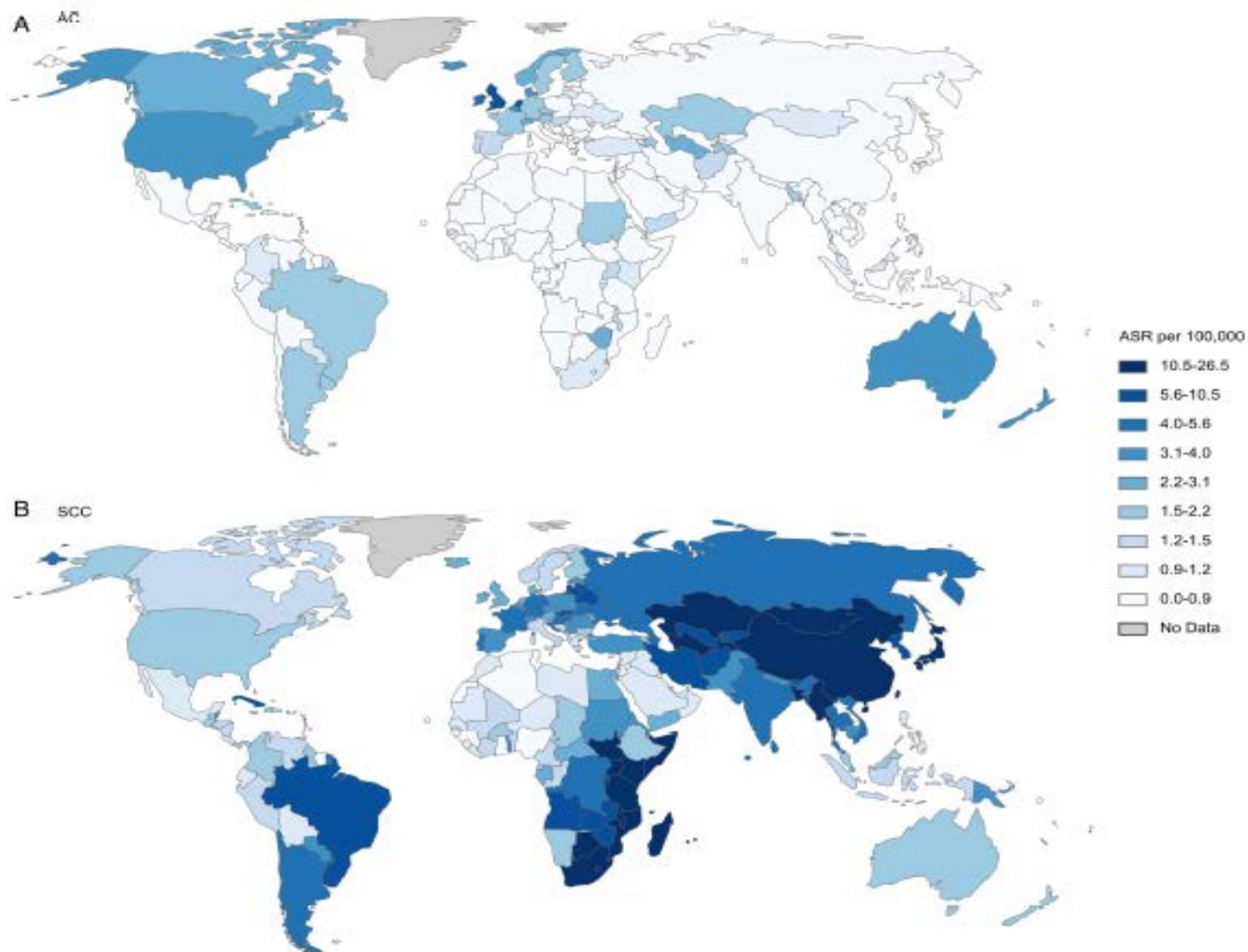


Figure 1 Age-standardised incidence rate (ASR) per 100 000 of (A) oesophageal adenocarcinoma and (B) squamous cell carcinoma in men. AC, adenocarcinoma; SCC, squamous cell carcinoma.



ΠΑΓΚΟΣΜΙΑ ΕΠΙΠΤΩΣΗ - ΓΥΝΑΙΚΕΣ

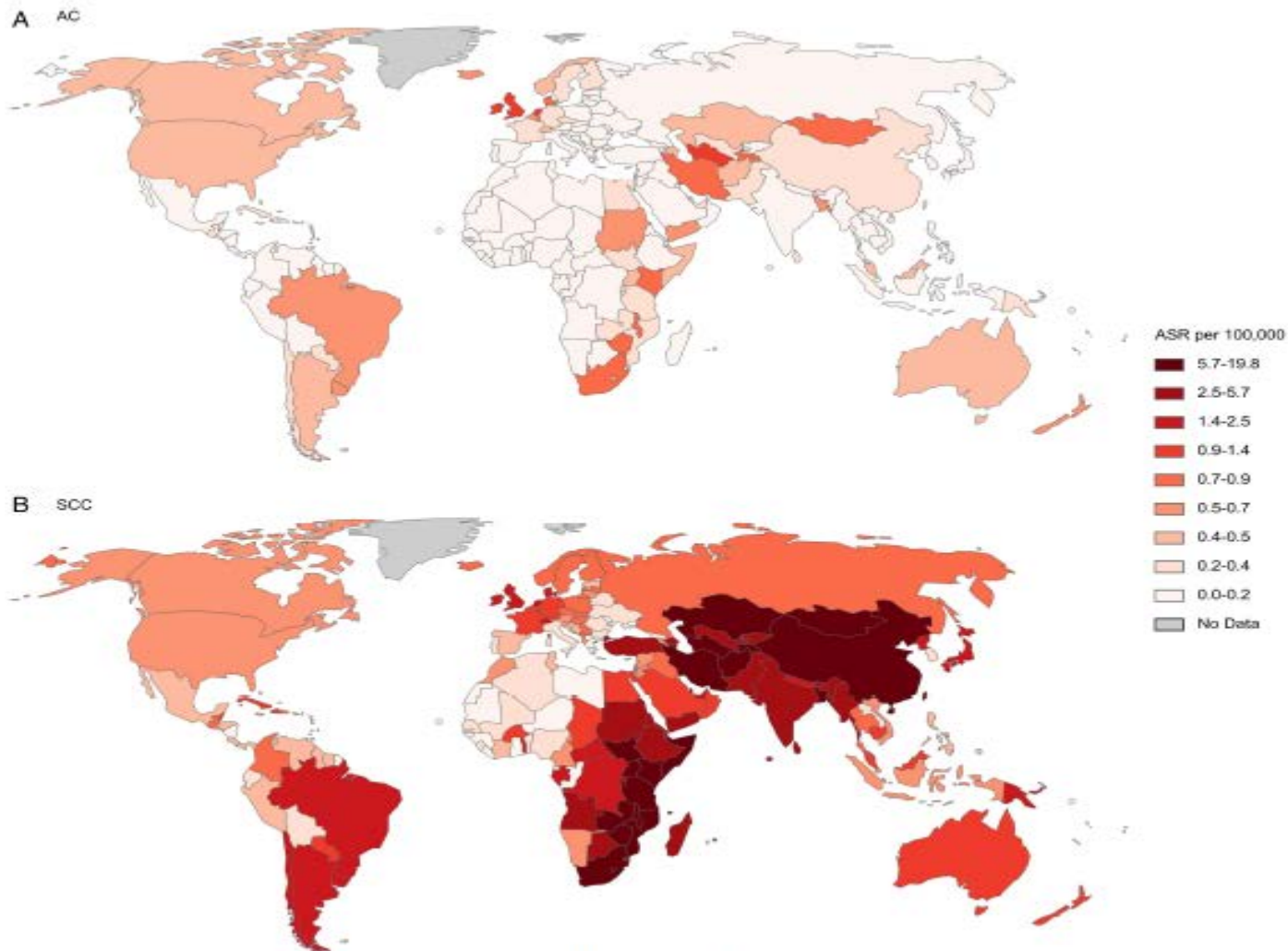


Figure 2 Age-standardised incidence rate (ASR) per 100 000 of (A) oesophageal adenocarcinoma and (B) squamous cell carcinoma in women. AC, adenocarcinoma; SCC, squamous cell carcinoma.



ΠΑΓΚΟΣΜΙΑ ΕΠΙΠΤΩΣΗ ΑΝΑ ΗΠΕΙΡΟ

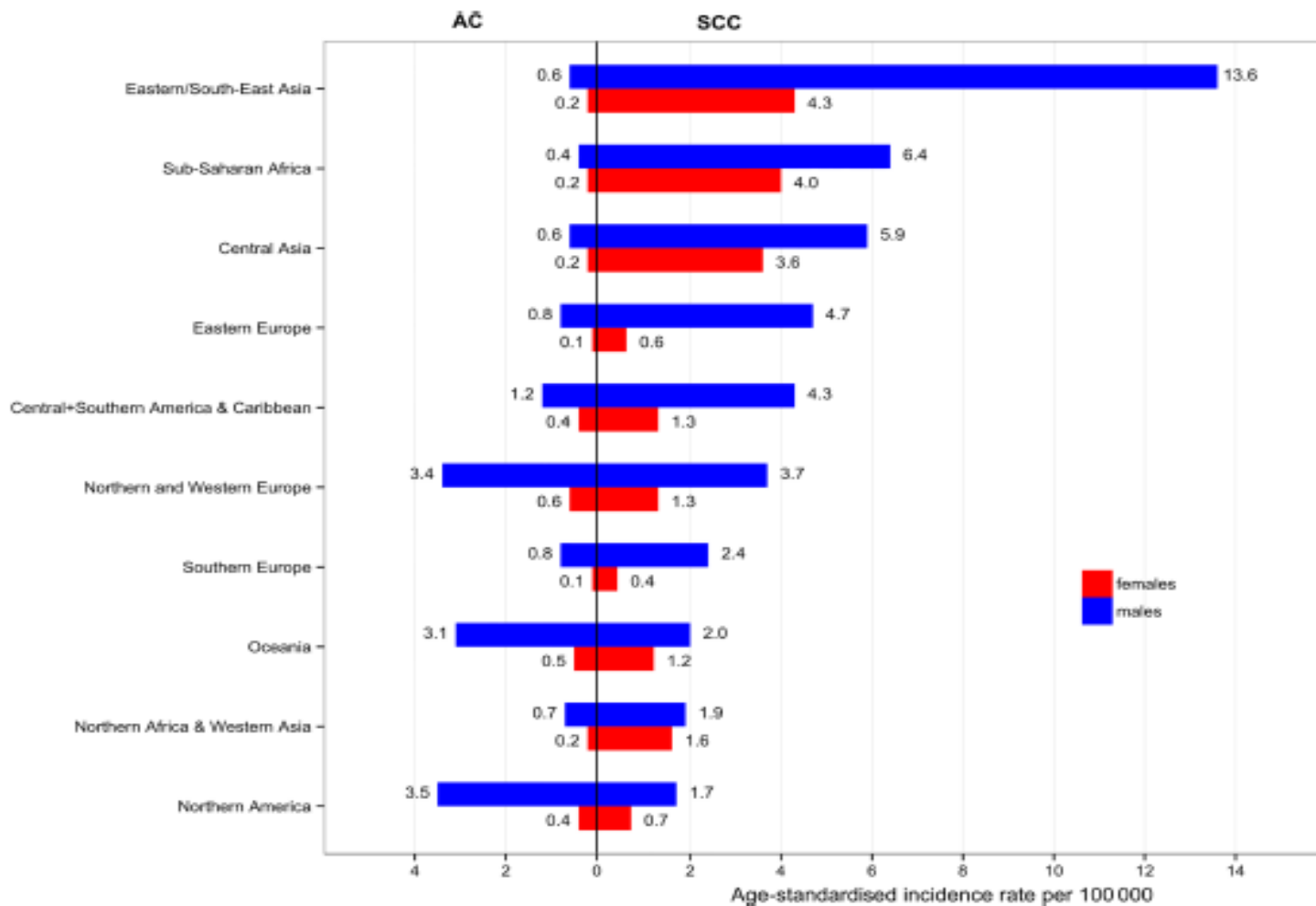


Figure 3 Age-standardised incidence rate per 100 000 of oesophageal cancer by histological subtype, region and sex. AC, adenocarcinoma; SCC, squamous cell carcinoma.



ΜΕΤΑΒΟΛΕΣ ΕΠΙΠΤΩΣΗΣ – USA - SWEDEN

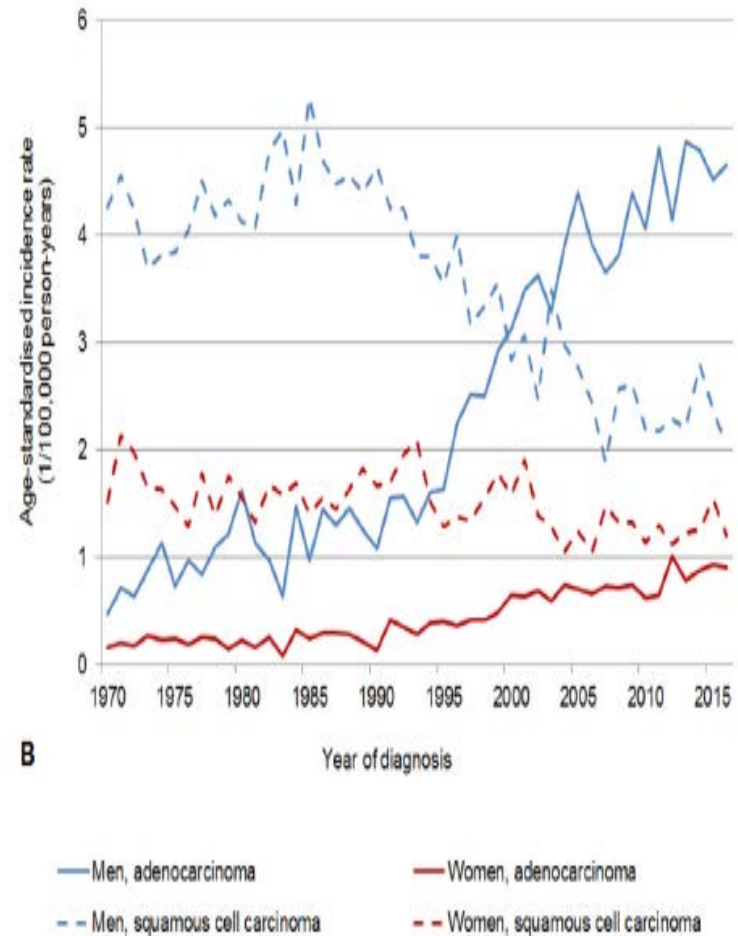
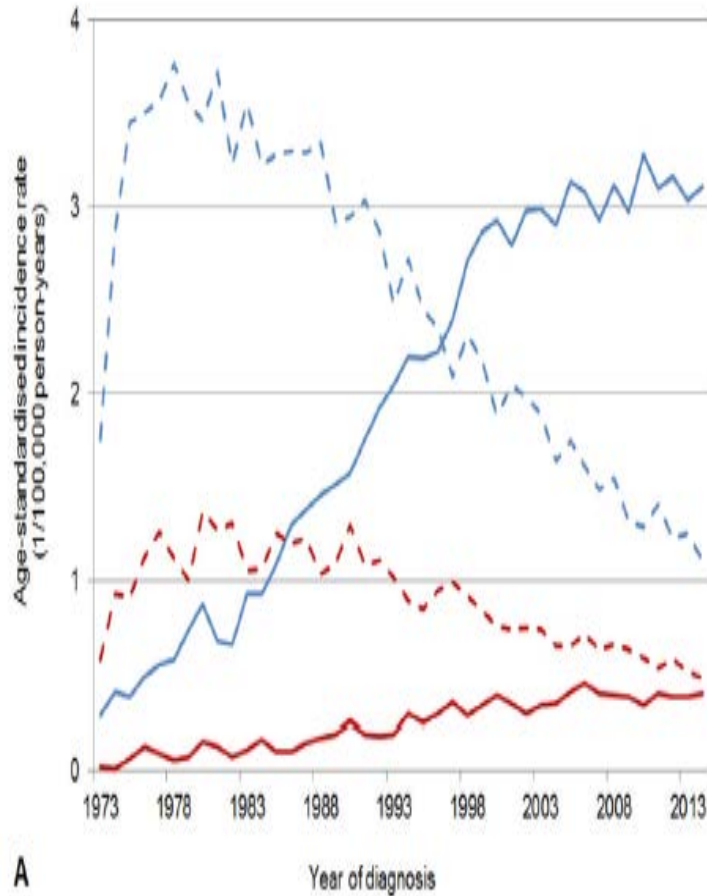


Fig. 1. Incidence trends of oesophageal squamous cell carcinoma and oesophageal adenocarcinoma in the United States (A) and in Sweden (B). Data sources: the Surveillance, Epidemiology, and End Results (SEER), SEER 9 registries research data, November 2017 submission; and the Swedish Cancer Registry.



ΚΑΡΚΙΝΟΣ ΟΙΣΟΦΑΓΟΥ

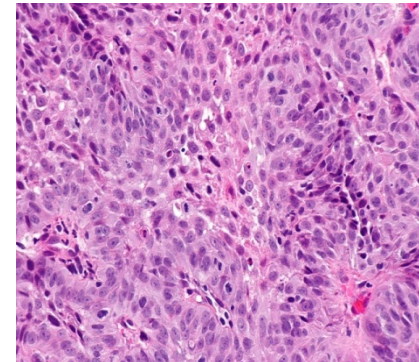
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ΠΑΡΑΓΟΝΤΕΣ ΚΙΝΔΥΝΟΥ

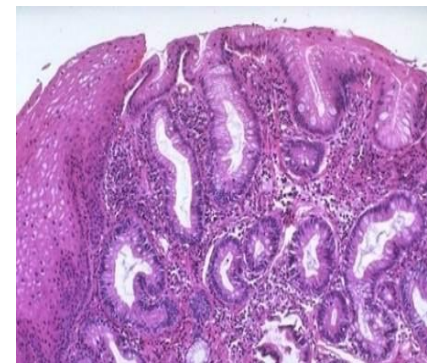
- **ESOPHAGEAL SQUAMOUS CELL CARCINOMA (ESCC)**

- ✓ TOBACCO
- ✓ ALCOHOL
- ✓ OTHERS (SOCIOECONOMIC, DIETARY, GENETIC)



- **ESOPHAGEAL ADENOCARCINOMA (EAC)**

- ✓ TOBACCO
- ✓ GERD
- ✓ OBESITY
- ✓ BARRETT'S
- ✓ OTHERS (SOCIOECONOMIC, DIETARY, GENETIC)





Socioeconomic Factors and Risk of Esophageal Adenocarcinoma: A Nationwide Swedish Case-Control Study

Catarina Jansson,¹ Anna L.V. Johansson,¹ Olof Nyren,¹ and Jesper Lagergren^{1,2}

Departments of ¹Medical Epidemiology and Biostatistics and ²Surgical Sciences, Karolinska Institutet, Stockholm, Sweden

Table 4. Risk of esophageal SCC in relation to socioeconomic factors

Socioeconomic factor	Cases/controls,* 157/792	OR ¹ (95% CI)	OR ² (95% CI)
SES			
Professionals	9/99	1.0 (Reference)	1.0 (Reference)
Intermediate nonmanual employees	24/134	1.8 (0.8-4.1)	2.2 (0.9-5.2)
Assistant nonmanual employees	20/101	1.8 (0.7-4.1)	1.9 (0.8-4.8)
Skilled manual workers	46/158	3.6 (1.7-7.7)	3.9 (1.7-8.9)
Unskilled/semiskilled manual workers	40/190	2.1 (1.0-4.7)	2.1 (0.9-4.9)
Self-employed	18/110	1.9 (0.8-4.5)	2.1 (0.8-5.1)
P⁵		0.01	0.02
Education (y)			
High (≥15)	20/155	1.0 (Reference)	1.0 (Reference)
Medium (10-12)	26/157	1.3 (0.7-2.5)	1.3 (0.7-2.6)
Low (≤9)	111/480	2.1 (1.2-3.5)	2.0 (1.1-3.6)
P		0.02	0.03



REVIEW

Smoking Cessation and Risk of Esophageal Cancer by Histological Type: Systematic Review and Meta-analysis

Qiao-Li Wang, Shao-Hua Xie, Wen-Tao Li, Jesper Lagergren

Affiliations of authors: Upper Gastrointestinal Surgery, Department of Molecular Medicine and Surgery, Karolinska Institutet, Karolinska University Hospital, Stockholm, Sweden (QLW, SHX, WTL, JL); Jockey Club School of Public Health and Primary Care, The Chinese University of Hong Kong, Hong Kong SAR, China (WTL); Division of Cancer Studies, King's College London, London, UK (JL).

Correspondence to: Shao-Hua Xie, PhD, Upper Gastrointestinal Surgery, Department of Molecular Medicine and Surgery, Karolinska Institutet, NS 67, 2nd Floor, Stockholm 17176, Sweden (e-mail: shaohua.xie@ki.se).

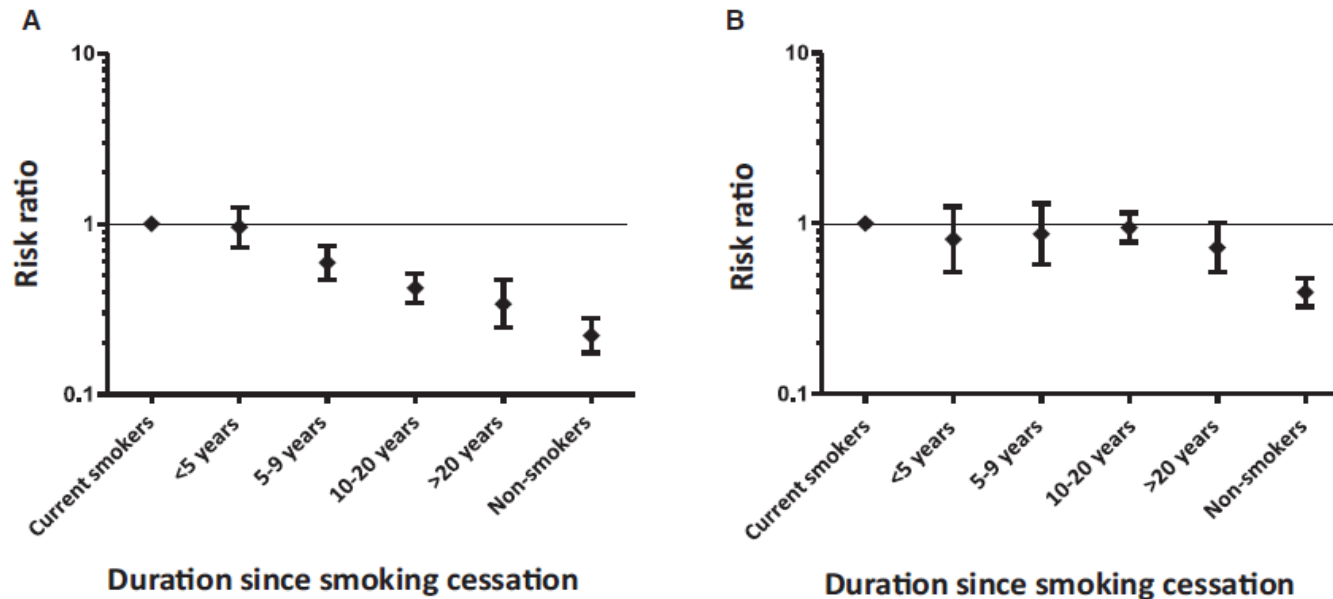


Figure 3. Risk ratio of esophageal squamous cell carcinoma and adenocarcinoma by duration since smoking cessation, using current smokers as reference. A) Effect sizes for esophageal squamous cell carcinoma. B) Effect sizes for esophageal adenocarcinoma. Error bars = 95% confidence interval.



Alcohol drinking and esophageal squamous cell carcinoma with focus on light-drinkers and never-smokers: a systematic review and meta-analysis

Farhad Islami^{1,2,3}, Veronika Fedirko¹, Irene Tramacere⁴, Vincenzo Bagnardi^{5,6}, Mazda Jenab¹, Lorenza Scotti⁵, Matteo Rota^{5,7}, Giovanni Corrao⁵, Werner Garavello^{4,8}, Joachim Schüz¹, Kurt Straif¹, Eva Negri⁴, Paolo Boffetta^{9,10} and Carlo La Vecchia^{4,11}

Table 1. Summary statistics for the association between alcohol consumption and esophageal squamous cell carcinoma

Subgroups by amount of alcohol intake ¹	No. of studies	I ² statistics (%)	p-value for I ² statistics	Random effects RR (95% CI)
Light intake				
Overall analysis	26	56.2	<0.001	1.31 (1.10–1.57)
Prospective studies	8	81.1	<0.001	1.35 (0.92–1.98)
More precise estimates ²	19	73.2	<0.001	1.32 (0.90–1.60)
Population-based controls	13	77.3	<0.001	1.16 (0.87–1.55)
Only ESCC ³	16	61.0	<0.001	1.25 (1.01–1.56)
Adjusted results ⁴	19	51.5	0.002	1.38 (1.14–1.67)
Moderate intake				
Overall analysis	47	85.3	<0.001	2.27 (1.89–2.72)
Prospective studies	12	86.7	<0.001	2.15 (1.55–2.98)
More precise estimates ²	38	87.1	<0.001	2.23 (1.84–2.71)
Population-based controls	23	85.0	<0.001	1.92 (1.49–2.47)
Only ESCC ³	27	85.8	<0.001	2.32 (1.80–2.99)
Adjusted results ⁴	28	82.8	<0.001	2.62 (2.07–3.31)
High intake				
Overall analysis	39	87.1	<0.001	4.89 (3.84–6.23)
Prospective studies	9	91.4	<0.001	3.35 (2.06–5.46)
More precise estimates ²	16	94.5	<0.001	3.35 (2.35–4.78)
Population-based controls	18	91.0	<0.001	3.77 (2.60–5.47)
Only ESCC ³	20	88.4	<0.001	5.38 (3.80–7.61)
Adjusted results ⁴	21	89.9	<0.001	5.54 (3.92–7.82)

¹Amount of daily ethanol intake was defined as: light, >0–12.5 g; moderate, >12.5 to <50 g; high, ≥50 g. The reference groups included non/occasional drinkers. ²Only studies with a standard error <0.5 (in logarithmic scale) for light and moderate intakes and with a standard error <0.3 for high intake. ³Studies that provided risk estimates for esophageal squamous cell carcinoma only. ⁴Adjusted at least for age, sex, and tobacco use.



The New England Journal of Medicine

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VOLUME 340

MARCH 18, 1999

NUMBER 11



SYMPTOMATIC GASTROESOPHAGEAL REFLUX AS A RISK FACTOR FOR ESOPHAGEAL ADENOCARCINOMA

JESPER LAGERGREN, M.D., REINHOLD BERGSTRÖM, PH.D., ANDERS LINDGREN, M.D., PH.D.,
AND OLOF NYRÉN, M.D., PH.D.

TABLE 2. SYMPTOMS OF REFLUX FIVE YEARS OR MORE BEFORE THE INTERVIEW AND THE RISK OF ESOPHAGEAL ADENOCARCINOMA, ADENOCARCINOMA OF THE GASTRIC CARDIA, AND ESOPHAGEAL SQUAMOUS-CELL CARCINOMA.*

SYMPTOMS OF REFLUX	CONTROLS (N=820)	ESOPHAGEAL ADENOCARCINOMA		ADENOCARCINOMA OF GASTRIC CARDIA		ESOPHAGEAL SQUAMOUS- CELL CARCINOMA	
		PATIENTS (N=189)	ADJUSTED ODDS RATIO (95% CI)	PATIENTS (N=262)	ADJUSTED ODDS RATIO (95% CI)	PATIENTS (N=167)	ADJUSTED ODDS RATIO (95% CI)
	no. (%)			no. (%)		no. (%)	
Heartburn, regurgitation, or both at least once a week							
No	685 (84)	76 (40)	1.0	187 (71)	1.0	142 (85)	1.0
Yes	135 (16)	113 (60)	7.7 (5.3–11.4)	75 (29)	2.0 (1.4–2.9)	25 (15)	1.1 (0.7–1.9)
Heartburn, regurgitation, or both at night at least once a week							
No	754 (92)	88 (47)	1.0	217 (83)	1.0	157 (94)	1.0
Yes	66 (8)	101 (53)	10.8 (7.0–16.7)	45 (17)	2.4 (1.5–3.8)	10 (6)	0.9 (0.4–2.0)

*In the multivariate logistic-regression model, adjustments were made for age, sex, socioeconomic status, body-mass index, tobacco smoking, alcohol use, intake of fruit and vegetables, energy intake, work in a stooped posture, physical activity at work, and physical activity during leisure time. Subjects without symptoms served as the reference group. CI denotes confidence interval.



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AND OLOF NYRÉN, M.D., PH.D.

**TABLE 3. ASSOCIATION OF SYMPTOMS OF REFLUX WITH DISEASE AFTER THE SUCCESSIVE
INCLUSION OF POTENTIAL CONFOUNDING VARIABLES IN THE MULTIVARIATE ANALYSIS.***

CONFOUNDING VARIABLE	ESOPHAGEAL ADENOCARCINOMA	GASTRIC CARDIA ADENOCARCINOMA	ESOPHAGEAL SQUAMOUS-CELL CARCINOMA
	odds ratio (95% confidence interval)		
Unadjusted	7.5 (5.4–10.6)	2.0 (1.5–2.8)	0.9 (0.6–1.4)
Age	7.6 (5.3–10.7)	2.0 (1.4–2.8)	0.9 (0.6–1.5)
Sex	7.5 (5.3–10.6)	2.0 (1.4–2.8)	1.0 (0.6–1.6)
Body-mass index	7.1 (4.9–10.2)	1.9 (1.4–2.7)	1.0 (0.6–1.6)
Tobacco smoking	7.1 (4.9–10.2)	2.0 (1.4–2.7)	1.1 (0.7–1.8)
Alcohol use	7.6 (5.2–11.0)	2.0 (1.4–2.8)	1.2 (0.7–2.1)
Educational level	7.6 (5.2–11.0)	2.0 (1.4–2.8)	1.2 (0.7–2.0)
Energy intake	7.6 (5.2–11.1)	2.0 (1.4–2.8)	1.2 (0.7–2.0)
Intake of fruit and vegetables	7.6 (5.2–11.1)	2.0 (1.4–2.8)	1.2 (0.7–2.0)
Work in a stooped posture	7.6 (5.2–11.1)	2.0 (1.4–2.8)	1.2 (0.7–2.0)
Physical activity at work	7.8 (5.3–11.4)	2.0 (1.4–2.9)	1.1 (0.7–1.9)
Physical activity during leisure time	7.7 (5.3–11.4)	2.0 (1.4–2.9)	1.2 (0.7–2.0)

*The odds ratios in each row have been adjusted for the variable noted in that row and all other variables listed above that row. Thus, the last row shows the odds ratios from the full multivariate model with 11 potential confounders included.



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AND OLOF NYRÉN, M.D., PH.D.

TABLE 4. FREQUENCY, SEVERITY, AND DURATION OF SYMPTOMS OF REFLUX FIVE YEARS OR MORE BEFORE THE INTERVIEW.*

VARIABLE	CONTROLS (N=820)	ESOPHAGEAL ADENOCARCINOMA		ADENOCARCINOMA OF GASTRIC CARDIA		ESOPHAGEAL SQUAMOUS- CELL CARCINOMA	
		PATIENTS (N=189)	ADJUSTED ODDS RATIO (95% CI)	PATIENTS (N=262)	ADJUSTED ODDS RATIO (95% CI)	PATIENTS (N=167)	ADJUSTED ODDS RATIO (95% CI)
	no. (%)			no. (%)		no. (%)	
Frequency of reflux symptoms							
No symptoms	685 (84)	76 (40)	1.0	187 (71)	1.0	142 (85)	1.0
1 time per week	95 (12)	37 (20)	5.1 (2.8–9.4)	30 (11)	2.0 (1.1–3.6)	9 (5)	0.9 (0.4–2.4)
2 to 3 times per week	16 (2)	35 (19)	6.3 (3.8–10.3)	27 (10)	1.9 (1.2–3.1)	10 (6)	1.2 (0.6–2.5)
>3 times per week	24 (3)	41 (22)	16.7 (8.7–28.3)	18 (7)	2.3 (1.2–4.3)	6 (4)	1.4 (0.5–3.7)
Reflux-symptom score†							
No symptoms	685 (84)	76 (40)	1.0	187 (71)	1.0	142 (85)	1.0
1–2 points	58 (7)	10 (5)	1.4 (0.7–3.0)	27 (10)	1.7 (1.0–2.9)	14 (8)	1.7 (0.8–3.5)
2.5–4 points	43 (5)	39 (21)	8.1 (4.7–16.1)	21 (8)	1.8 (1.0–3.2)	5 (3)	0.6 (0.2–1.7)
4.5–6.5 points	34 (4)	64 (34)	20.0 (11.6–34.6)	27 (10)	2.8 (1.6–5.0)	6 (4)	1.1 (0.4–3.0)
Duration of reflux symptoms							
No symptoms	685 (84)	76 (40)	1.0	187 (71)	1.0	142 (85)	1.0
<12 yr	41 (5)	31 (16)	7.5 (4.2–13.5)	19 (7)	1.6 (0.9–2.9)	10 (6)	1.6 (0.7–3.7)
12–20 yr	67 (8)	42 (22)	5.2 (3.1–8.6)	34 (13)	1.8 (1.1–2.9)	10 (6)	0.9 (0.4–1.9)
>20 yr	27 (3)	40 (21)	16.4 (8.3–28.4)	22 (8)	3.3 (1.8–6.3)	5 (3)	1.2 (0.4–3.7)



Body mass index in relation to oesophageal and oesophagogastric junction adenocarcinomas: a pooled analysis from the International BEACON Consortium

Cathrine Hoyo,^{1*†} Michael B Cook,^{2†} Farin Kamangar,^{2,3} Neal D Freedman,² David C Whiteman,^{4,5} Leslie Bernstein,⁶ Linda M Brown,⁷ Harvey A Risch,⁸ Weimin Ye,⁹ Linda Sharp,¹⁰ Anna H Wu,¹¹ Mary H Ward,² Alan G Casson,¹² Liam J Murray,¹³ Douglas A Corley,¹⁴ Olof Nyrén,⁹ Nirmala Pandeya,^{4,5} Thomas L Vaughan,¹⁵ Wong-Ho Chow² and Marilie D Gammon¹⁶

Table 2 Adjusted summary odds ratios and 95% confidence intervals for the association between body mass index and adenocarcinomas of the oesophagus and oesophagogastric junction among all subjects (men and women combined)

BMI (kg/m ²)	OA						OGJA						All adenocarcinomas					
	Controls (n)	Cases (n)	OR	95% CI	I ²	N	Controls (n)	Cases (n)	OR	95% CI	I ²	N	Controls (n)	Cases (n)	OR	95% CI	I ²	N
<25.0	4744	577		Referent			4686	663		Referent			4777	1240		Referent		
25.0–29.9	4232	862	1.54	1.26–1.88	55	12	4147	742	1.28	1.13–1.45	0	10	4232	1604	1.41	1.24–1.60	37	12
30.0–34.9	1109	331	2.39	1.86–3.06	42	12	1081	304	2.08	1.75–2.47	0	10	1109	635	2.23	1.83–2.71	41	12
35.0–39.9	273	86	2.79	1.89–4.12	23	11	266	85	2.36	1.75–3.17	0	9	273	171	2.40	1.89–3.04	0	11
≥40	94	41	4.76	2.96–7.66	0	9	91	28	3.07	1.89–4.99	0	8	99	69	3.65	2.50–5.34	0	10
Continuous	10 481	1897	1.09	1.06–1.12	76	12	10 295	1822	1.07	1.05–1.09	54	10	10 481	3719	1.08	1.06–1.10	75	12

Results were adjusted for age (categorical: <50, 50–59, 60–69, ≥70 years), gender, pack-years of smoking (categorical: <15, 15 to <30, 30 to <45, ≥45), education (study-specific) and other study-specific adjustment variables (e.g. study centre, where applicable). Summary odds ratios and 95% confidence intervals were obtained from random-effects models. All 12 studies were included for analysis unless otherwise specified.

I² estimates variability in results across studies.

OA=oesophageal adenocarcinoma; OGJA=oesophagogastric junction adenocarcinoma; AA=all adenocarcinomas (OA and OGJA); BMI=body mass index; OR=odds ratio; N=number of studies in the analysis.



Body-mass index and incidence of cancer: a systematic review and meta-analysis of prospective observational studies



Andrew G Renehan, Margaret Tyson, Matthias Egger, Richard F Heller, Marcel Zwahlen

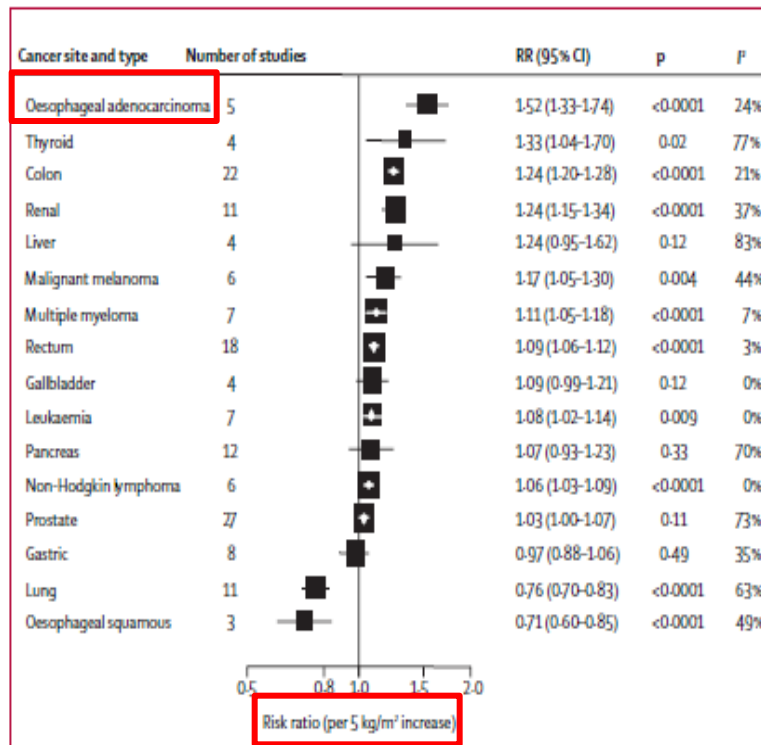


Figure 3: Summary risk estimates by cancer sites in men

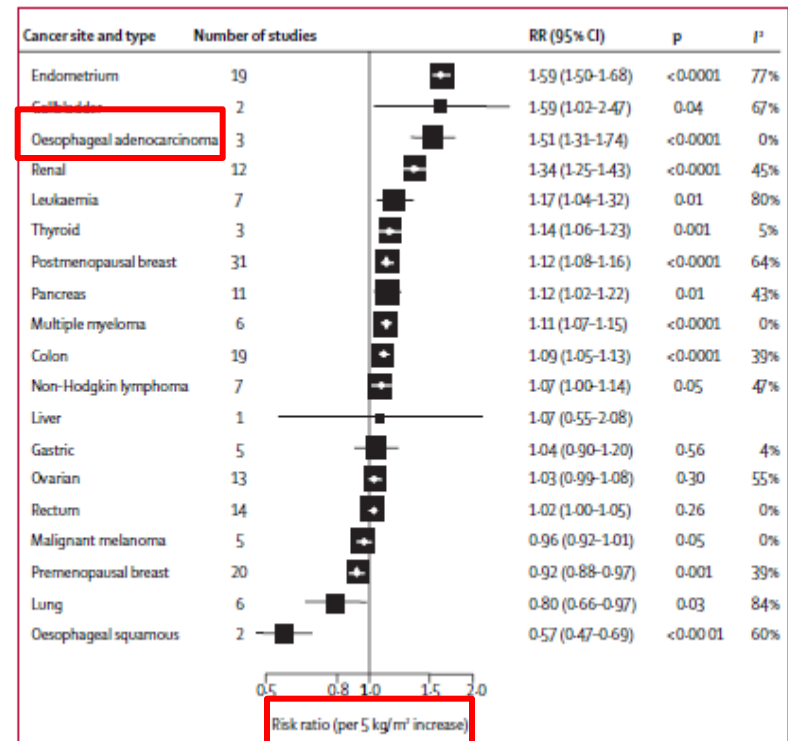


Figure 4: Summary risk estimates by cancer sites in women

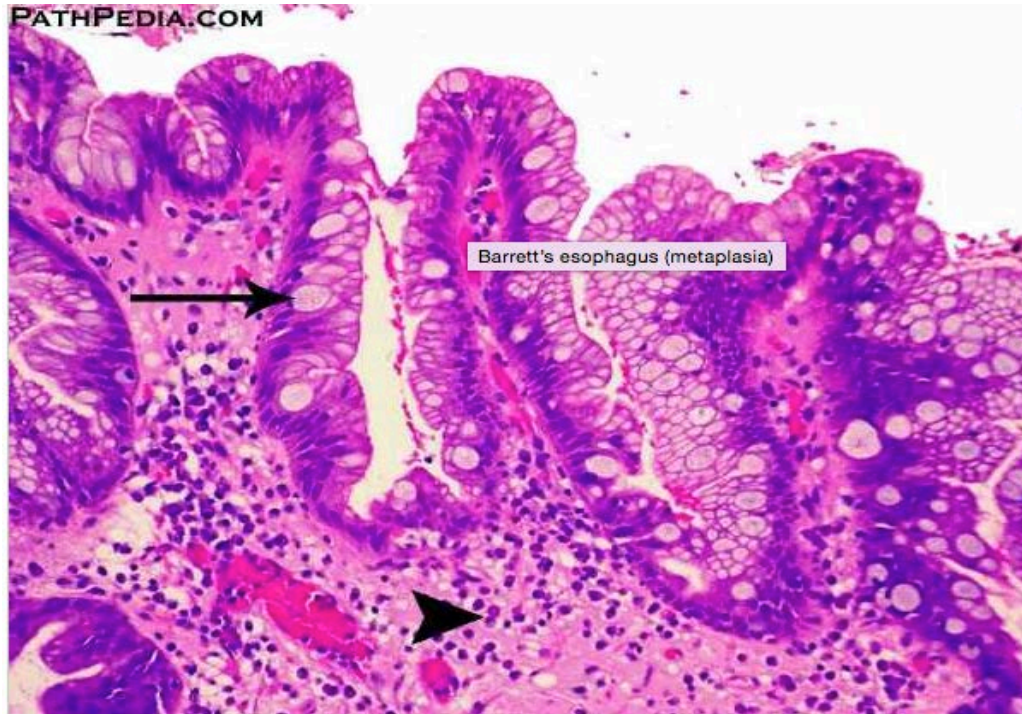


ΚΑΡΚΙΝΟΣ ΟΙΣΟΦΑΓΟΥ

ΟΙΣΟΦΑΓΟΣ BARRETT



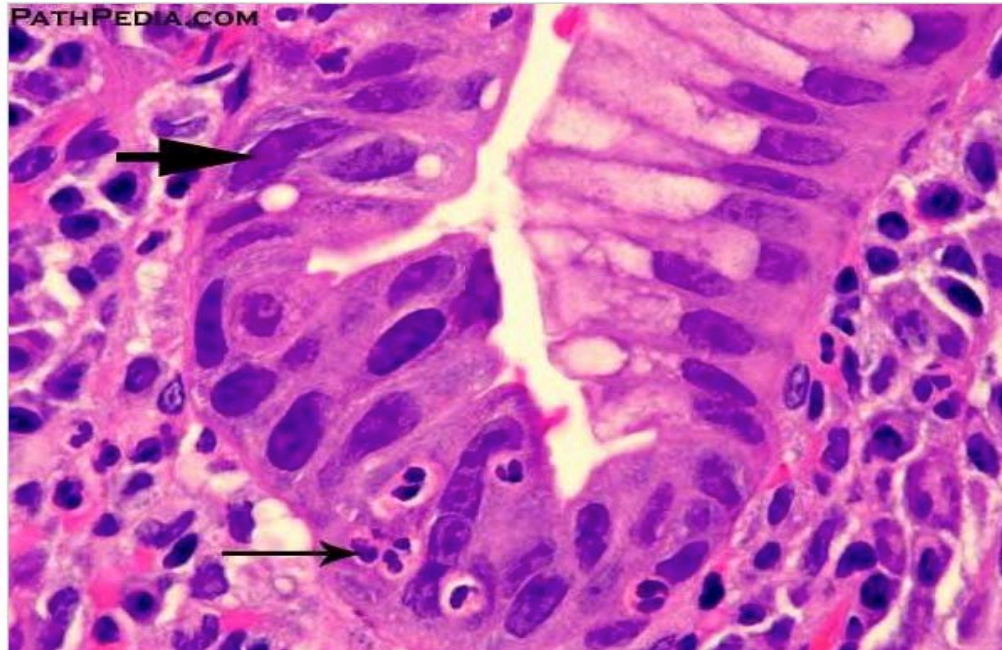
INTESTINAL METAPLASIA



[BARRETT'S METAPLASIA]. This photomicrograph shows surface glandular epithelium with tall columnar cells punctuated by round to oval goblet cells (long arrow). The columnar epithelium resembles intestinal epithelium but is not identical in nature. It is unclear why this metaplasia occurs but long-standing gastro-esophageal reflux disease (GERD) is present in all patients. Barrett's metaplasia is also characterized by universal presence of chronic inflammation (lower arrowhead)



INDEFINITE FOR DYSPLASIA



[BARRETT'S METAPLASIA]. Inflammation present in Barrett's metaplasia may lead to nuclear atypia of varying degrees. In some cases the nuclear atypia simulates dysplasia but histologic features diagnostic of dysplasia are not seen. Also, this is one area in diagnostic histopathology where consensus among pathologists to what is a low grade dysplasia in a given case is lacking. To address this issue of reactive atypia versus low-grade dysplasia a diagnosis of "indefinite for dysplasia" may be used. This case with inflammation in glands (bottom long arrow) shows loss of polarity, pseudo-stratification, mild nuclear enlargement and hyperchromasia, and distinct nucleoli may be called "nuclear atypia indefinite for dysplasia."



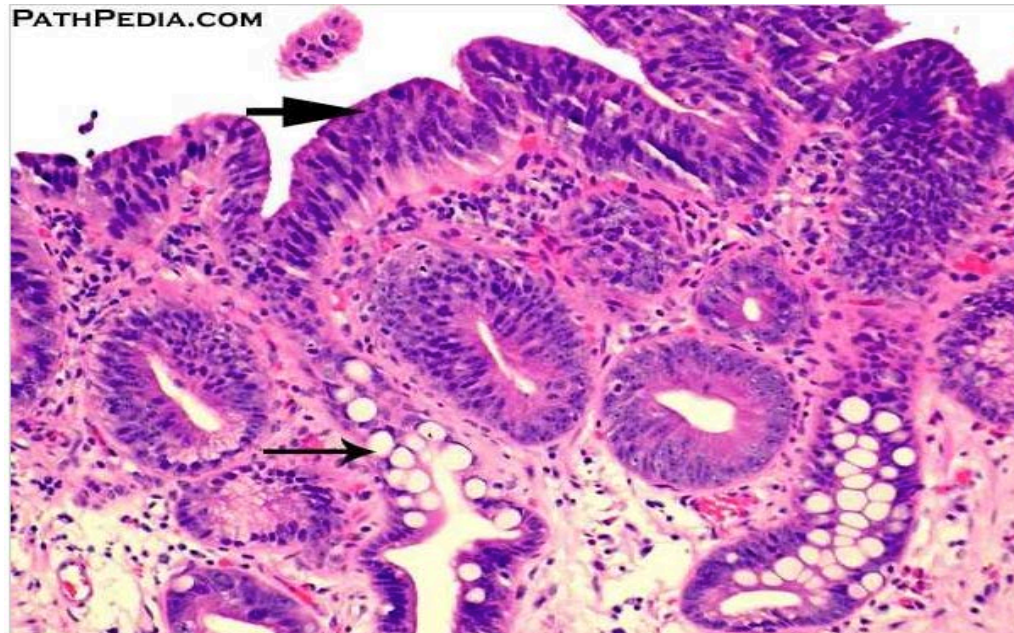
LOW GRADE DYSPLASIA



[BARRETT'S METAPLASIA]. A low-grade dysplasia in Barrett's esophagus, at a minimum, requires nuclear enlargement (right arrow), nuclear hyperchromasia, and nuclear contour irregularity. Prominent nucleoli and mitoses are common but reactive epithelium may also show mitoses and prominent nucleoli. Generally, dysplasia reaches the surface epithelium but not all biopsy specimens show surface epithelium involvement. The dysplastic epithelium in this case can be contrasted to normal glandular epithelium on the right (arrowhead).



HIGH GRADE DYSPLASIA



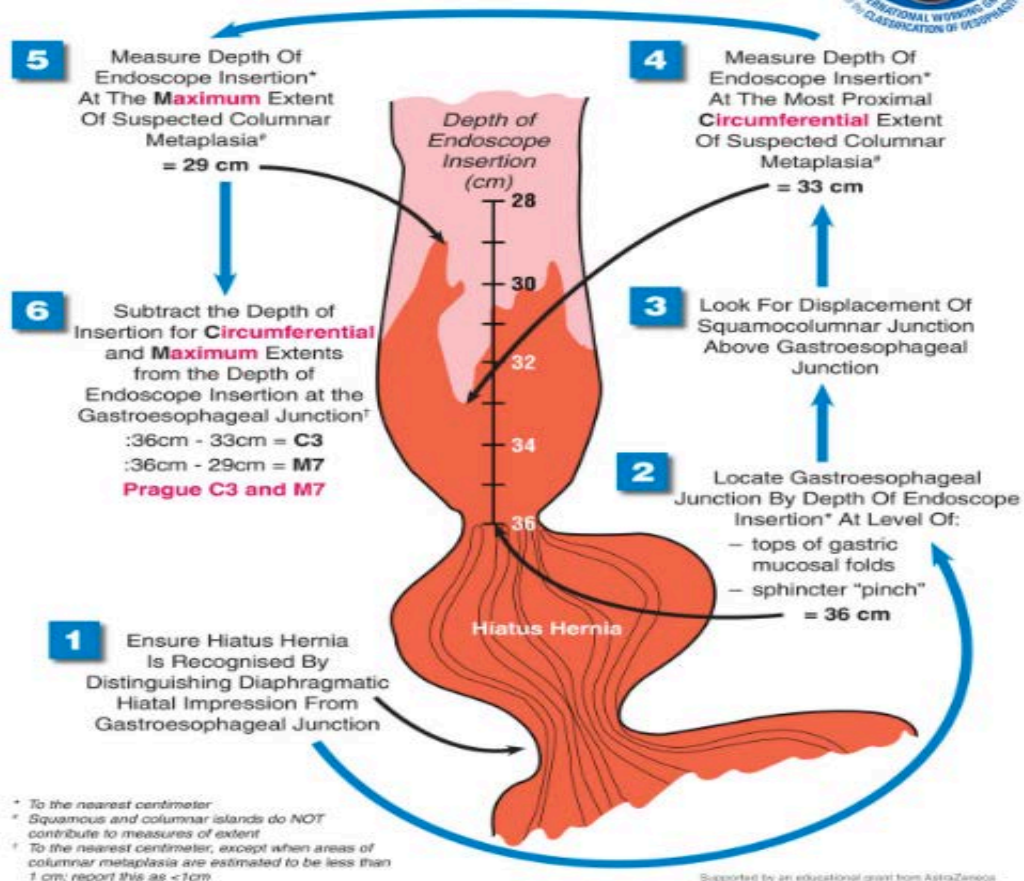
[BARRETT'S METAPLASIA]. High-grade dysplasia in Barrett's is difficult to miss because the changes stand out at low magnifications. The diagnosis of high-grade dysplasia should be made when surface epithelium shows all features of high-grade dysplasia (top arrow) such as enlarged hyperchromatic nuclei with little cytoplasm left in cells. This requirement of surface epithelium involvement help ensure that neither low grade dysplasia nor reactive atypia in deeper glands is confused with high-grade dysplasia since patients may opt for surgical removal of esophagus to rule out invasive adenocarcinoma elsewhere in the esophagus. The diagnosis of high-grade dysplasia must be confirmed by QA review to avoid diagnostic pitfalls. The physician may opt for a repeat biopsy to confirm the findings before surgical removal of the esophagus.



PRAGUE CLASSIFICATION

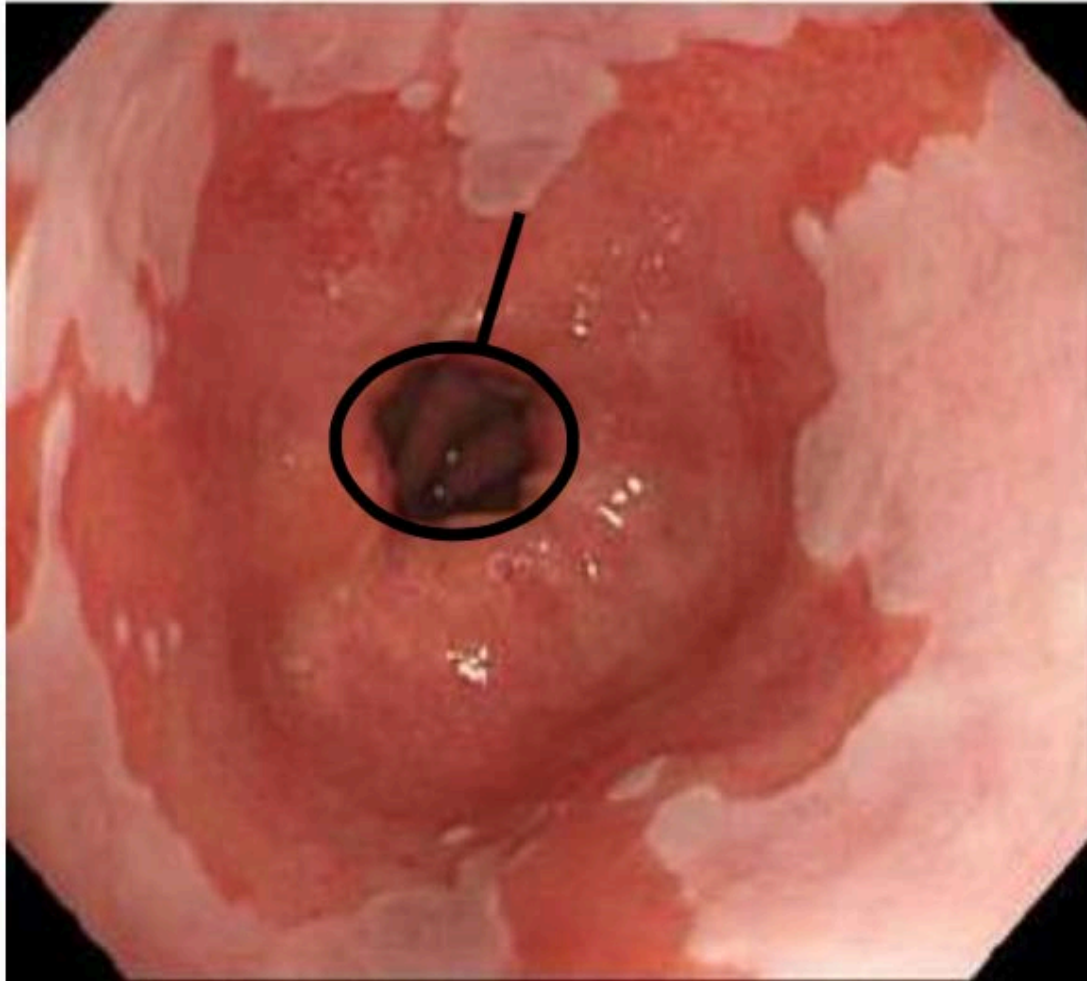
PRAGUE CRITERIA For Endoscopically Suspected Esophageal Columnar Metaplasia/Barrett's Esophagus

Developed by the Barrett's Oesophagus Subgroup of the International Working Group for the Classification of Reflux Oesophagitis (IWGCO)



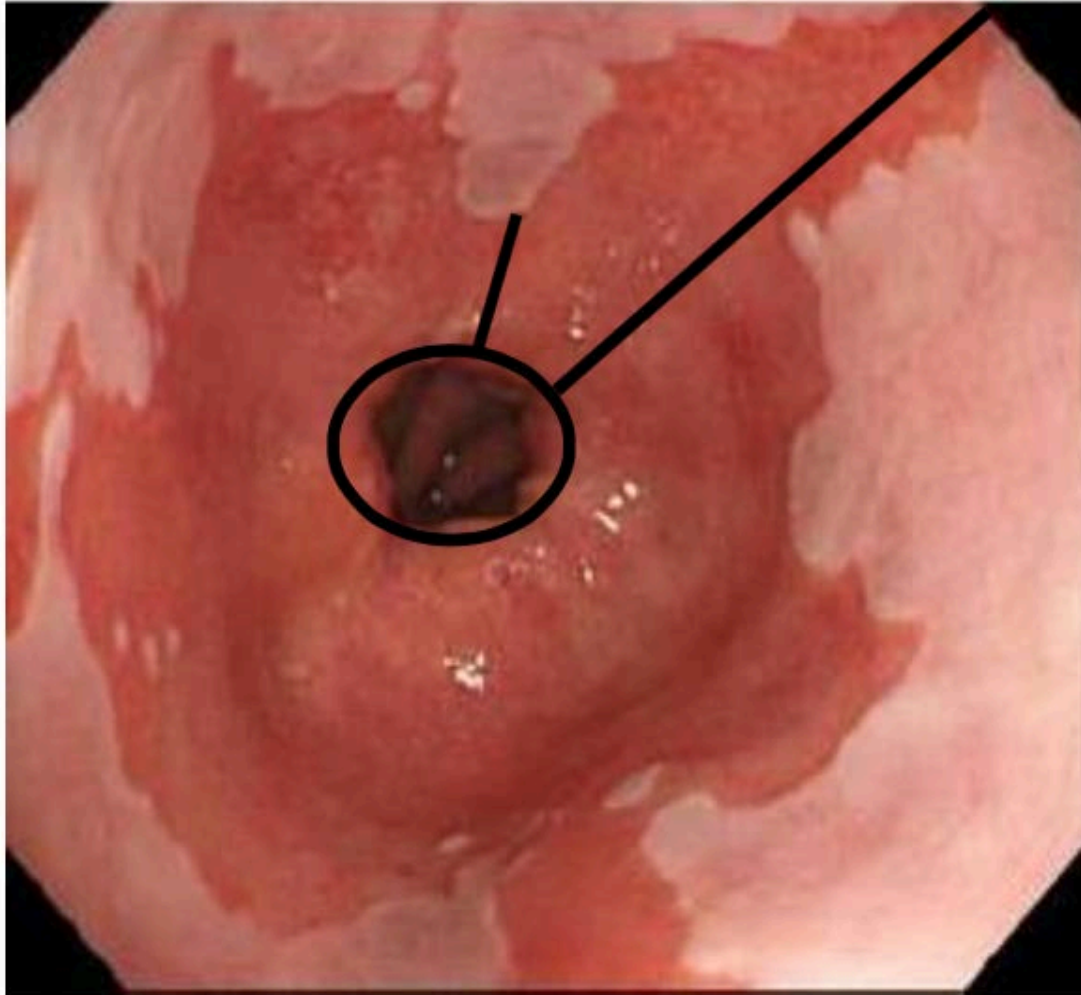


PRAGUE CLASSIFICATION - C





PRAGUE CLASSIFICATION - M



ΠΑΘΗΣΕΙΣ ΟΙΣΟΦΑΓΟΥ

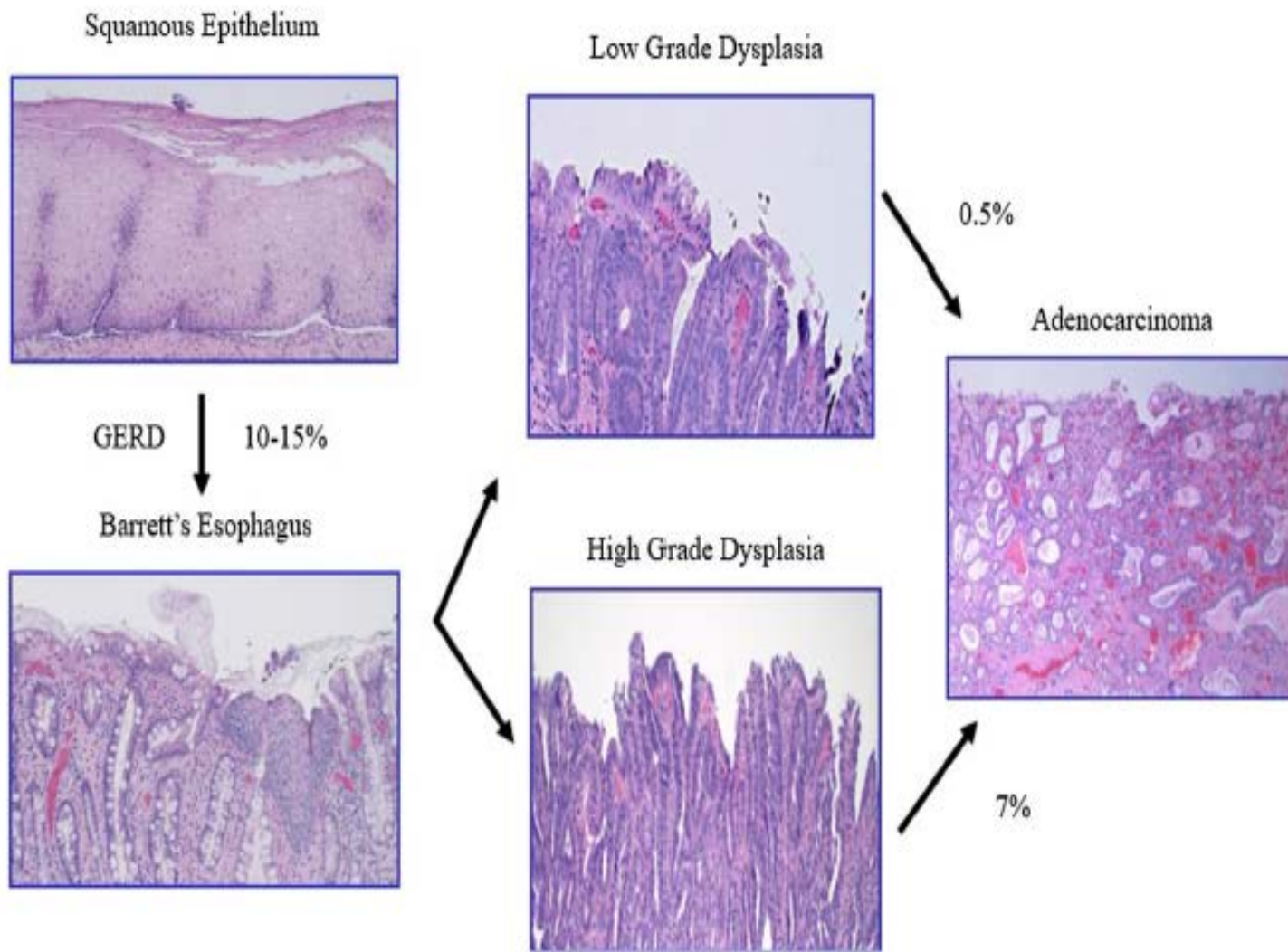


Fig. 1 Malignant transformation of Barrett's esophagus



Association Between Length of Barrett's Esophagus and Risk of High-grade Dysplasia or Adenocarcinoma in Patients Without Dysplasia

CLINICAL GASTROENTEROLOGY AND HEPATOLOGY 2013;11:1430-1436

RAJESWARI ANAPARTHY,* SRINIVAS GADDAM,* VIJAY KANAKADANDI,* BENJAMIN R. ALSOP,* NEIL GUPTA,* APRIL D. HIGBEE,* SACHIN B. WANI,* MANDEEP SINGH,* AMIT RASTOGI,* AJAY BANSAL,* BROOKS D. CASH,‡ PATRICK E. YOUNG,‡ DAVID A. LIEBERMAN,§ GARY W. FALK,|| JOHN J. VARGO,¶ PRASHANTI THOTA,¶ RICHARD E. SAMPLINER,# and PRATEEK SHARMA*

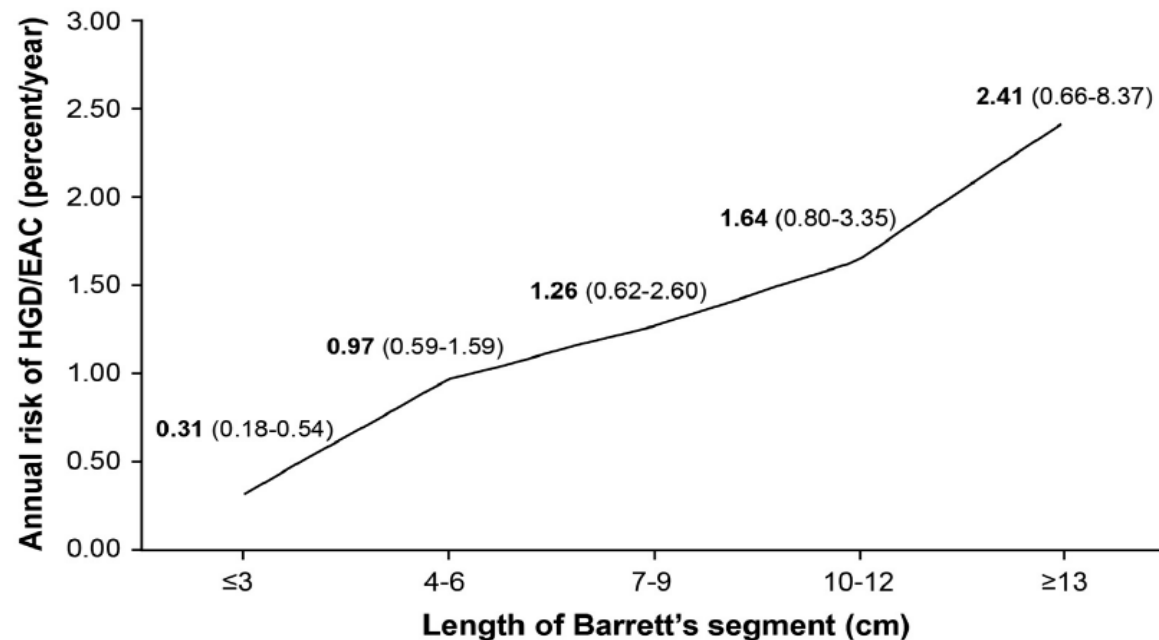


Figure 2. Annual risk of HGD/EAC (%/year) in NDBE patients.

Incident rates of HGD/EAC with 95% CI (in parentheses)



ΚΛΙΝΙΚΗ ΕΙΚΟΝΑ ΚΑΡΚΙΝΟΥ ΟΙΣΟΦΑΓΟΥ

- **ΔΥΣΦΑΓΙΑ**
- **ΔΥΣΚΑΤΑΠΟΣΙΑ**
- **ΑΙΜΟΡΡΑΓΙΑ ΠΕΠΤΙΚΟΥ (ΑΙΜΑΤΕΜΕΣΗ, ΜΕΛΑΙΝΑ ΚΕΝΩΣΗ)**
- **ΕΝΣΦΗΝΩΣΗ ΒΛΩΜΟΥ**
- **ΑΠΩΛΕΙΑ ΒΑΡΟΥΣ**
- **ΥΠΟΤΡΟΠΙΑΖΟΥΣΕΣ ΛΟΙΜΩΞΕΙΣ ΑΝΑΠΝΕΥΣΤΙΚΟΥ**
- **ΒΡΑΓΧΟΣ ΦΩΝΗΣ**

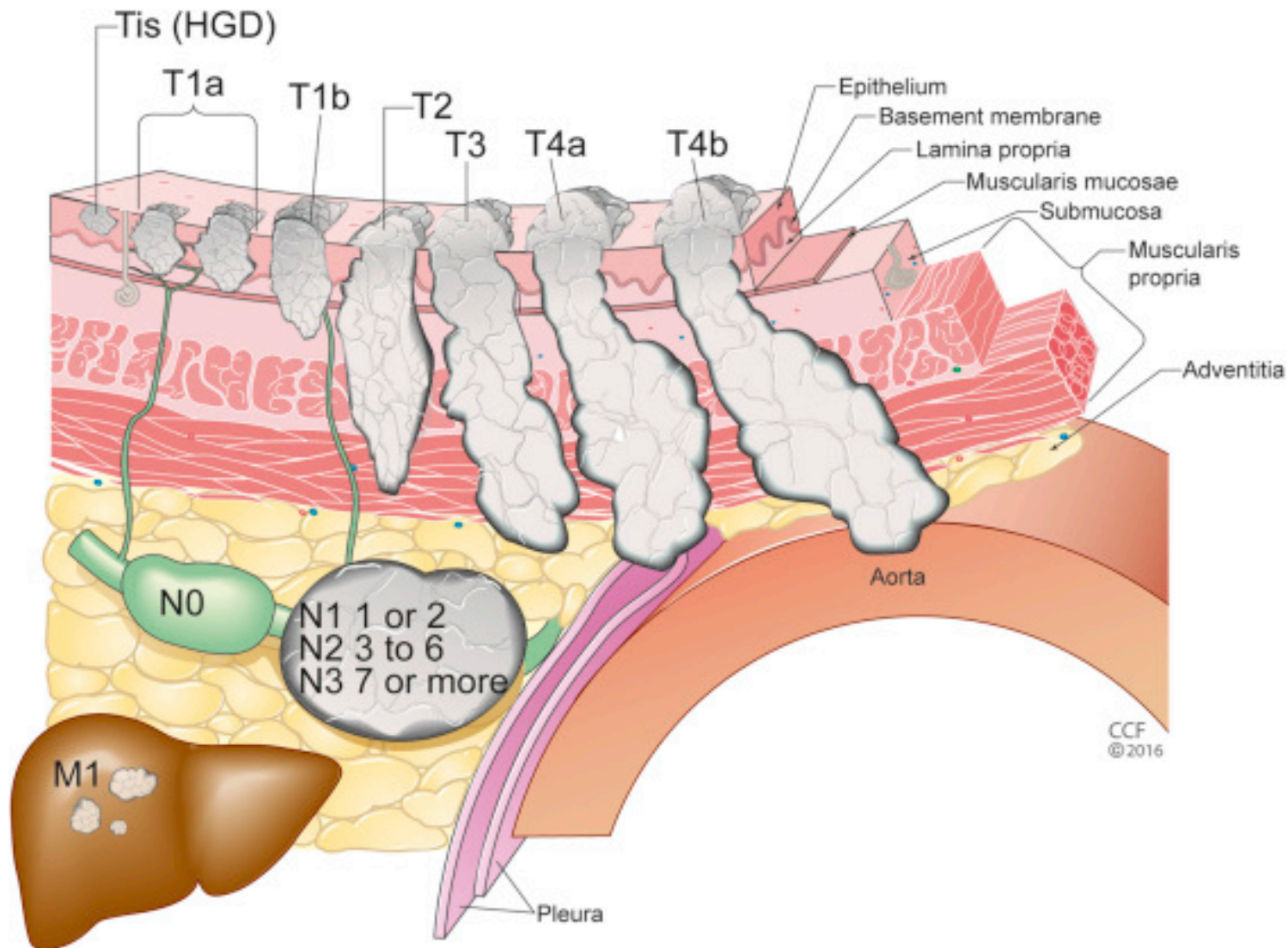


ΚΛΙΝΙΚΗ ΣΤΑΔΙΟΠΟΙΗΣΗ ΚΑΡΚΙΝΟΥ ΟΙΣΟΦΑΓΟΥ

- **ΓΑΣΤΡΟΣΚΟΠΗΣΗ** (Ακριβής εντοπισμός νόσου, σχεδιασμός επέμβασης)
- **CT Α/Κ ΚΟΙΛΙΑΣ**
- **CT ΘΩΡΑΚΟΣ**
- **MRI** (Συμπληρωματικά της CT – αποσαφήνιση ηπατικών βλαβών, διερεύνηση σχέσης όγκου με αορτή, τραχεία, βρόγχους)
- **PET – CT** (Διερεύνηση απομακρυσμένης νόσου – N staging , M)
- **EUS** (T staging , περιοισοφαγικοί λεμφαδένες)
- **CT ΤΡΑΧΗΛΟΥ** (Σε Ca μέσου, ανωτέρου οισοφάγου και σε κλινική υποψία θετικών τραχηλικών λεμφαδένων)
- **ΒΡΟΓΧΟΣΚΟΠΗΣΗ** (Διερεύνηση πιθανής διήθησης τραχείας – βρόγχων)

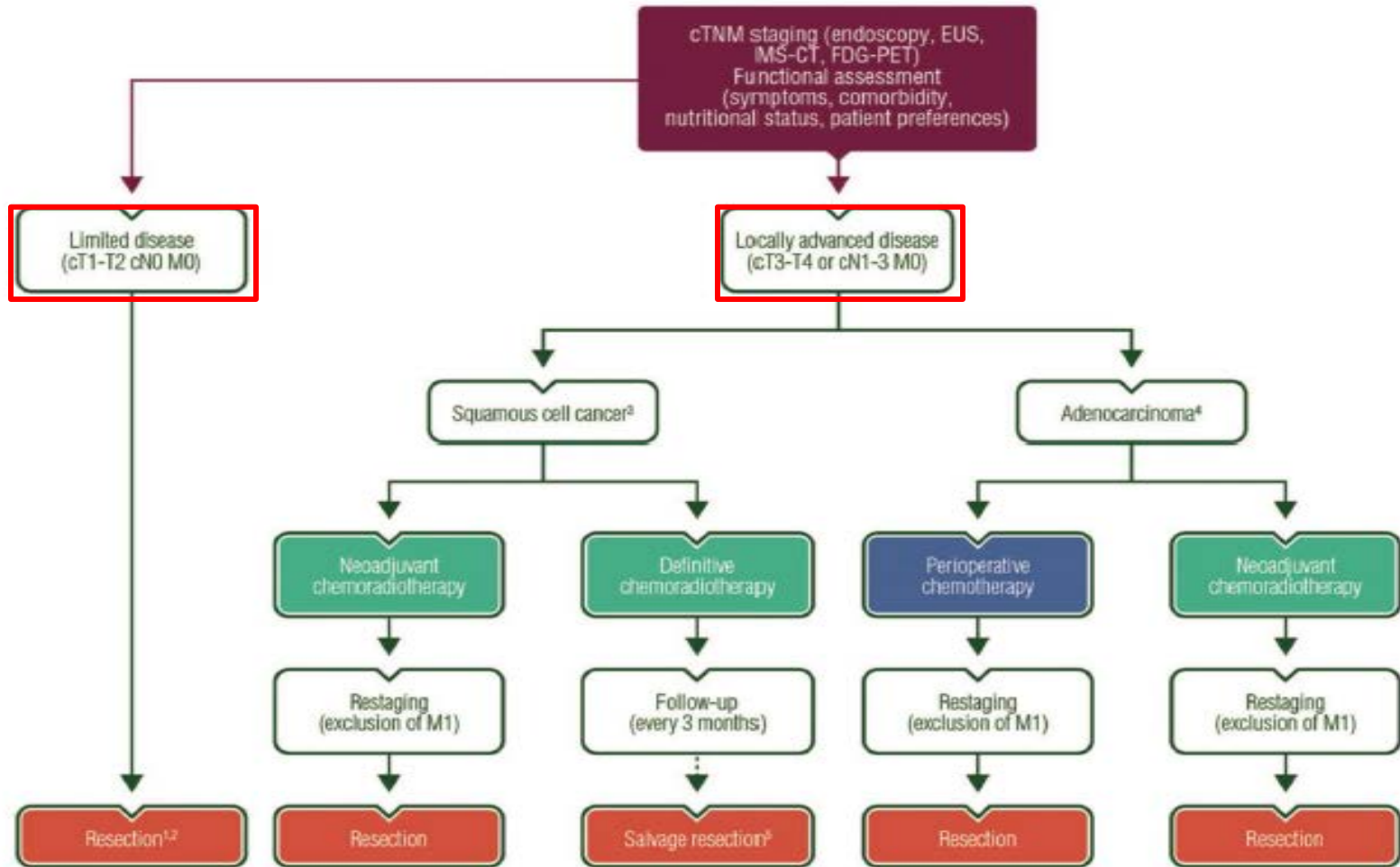


TNM STAGING - ESOPHAGEAL CANCER





ΑΛΓΟΡΙΘΜΟΣ ΑΝΤΙΜΕΤΩΠΙΣΗΣ ΚΑΡΚΙΝΟΥ ΟΙΣΟΦΑΓΟΥ



ΜΟΡΙΑΚΟΙ ΥΠΟΤΥΠΟΙ ΚΑΡΚΙΝΟΥ ΚΑΡΔΙΟΟΙΣΟΦΑΓΙΚΗΣ ΣΥΜΒΟΛΗΣ

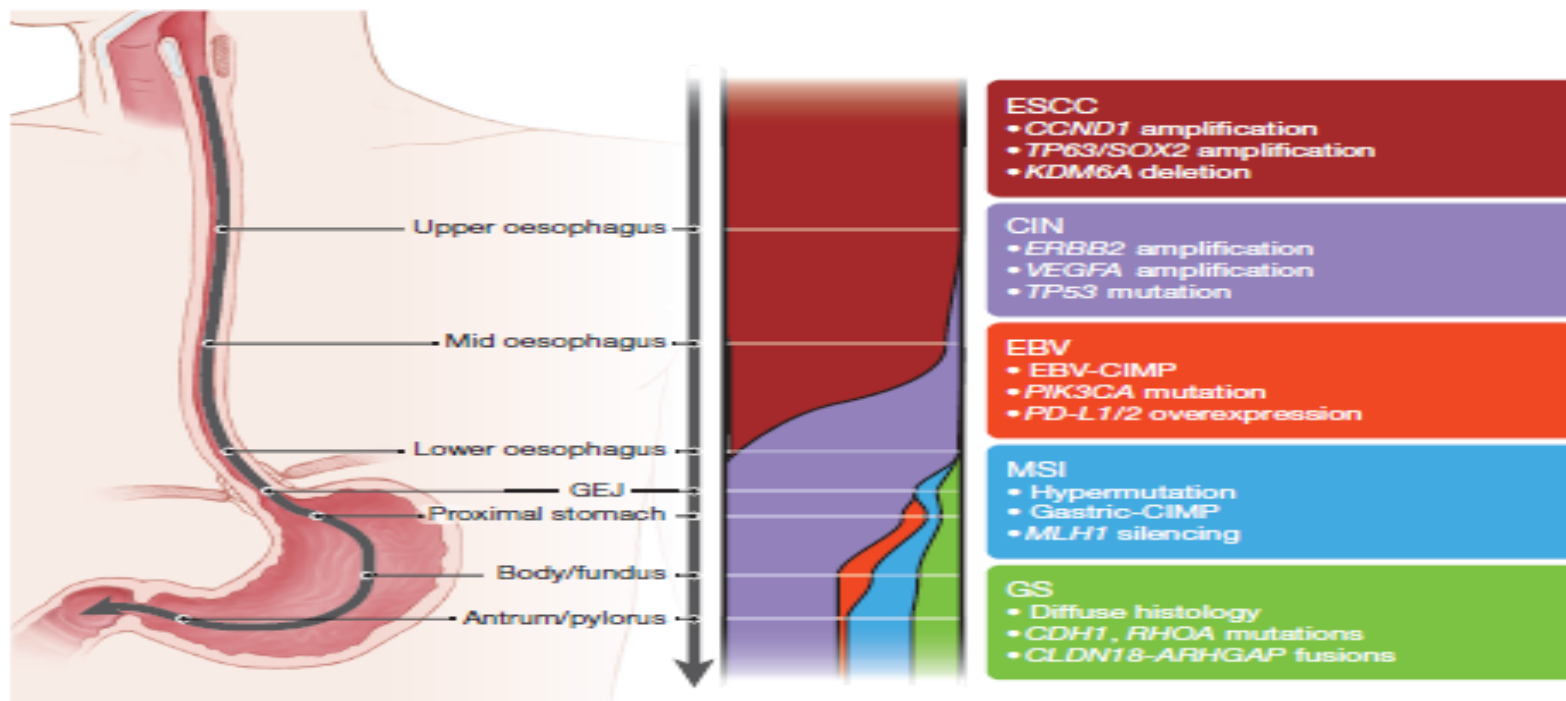


Figure 6 | Gradations of molecular subclasses of gastroesophageal carcinoma. Schematic representing shifting proportion of subtypes of gastroesophageal carcinoma from the proximal oesophagus to the distal stomach. The widths of the colour bands represent the proportion of the subtypes present within anatomic regions. Key features of subtypes are indicated in associated text.



ΤΑΞΙΝΟΜΗΣΗ ΚΑΤΑ SIEWERT

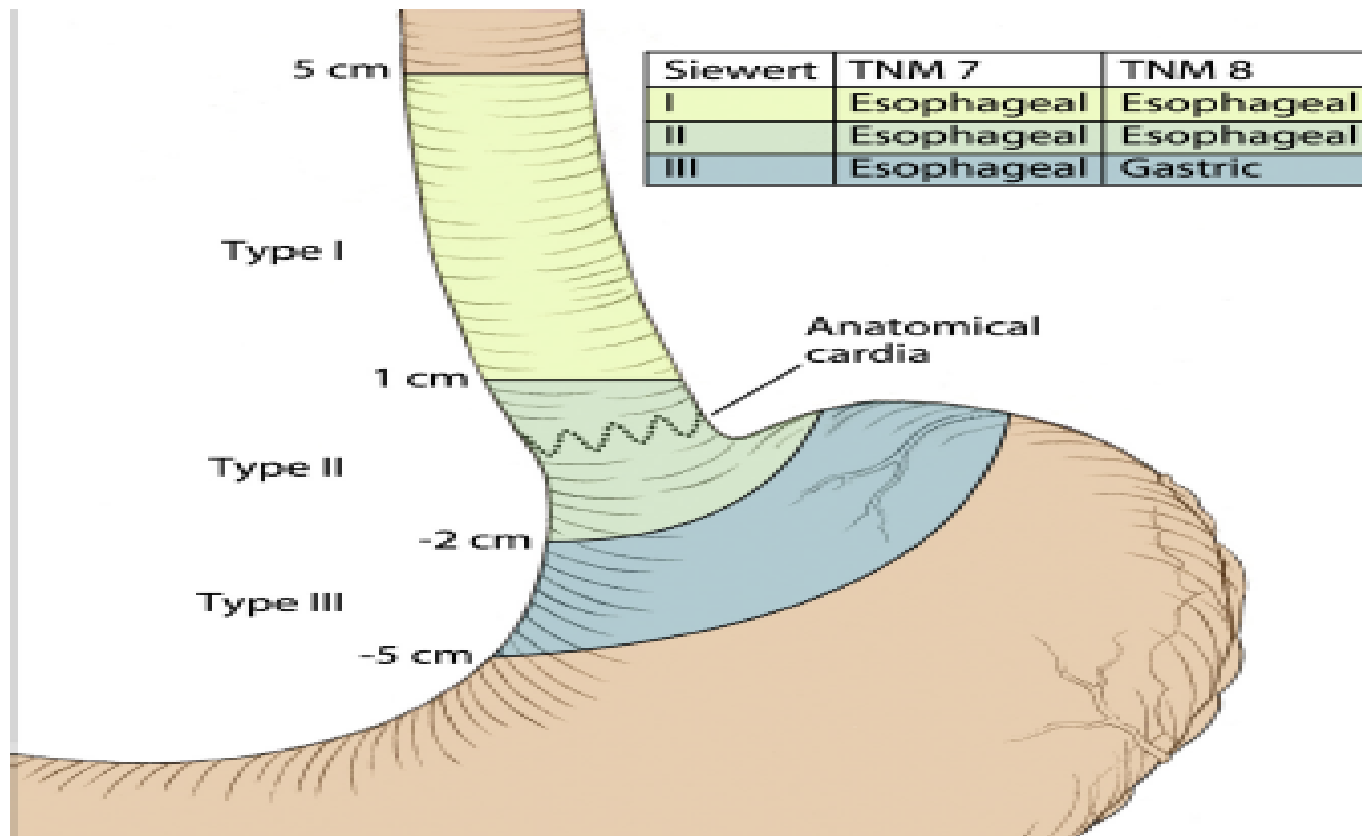


Fig. 1. Siewert classification of GEJ cancer and esophageal versus gastric cancer allocation based on the seventh and eighth editions of the AJCC TNM staging system. Data in Table from Rice TW, Blackstone EH, Rusch VW. 7th edition of the AJCC Cancer Staging Manual: esophagus and esophagogastric junction. *Ann Surg Oncol* 2010;17: 1721-4 and Rice TW, Patil DT, Blackstone EH. 8th edition AJCC/UICC staging of cancers of the esophagus and esophagogastric junction: application to clinical practice. *Ann Cardiothorac Surg* 2017;6:119-30.



ΛΕΜΦΑΔΕΝΙΚΗ ΔΙΑΣΠΟΡΑ

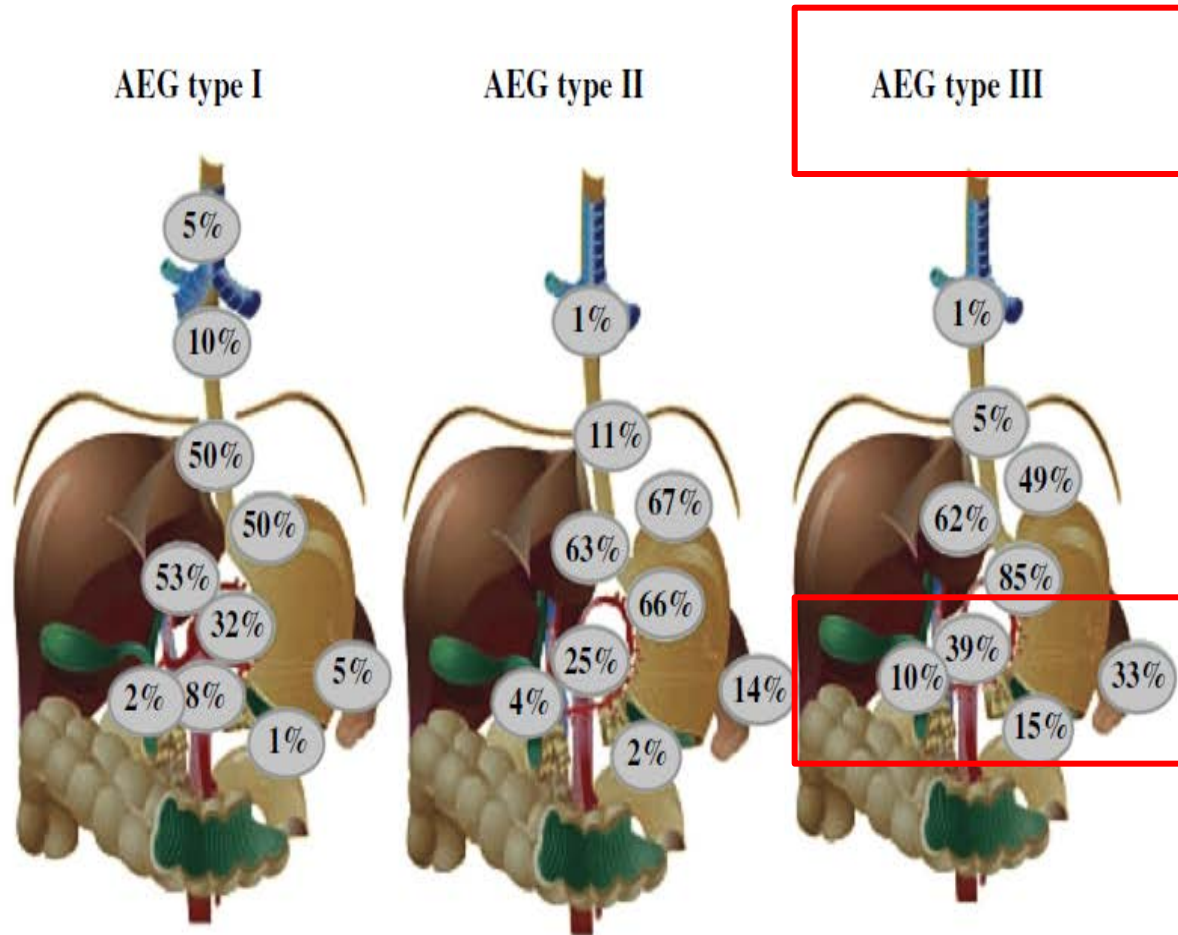


FIG. 2 Distribution of lymph node metastases in patients with resected adenocarcinoma of the esophagogastric junction (AEG) types I, II, and III. [Adapted from: Siewert JR, Stein HJ, Feith M. Adenocarcinoma of the esophago-gastric junction. *Scand J Surg*. 2006;95:260–69.]



SIEWERT III

Lancet Oncol 2011; 12: 296-305

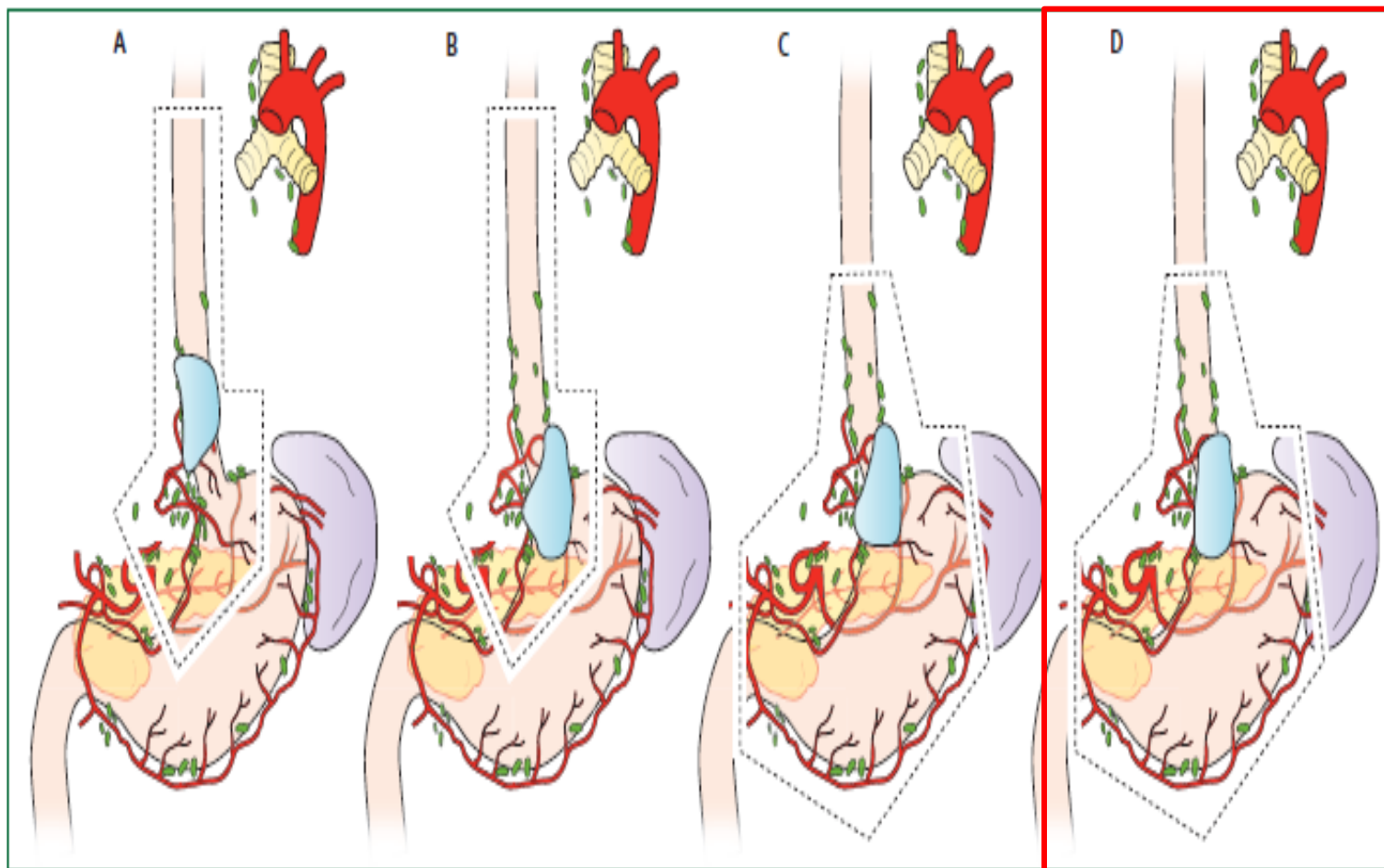


Figure 4: Schematic representation of recommended extent of surgical resection for oesophagogastric junction adenocarcinomas

Type I (A; subtotal oesophagectomy with superior polar gastrectomy), type II (subtotal oesophagectomy with superior polar gastrectomy [B] or total gastrectomy with inferior oesophagectomy [C]), and type III (D; total gastrectomy). Blue region is tumour site.



ΛΕΜΦΑΔΕΝΙΚΗ ΔΙΑΣΠΟΡΑ

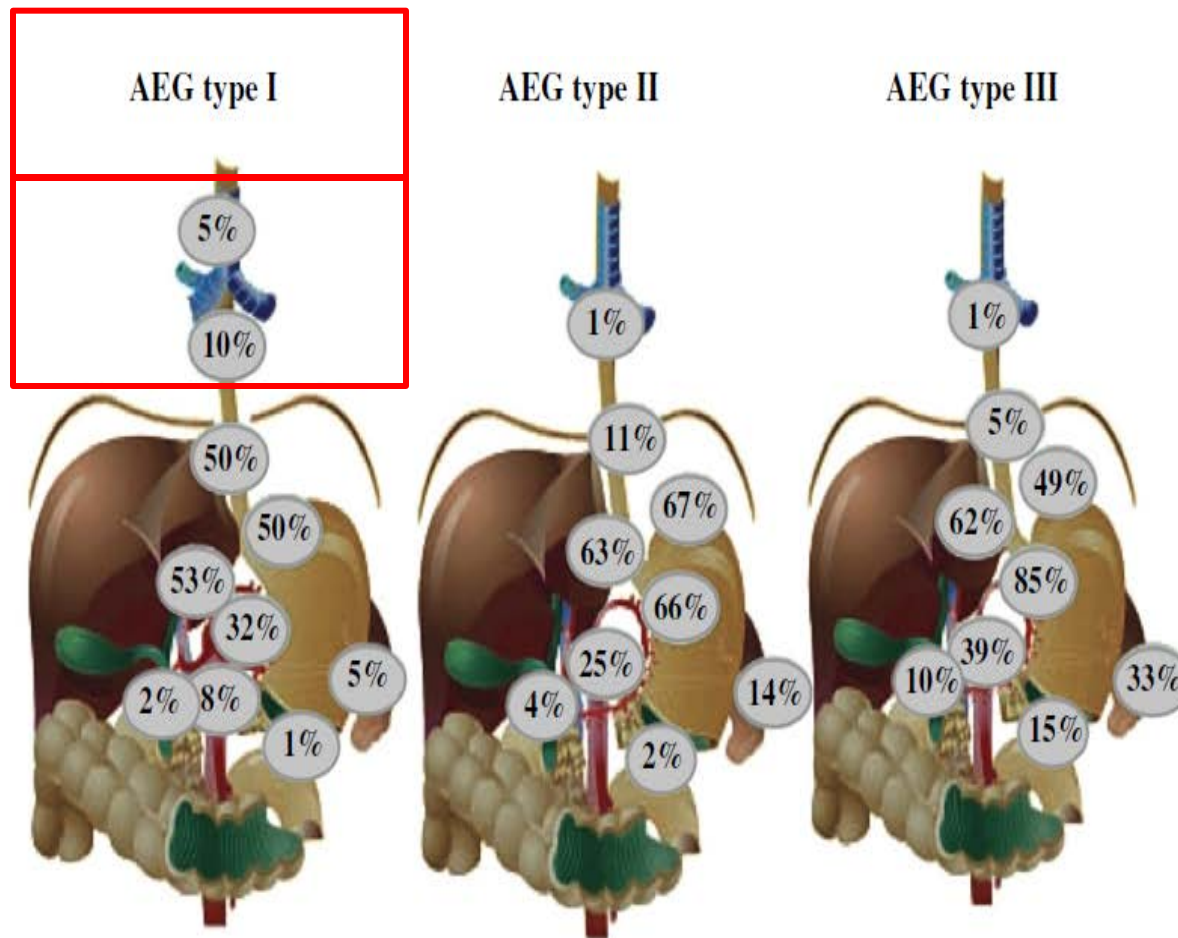


FIG. 2 Distribution of lymph node metastases in patients with resected adenocarcinoma of the esophagogastric junction (AEG) types I, II, and III. [Adapted from: Siewert JR, Stein HJ, Feith M. Adenocarcinoma of the esophago-gastric junction. *Scand J Surg*. 2006;95:260–69.]

SIEWERT I

Lancet Oncol 2011; 12: 296-306

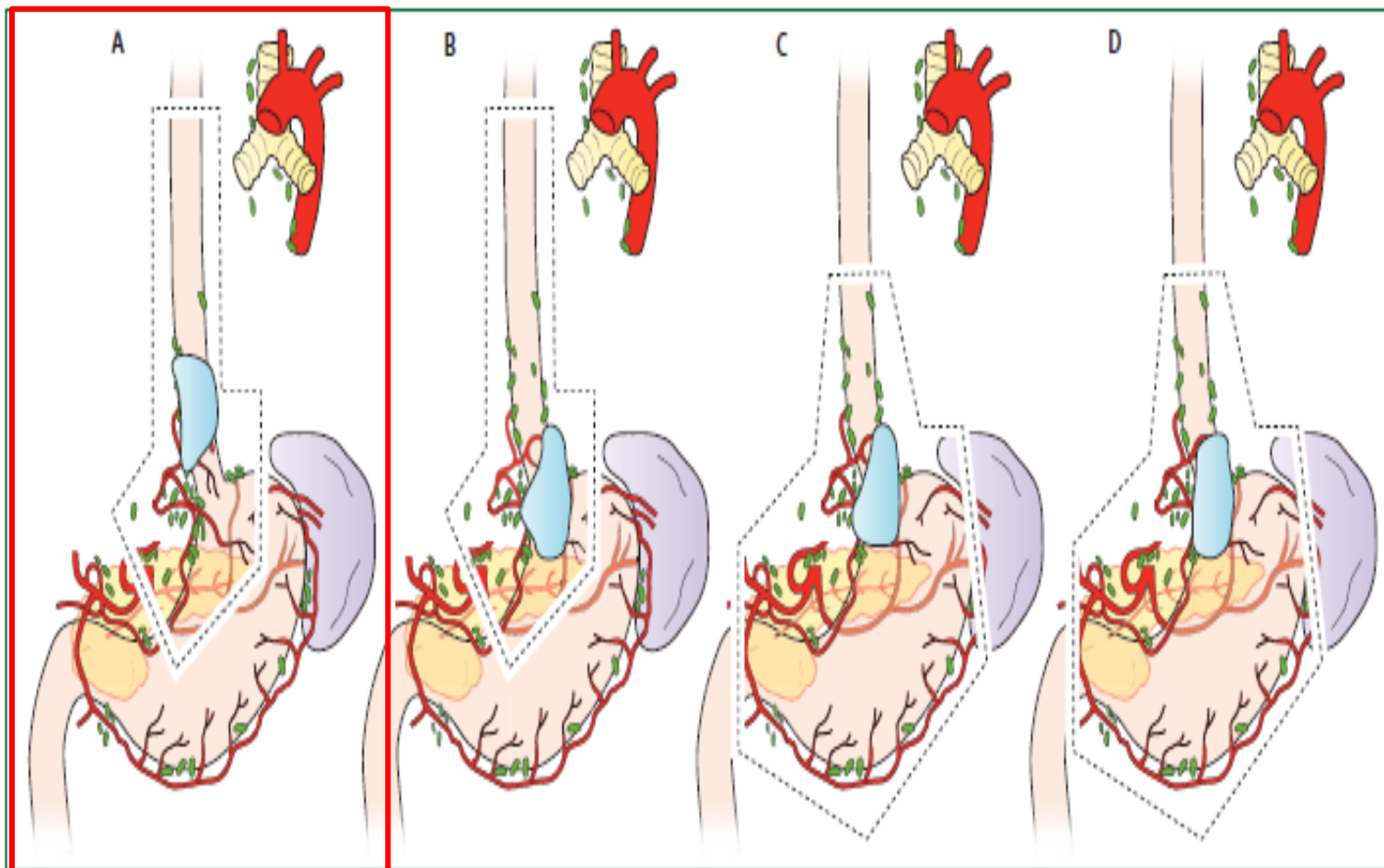


Figure 4: Schematic representation of recommended extent of surgical resection for oesophagogastric junction adenocarcinomas

Type I (A; subtotal oesophagectomy with superior polar gastrectomy), type II (subtotal oesophagectomy with superior polar gastrectomy [B] or total gastrectomy with inferior oesophagectomy [C]), and type III (D; total gastrectomy). Blue region is tumour site.



ΛΕΜΦΑΔΕΝΙΚΗ ΔΙΑΣΠΟΡΑ

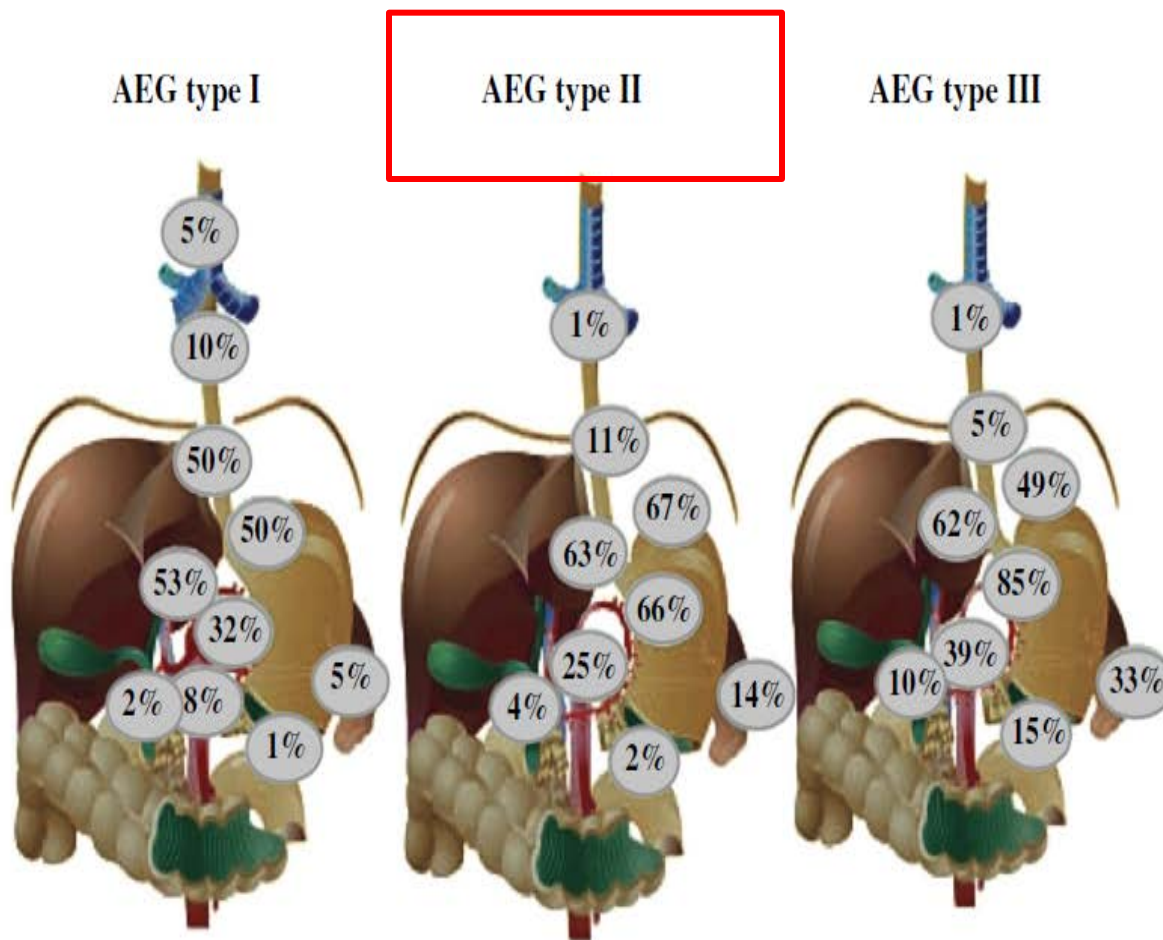


FIG. 2 Distribution of lymph node metastases in patients with resected adenocarcinoma of the esophago-gastric junction (AEG) types I, II, and III. [Adapted from: Siewert JR, Stein HJ, Feith M. Adenocarcinoma of the esophago-gastric junction. *Scand J Surg*. 2006;95:260–69.]



SIEWERT II

Lancet Oncol 2011; 12: 296-305

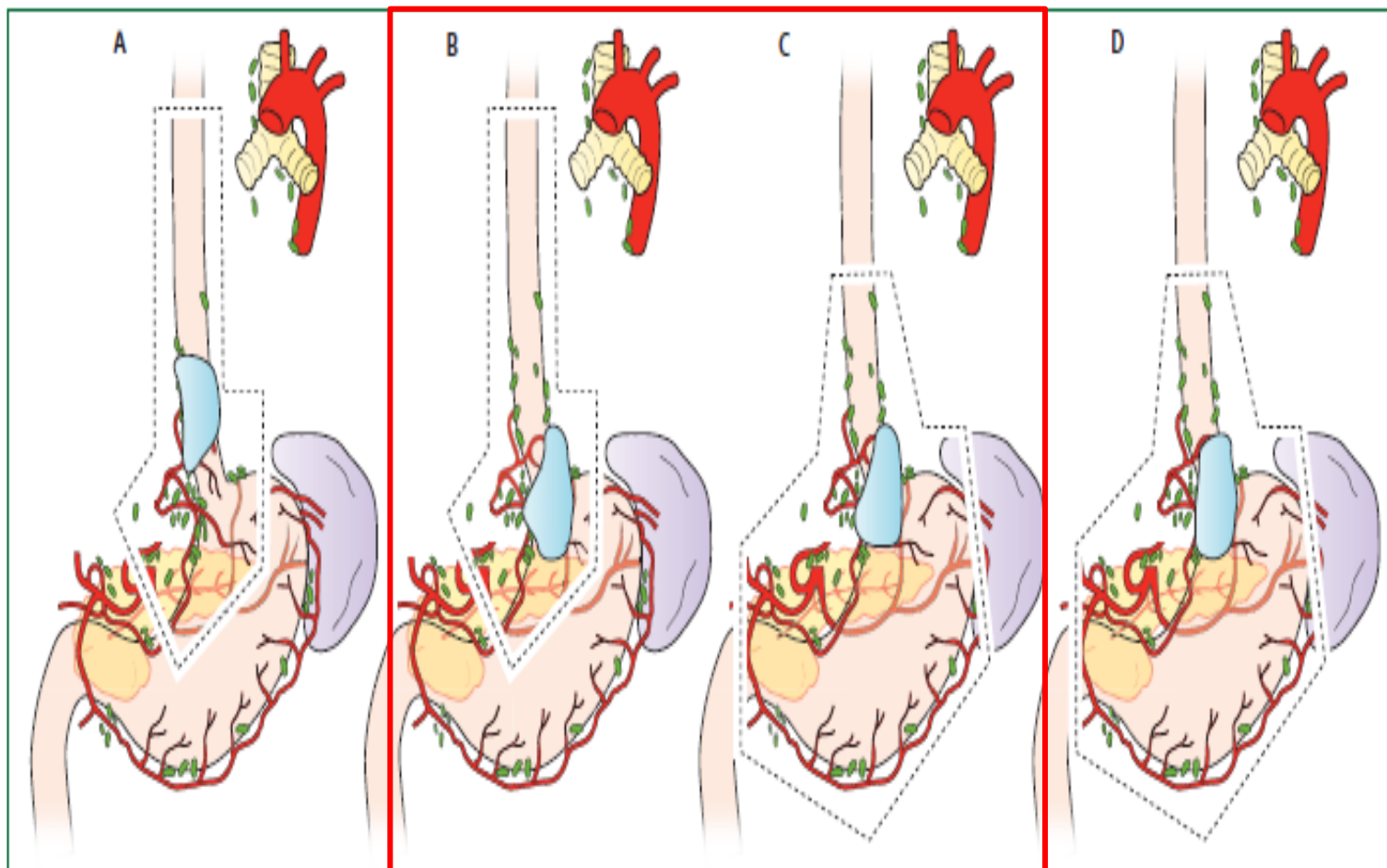


Figure 4: Schematic representation of recommended extent of surgical resection for oesophagogastric junction adenocarcinomas

Type I (A; subtotal oesophagectomy with superior polar gastrectomy), type II (subtotal oesophagectomy with superior polar gastrectomy [B] or total gastrectomy with inferior oesophagectomy [C]), and type III (D; total gastrectomy). Blue region is tumour site.

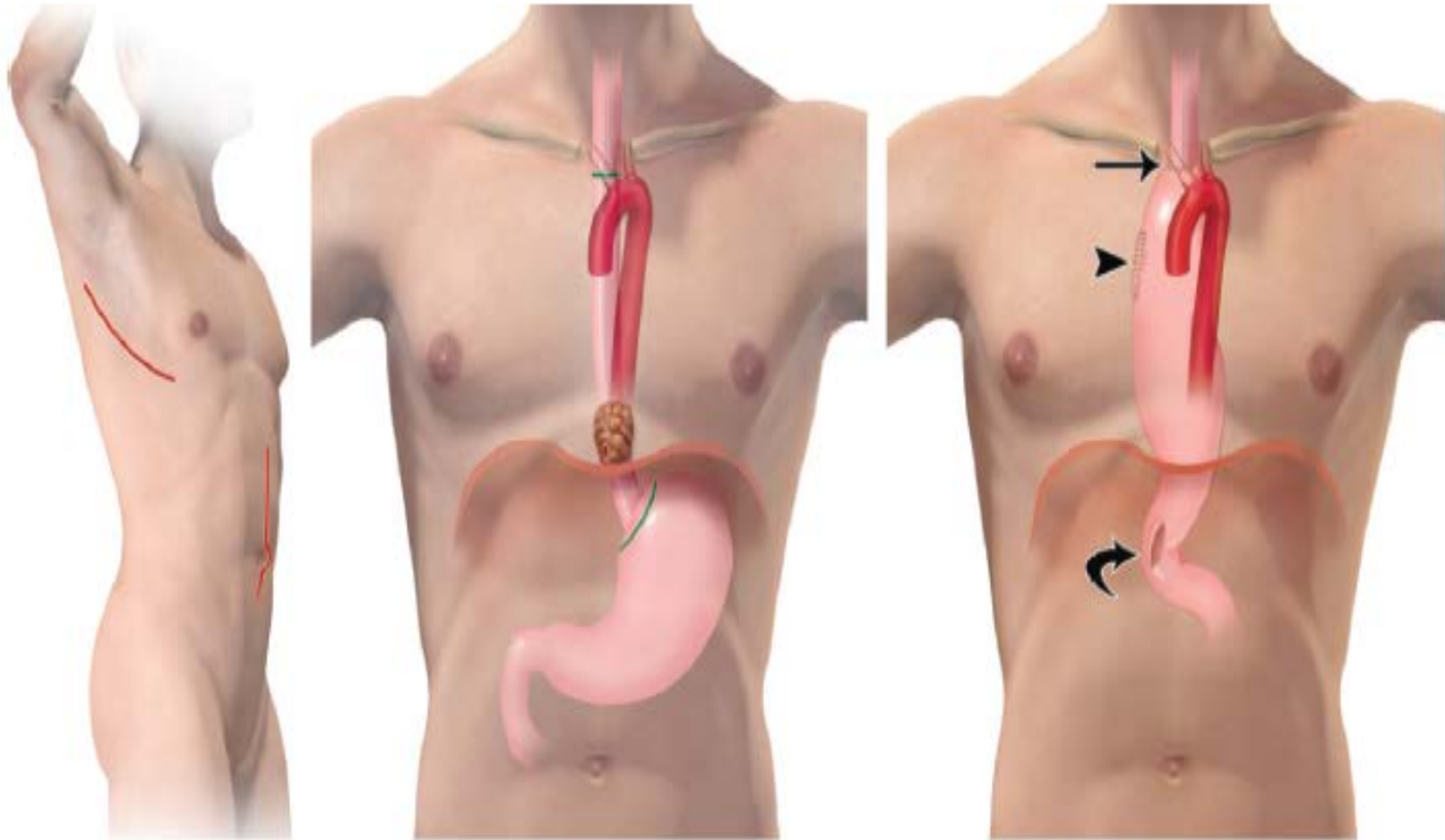


ΤΥΠΟΙ ΟΙΣΟΦΑΓΕΚΤΟΜΩΝ

- **IVOR – LEWIS ΟΙΣΟΦΑΓΕΚΤΟΜΗ**
- **TRANSIATAL ΟΙΣΟΦΑΓΕΚΤΟΜΗ**
- **McKEWON ΟΙΣΟΦΑΓΕΚΤΟΜΗ**
- **THREE FIELD ΟΙΣΟΦΑΓΕΚΤΟΜΗ**
- **ΟΙΣΟΦΑΓΕΚΤΟΜΗ ΜΕ ΑΡΙΣΤΕΡΗ ΘΩΡΑΚΟΚΟΙΛΙΑΚΗ ΤΟΜΗ**
- **ΦΑΡΥΓΓΟ-ΛΑΡΥΓΓΟ-ΟΙΣΟΦΑΓΕΚΤΟΜΗ**

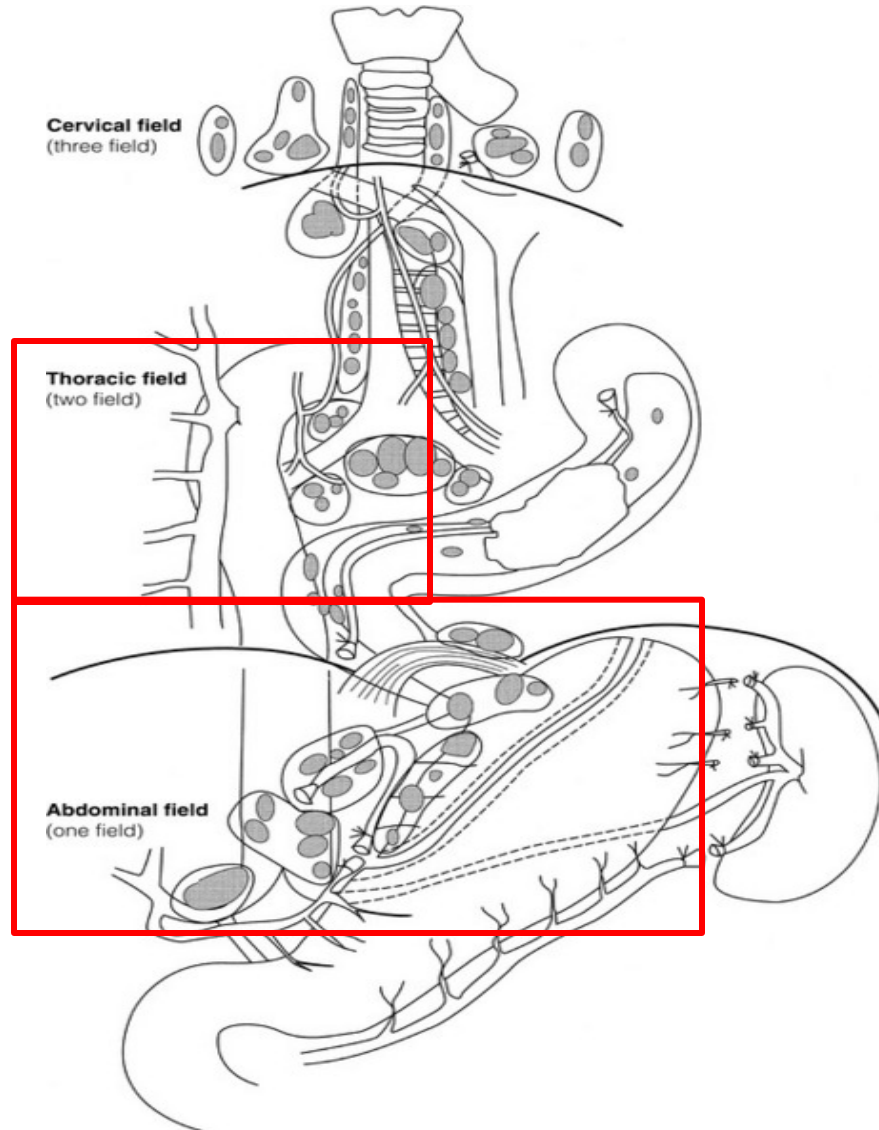


IVOR-LEWIS ΟΙΣΟΦΑΓΕΚΤΟΜΗ



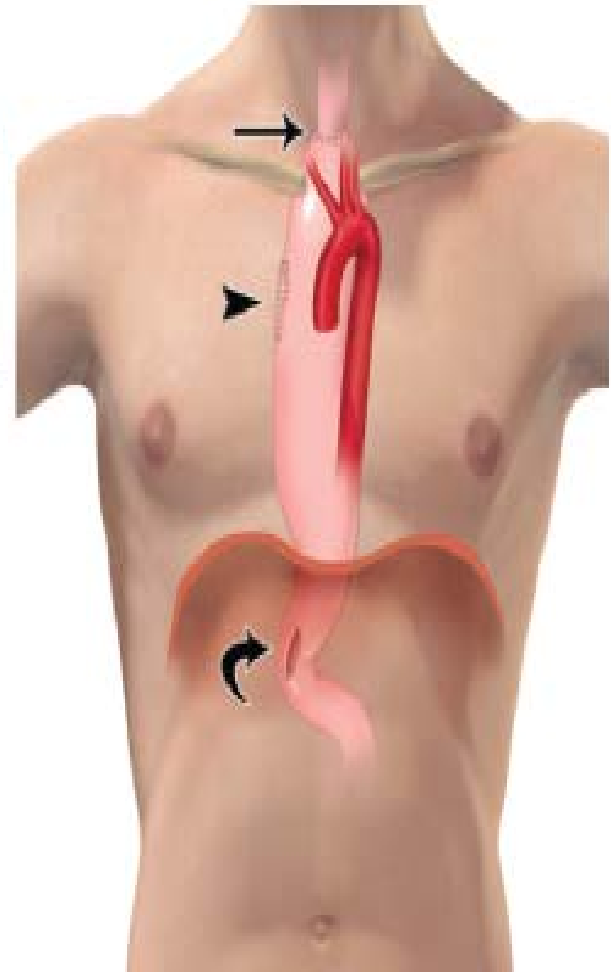


ΛΕΜΦΑΔΕΝΙΚΟΣ ΚΑΘΑΡΙΣΜΟΣ - IVOR-LEWIS



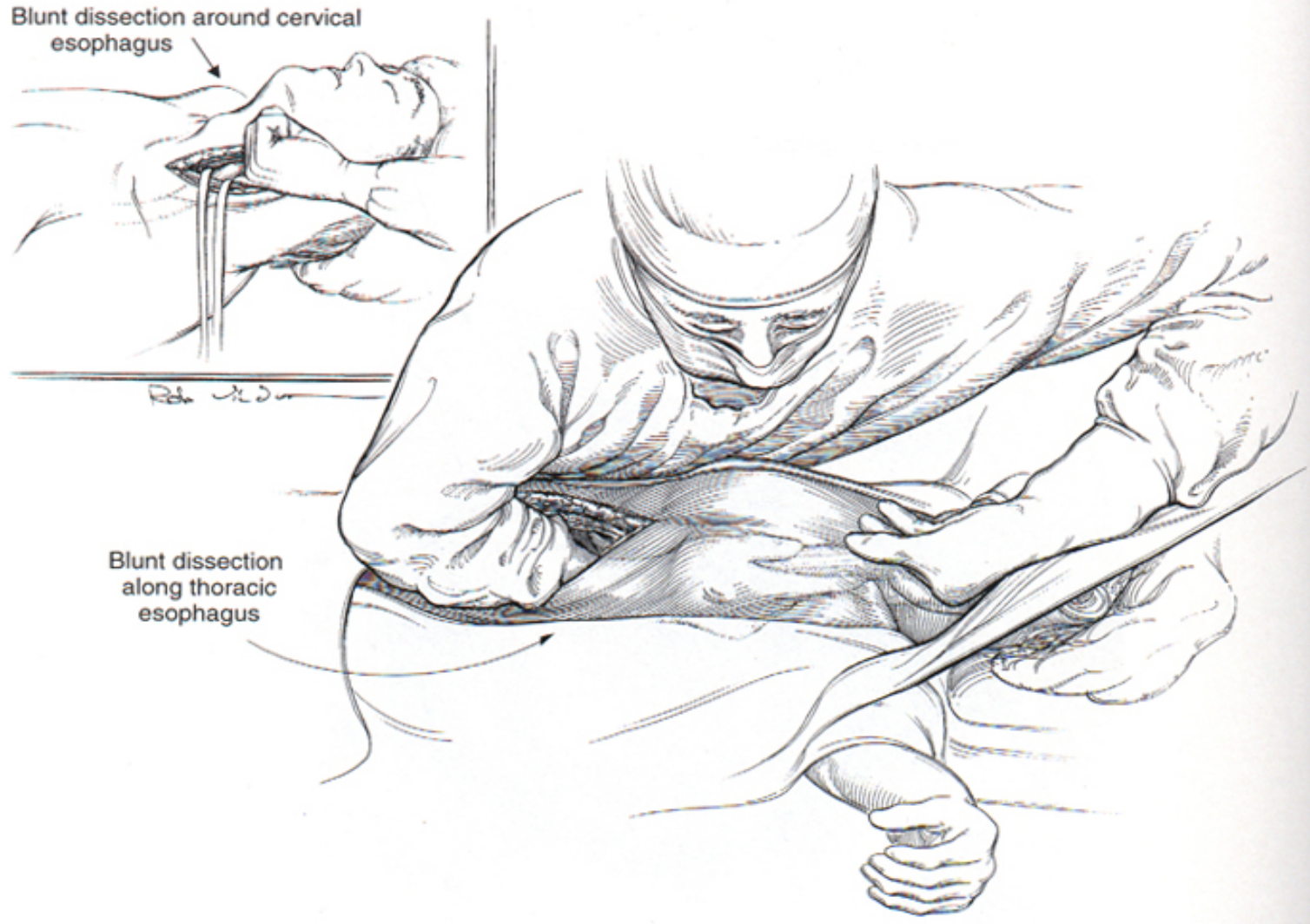


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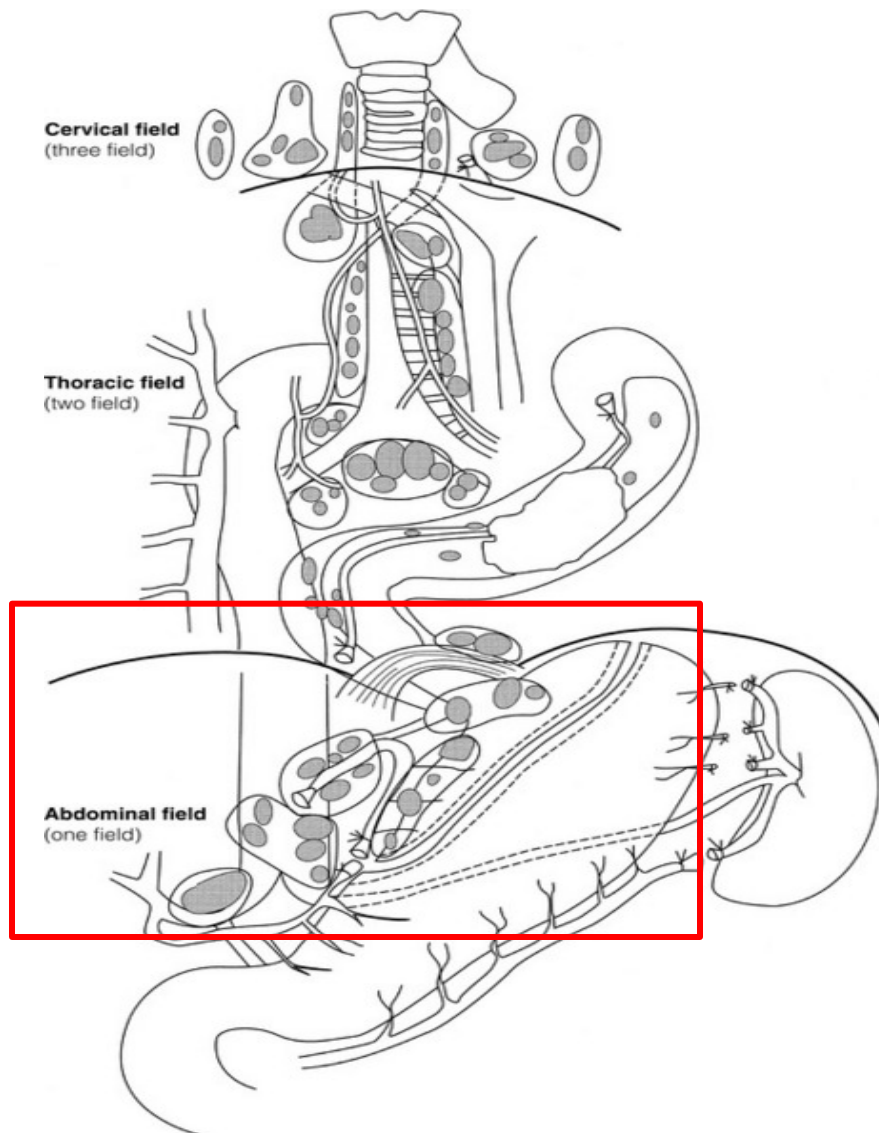


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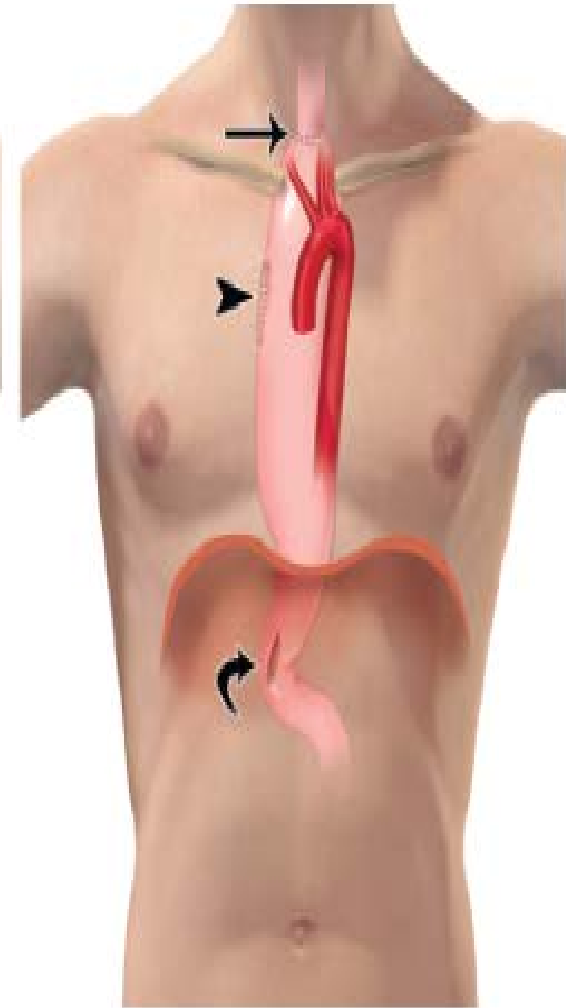
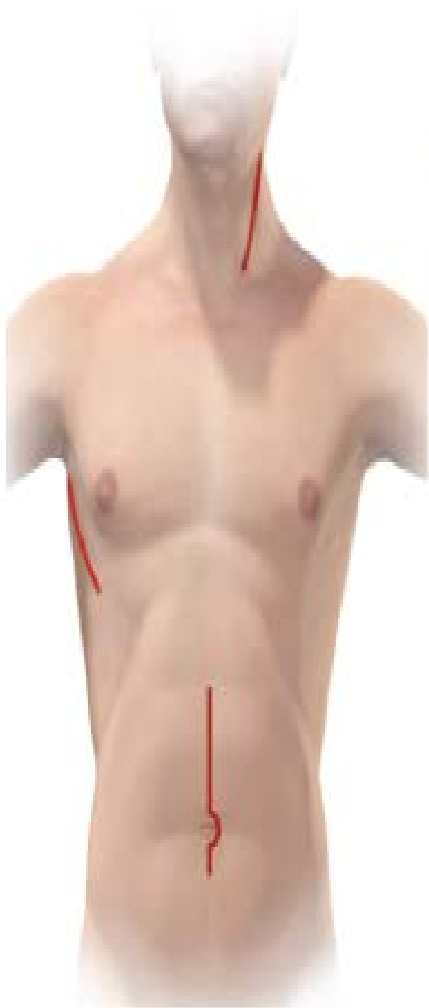


ΛΕΜΦΑΔΕΝΙΚΟΣ ΚΑΘΑΡΙΣΜΟΣ – TRANSHIATAL



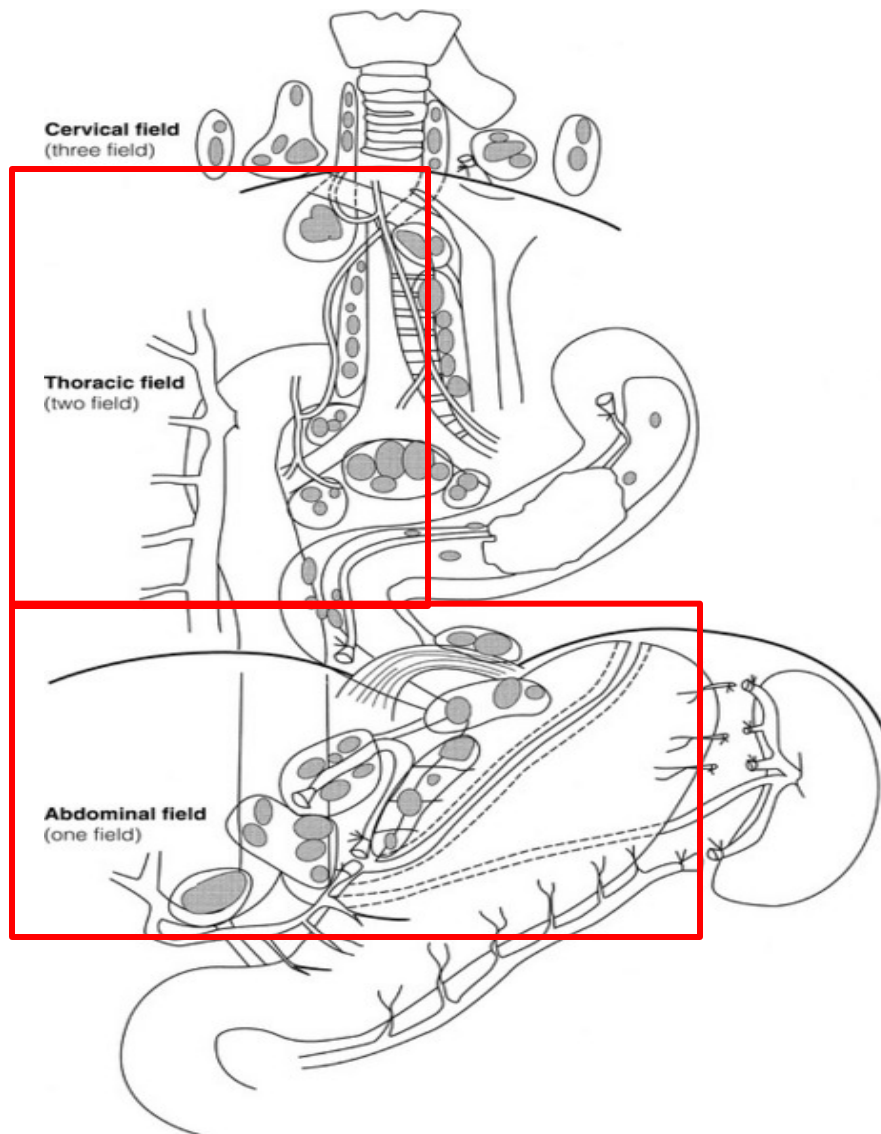


ΜεΚΕΩΝ ΟΙΣΟΦΑΓΕΚΤΟΜΗ





ΛΕΜΦΑΔΕΝΙΚΟΣ ΚΑΘΑΡΙΣΜΟΣ - ΜcKEWON



ΠΑΘΗΣΕΙΣ ΟΙΣΟΦΑΓΟΥ

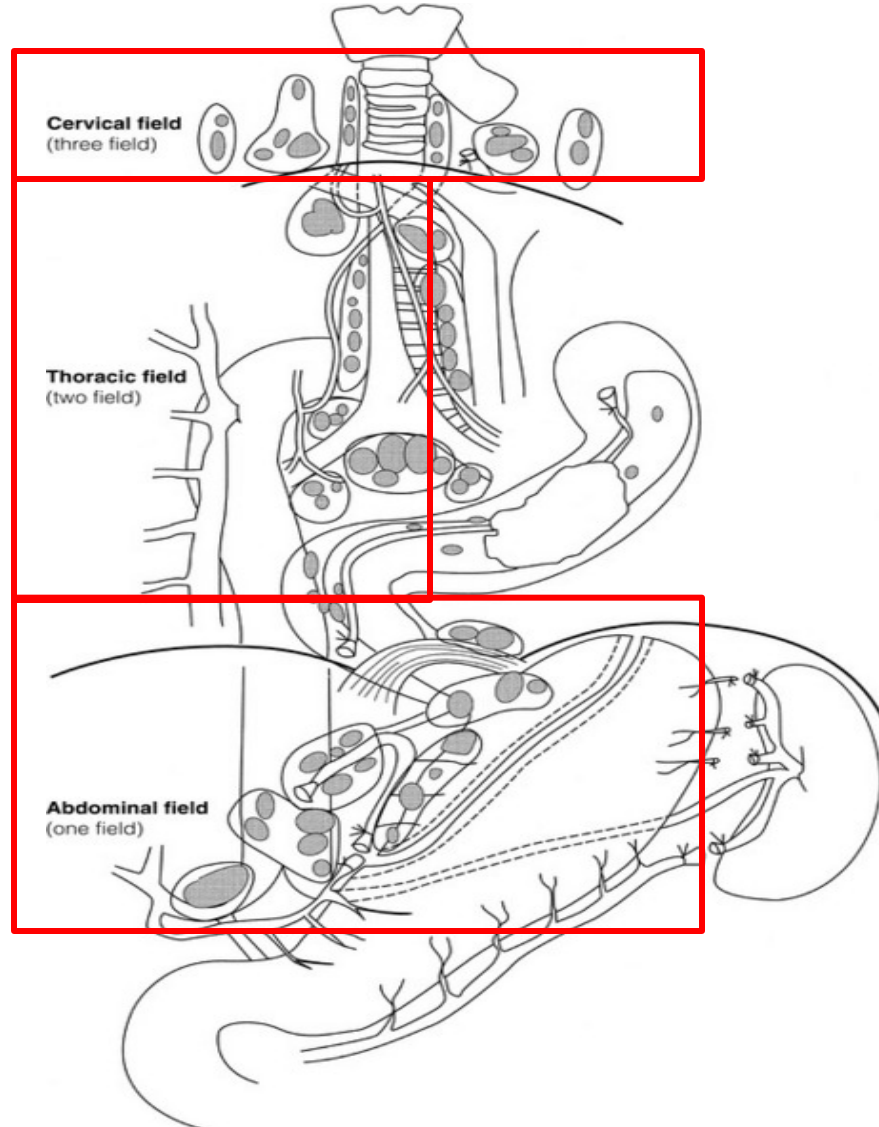


THREE FIELD ΟΙΣΟΦΑΓΕΚΤΟΜΗ



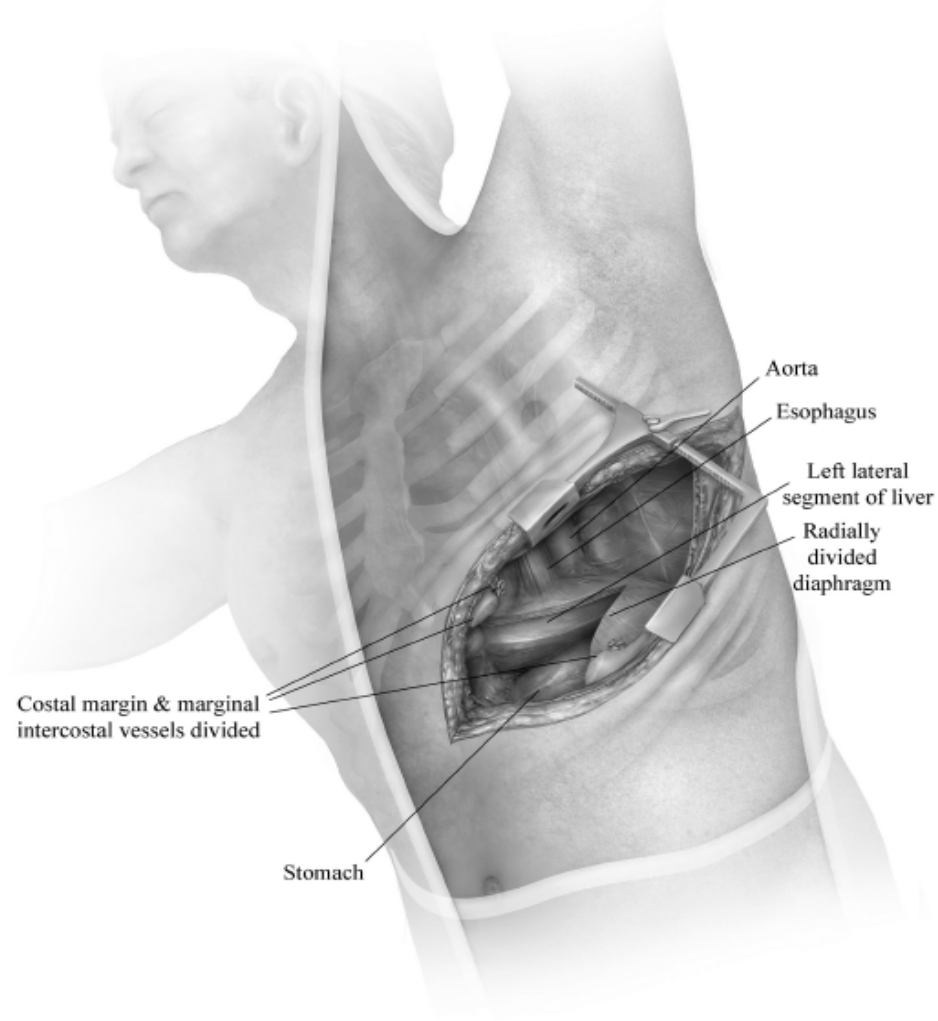


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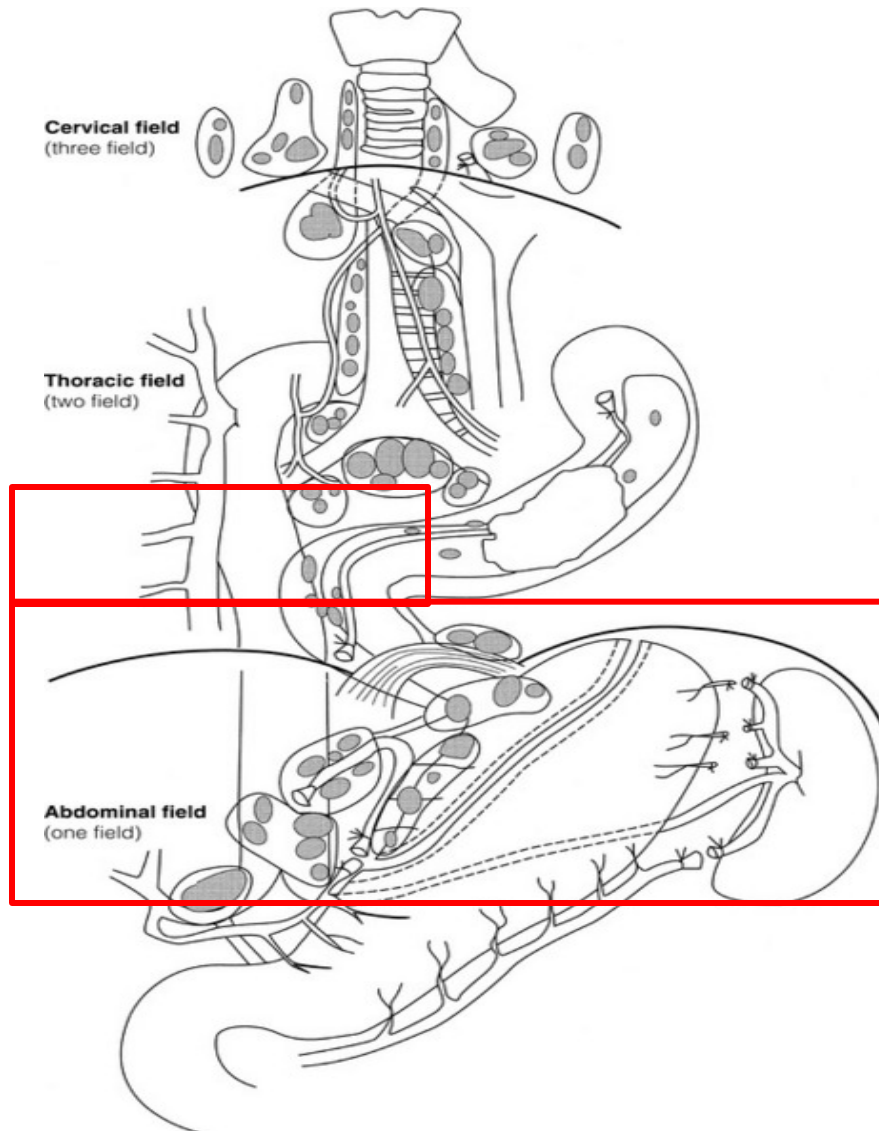


ΟΙΣΟΦΑΓΕΚΤΟΜΗ ΜΕΣΩ ΑΡΙΣΤΕΡΗΣ ΘΩΡΑΚΟΚΟΙΛΙΑΚΗΣ ΤΟΜΗΣ





ΛΕΜΦΑΔΕΝΙΚΟΣ ΚΑΘΑΡΙΣΜΟΣ - ΑΡΙΣΤΕΡΗ ΘΩΡΑΚΟΚΟΙΛΙΑΚΗ

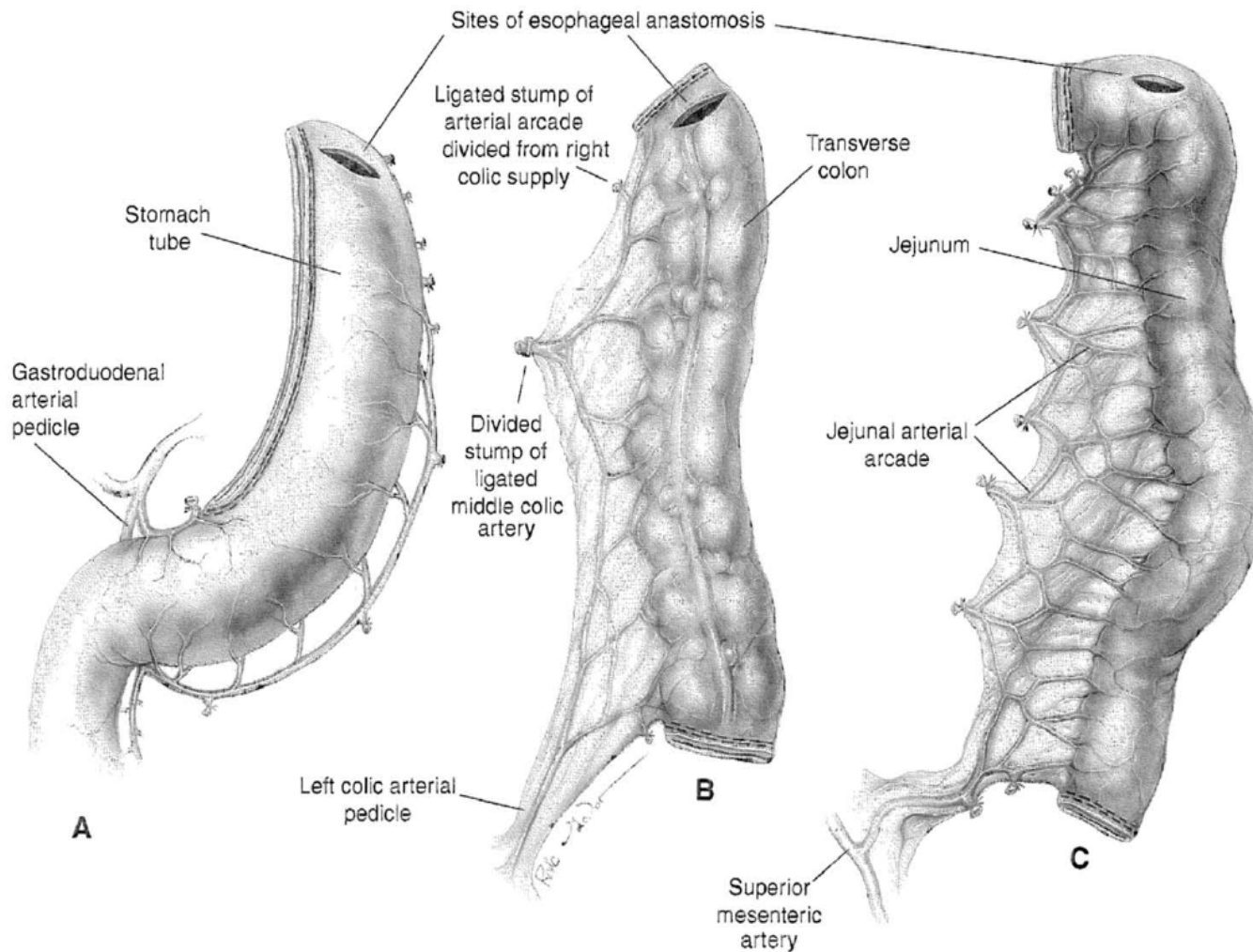




ΑΠΟΚΑΤΑΣΤΑΣΗ ΣΥΝΕΧΕΙΑΣ ΠΕΠΤΙΚΟΥ

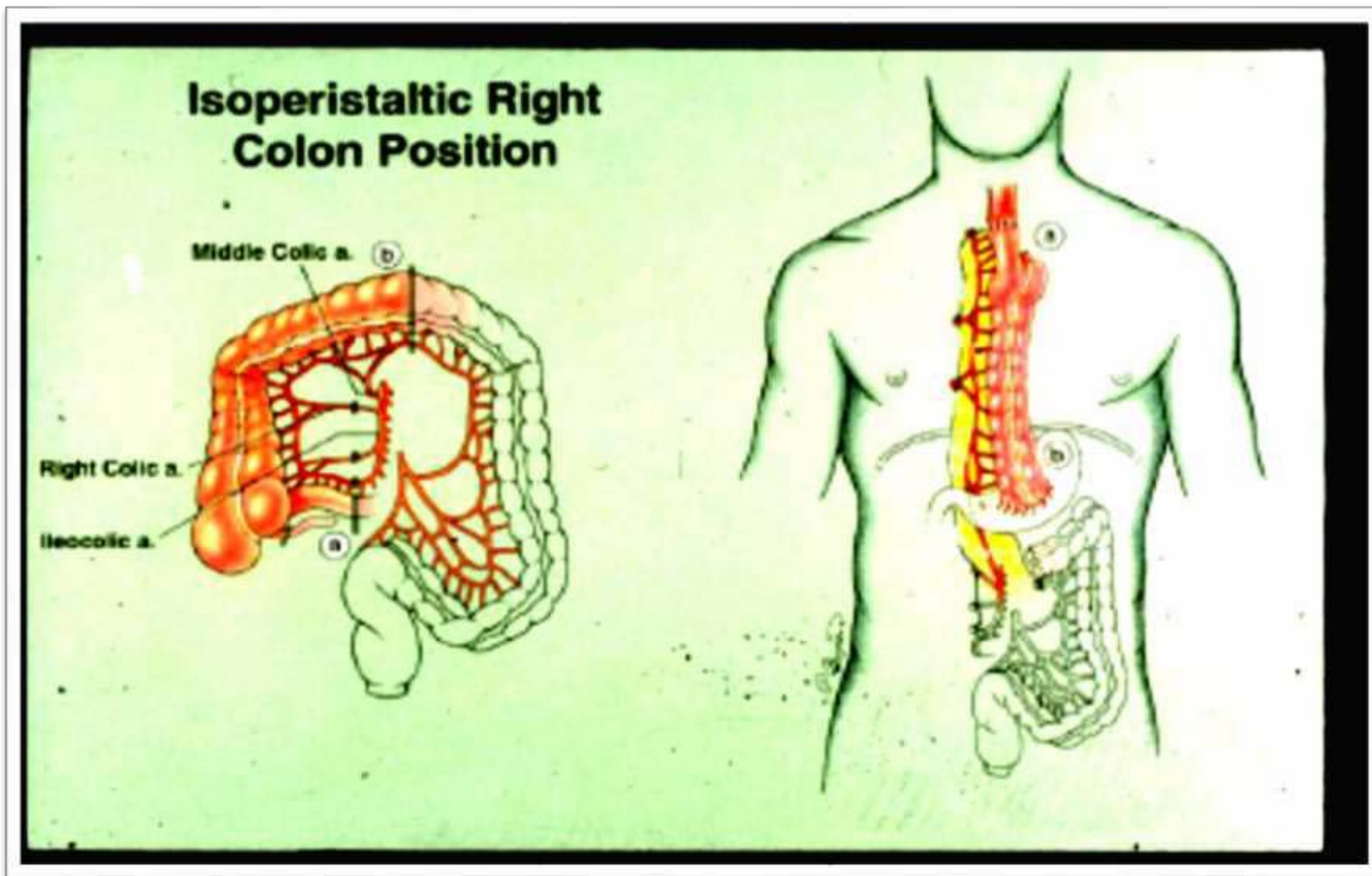


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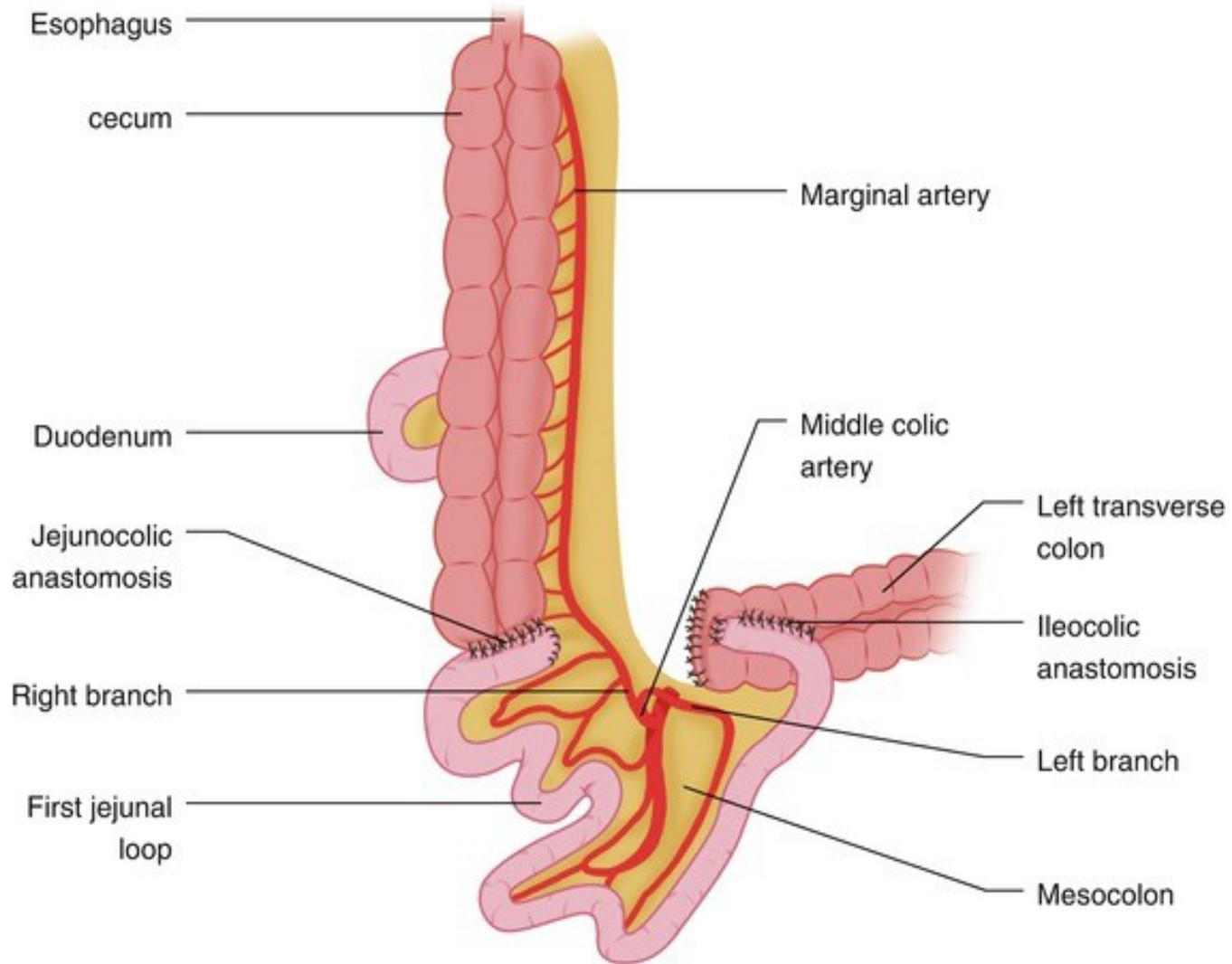


RIGHT COLON



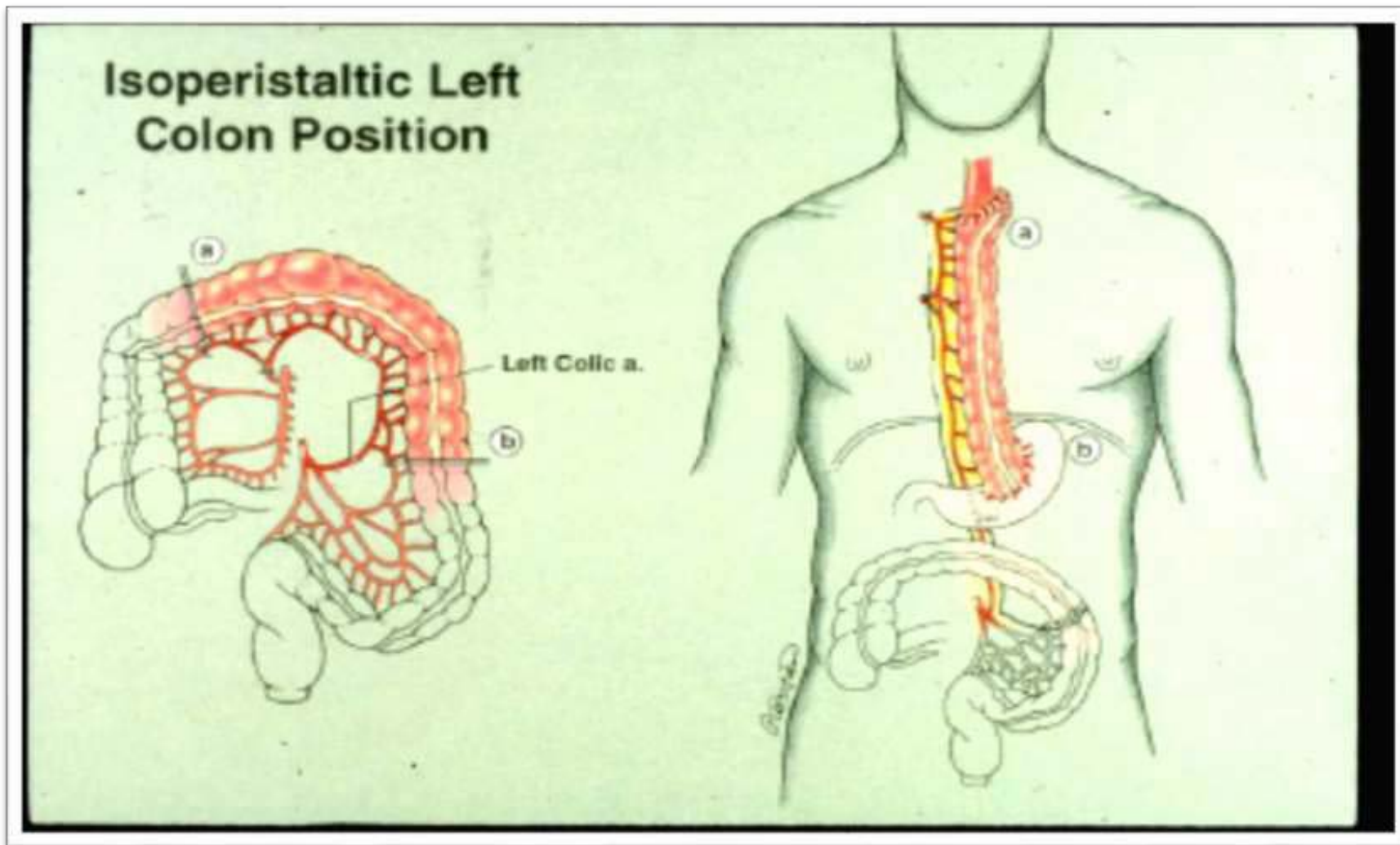


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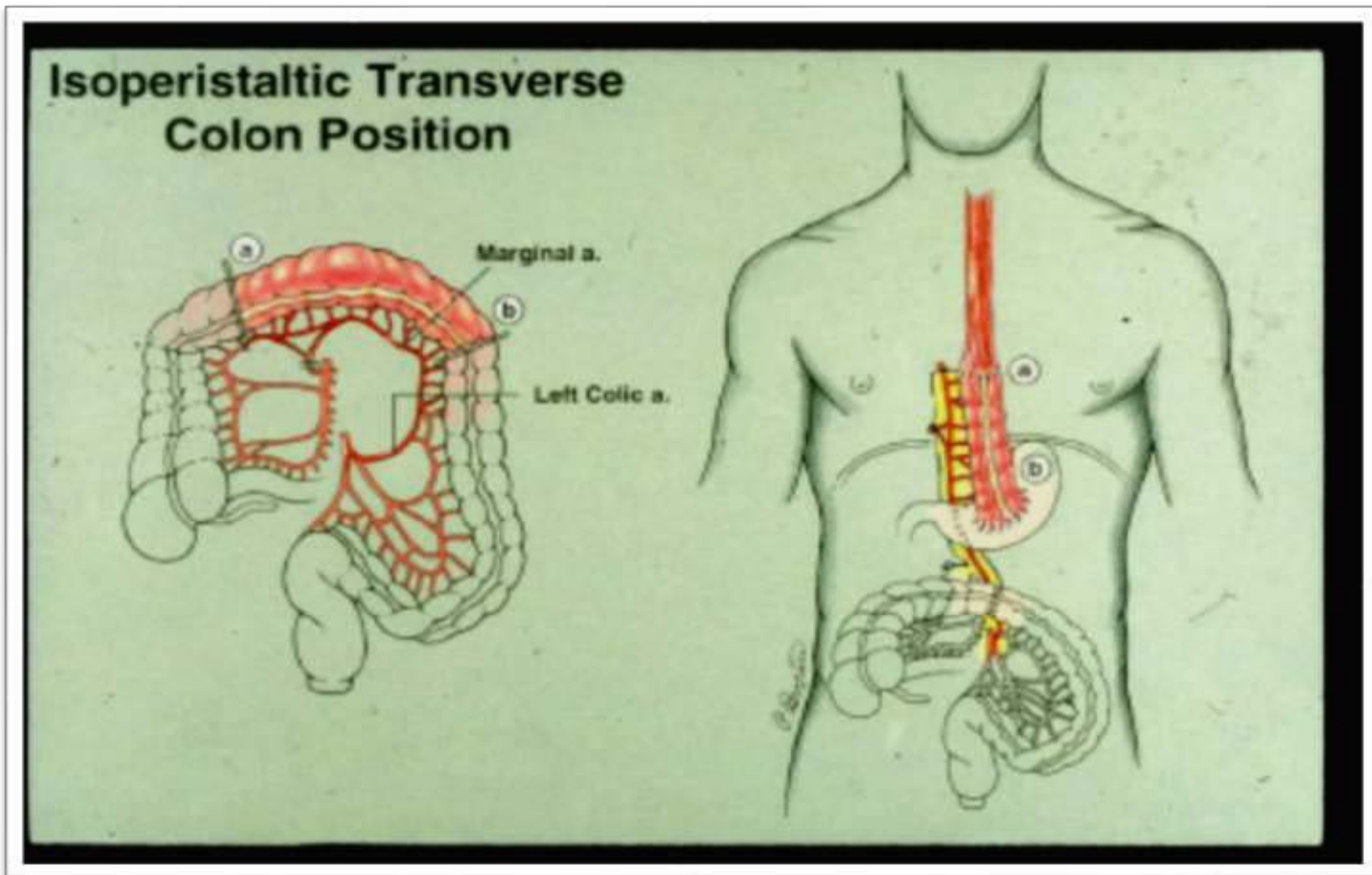


LEFT COLON



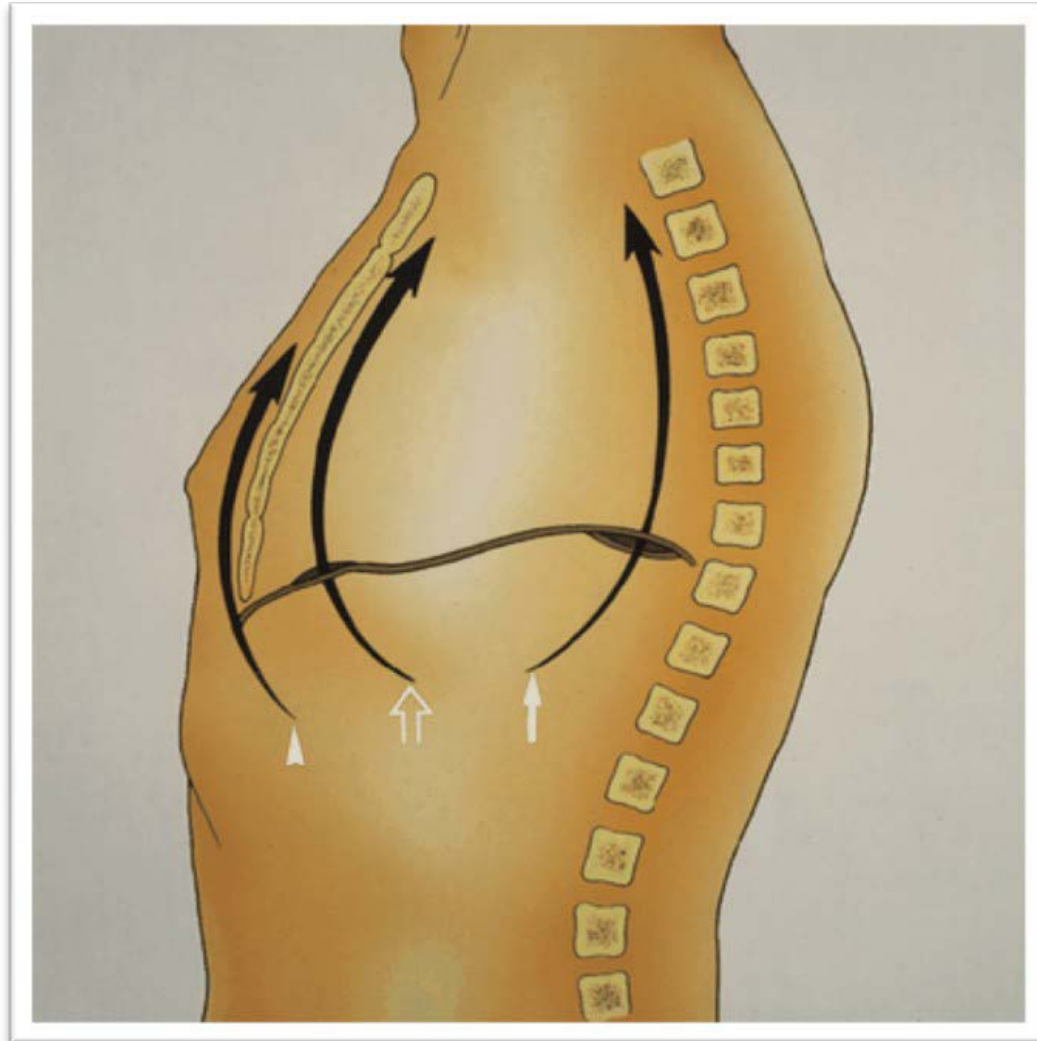


TRANSVERSE COLON





ROUTES OF RECONSTRUCTION



ΠΑΘΗΣΕΙΣ ΟΙΣΟΦΑΓΟΥ

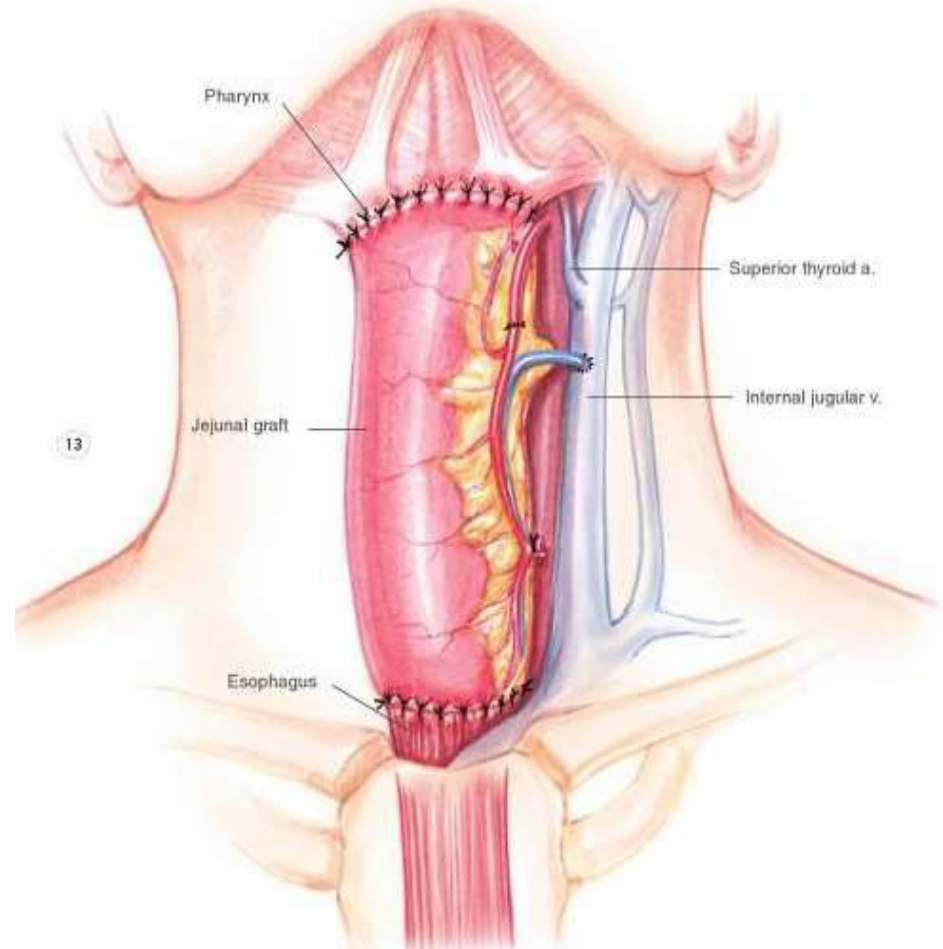


FREE JEJUNAL GRAFT





FREE JEJUNAL GRAFT

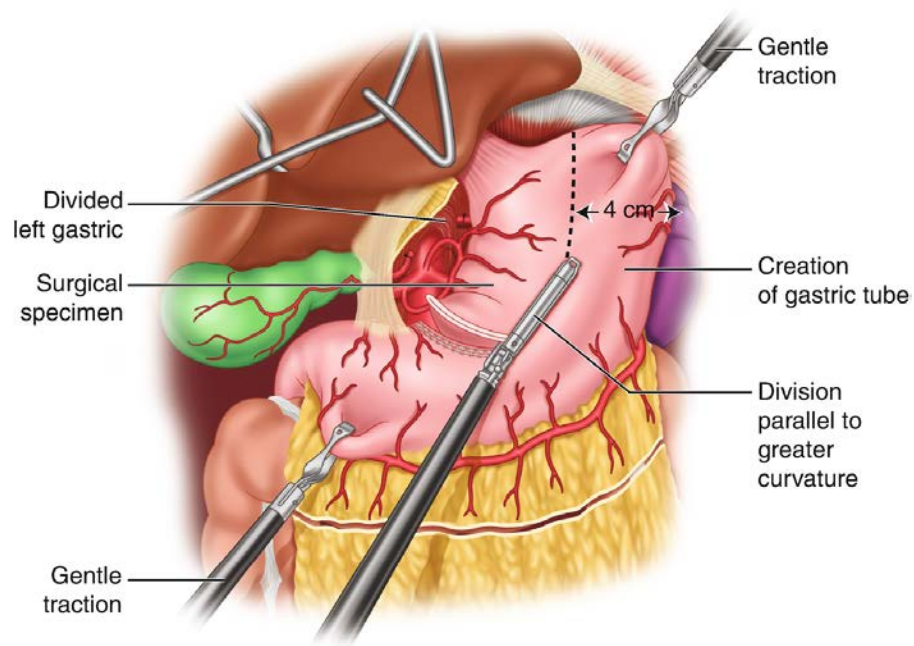
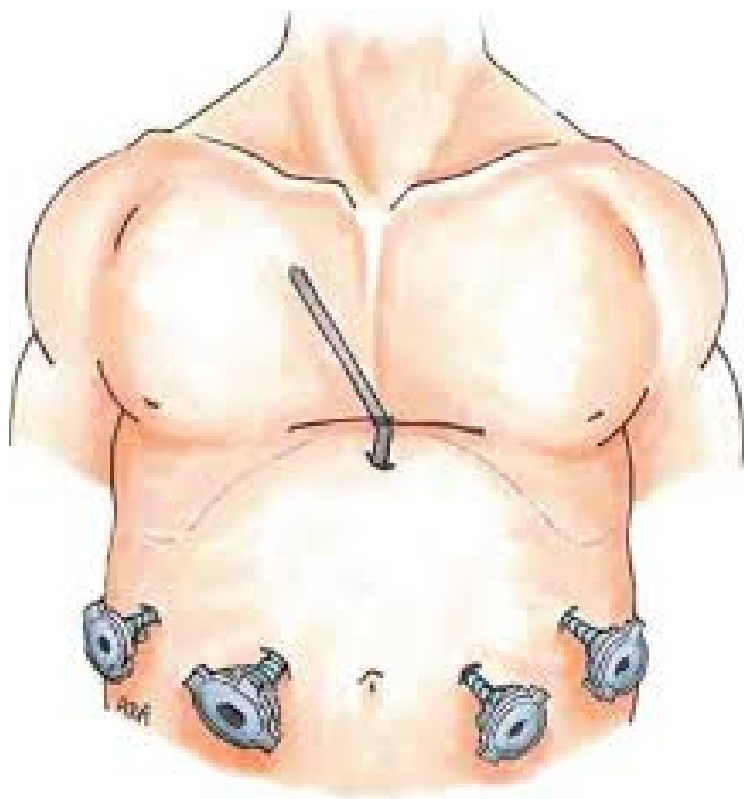




ΕΛΑΧΙΣΤΑ ΕΠΕΜΒΑΤΙΚΗ ΟΙΣΟΦΑΓΕΚΤΟΜΗ (ΜΙΕ, HYBRID, RAMIE)

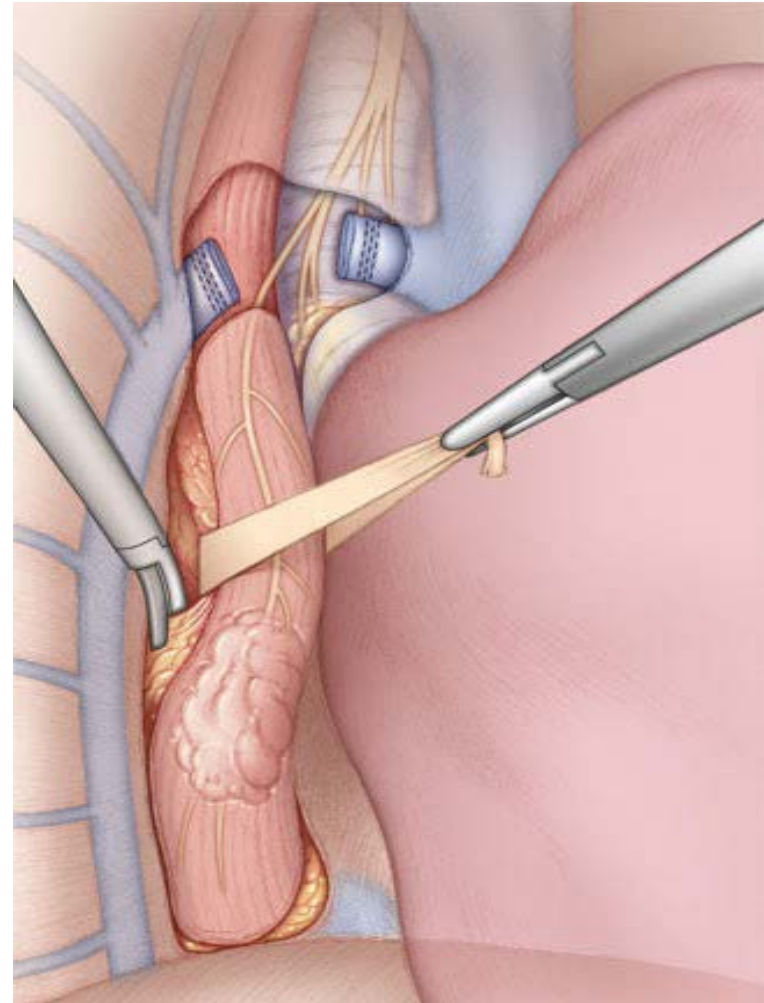
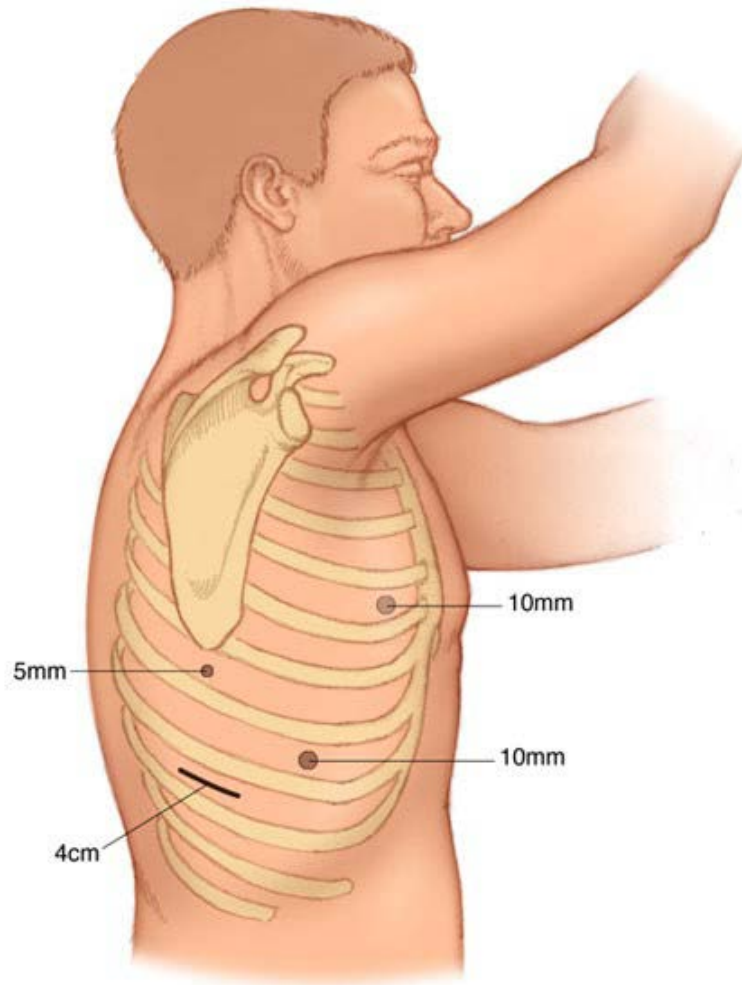


MINIMALLY INVASIVE ESOPHAGECTOMY (MIE)



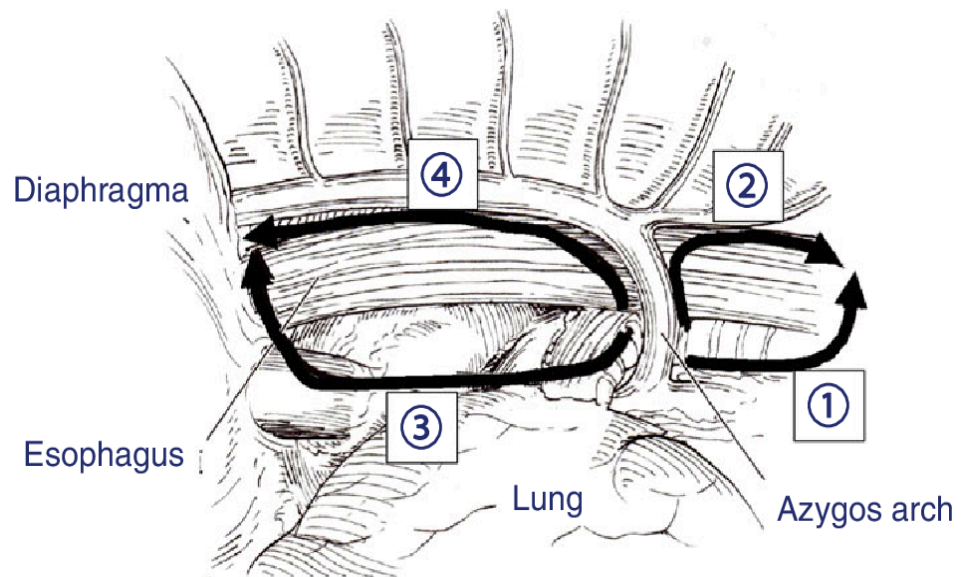
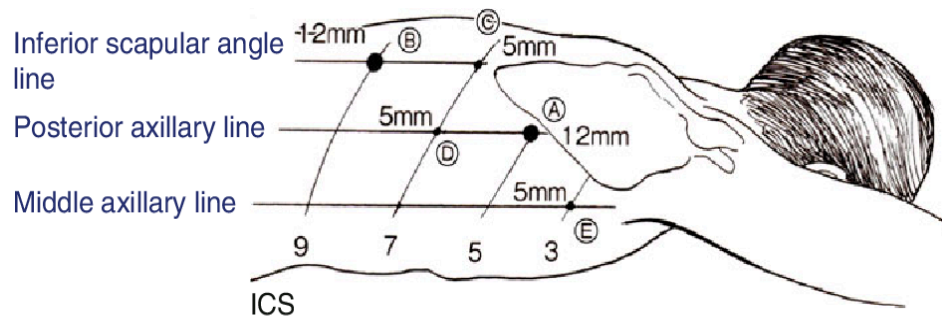
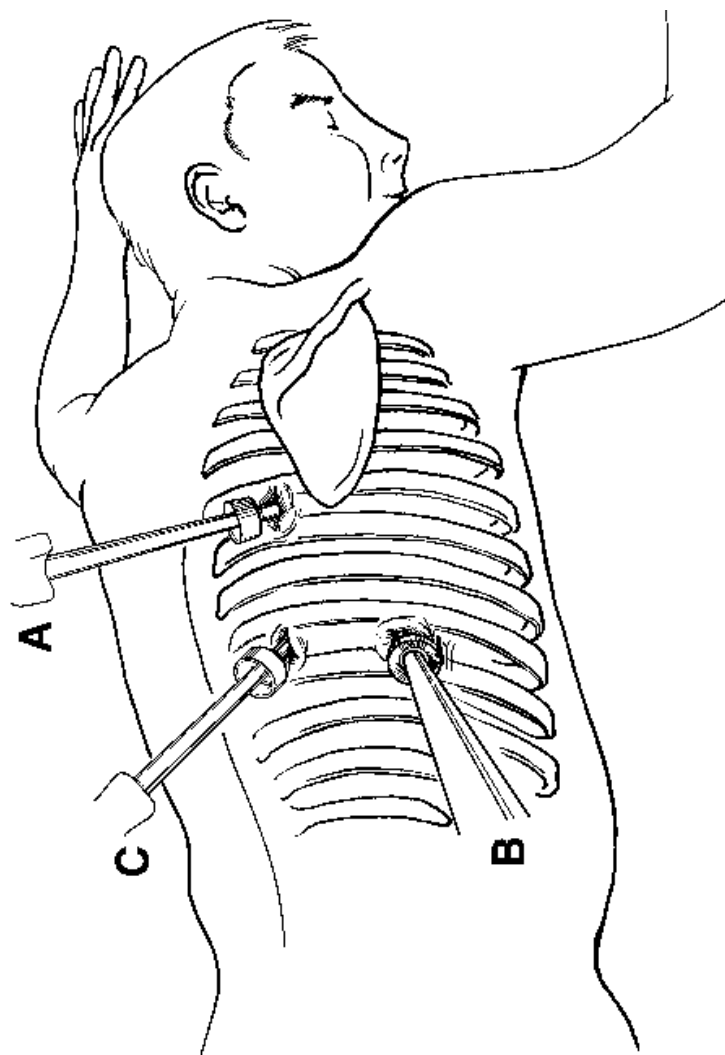


MINIMALLY INVASIVE ESOPHAGECTOMY (MIE)





MINIMALLY INVASIVE ESOPHAGECTOMY (MIE)





HYBRID ESOPHAGECTOMY

HYBRID APPROACHES TO MIE

Intrathoracic anastomosis

Laparoscopic Ivor Lewis

Laparoscopy

+

Right thoracotomy

Thoracoscopic Ivor Lewis

Laparotomy

+

Right thoracoscopy

Cervical anastomosis

Laparoscopic McKeown

Right thoracotomy

+

Laparoscopy
and
Left cervicotomy

Thoracoscopic McKeown

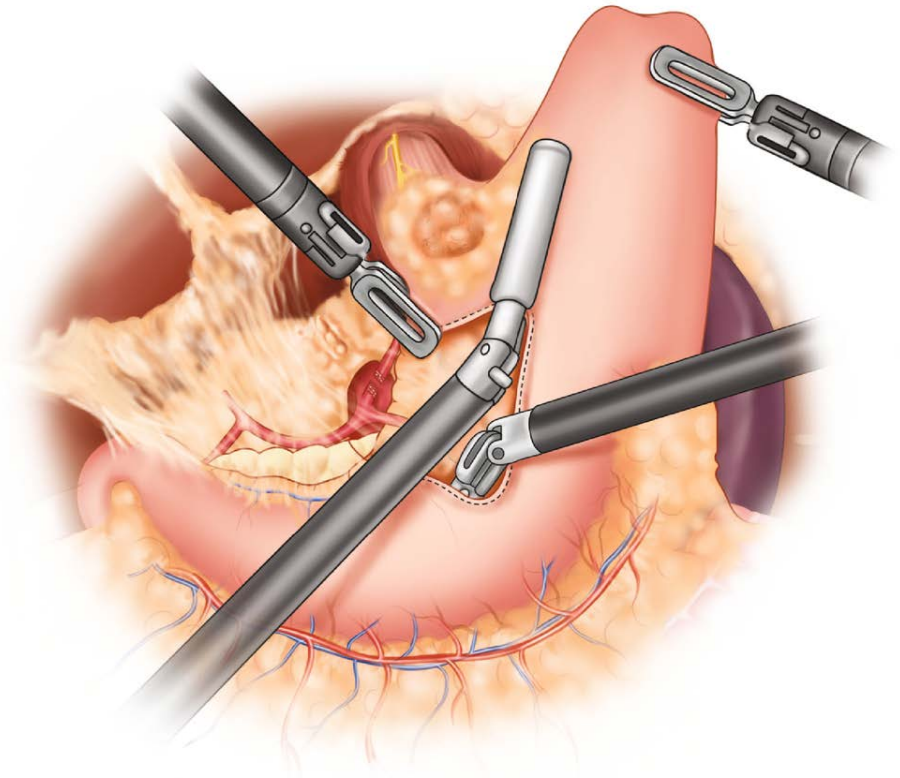
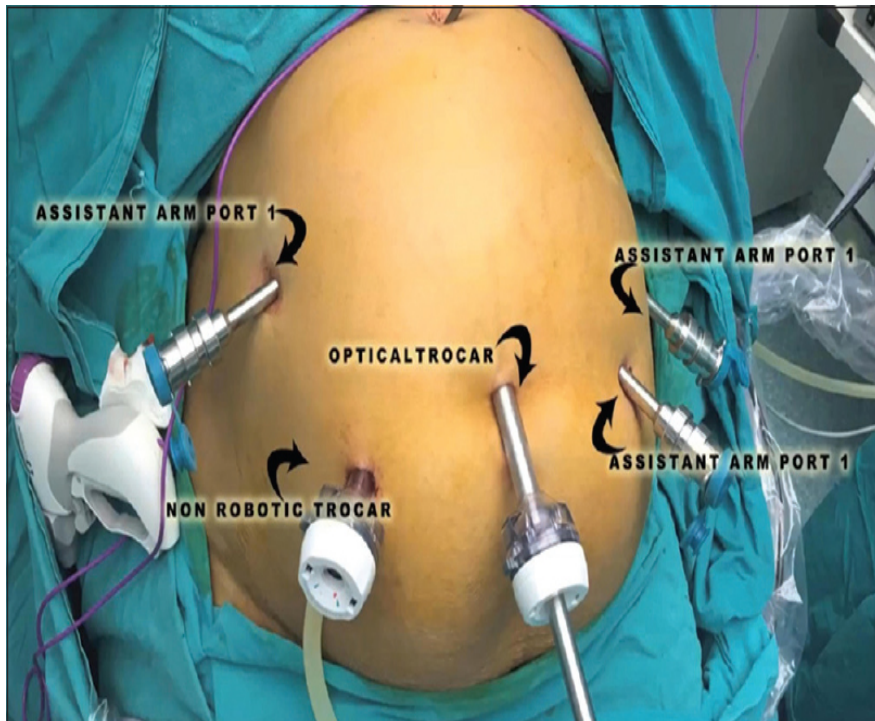
Right VATS

+

Laparotomy
and
Left cervicotomy

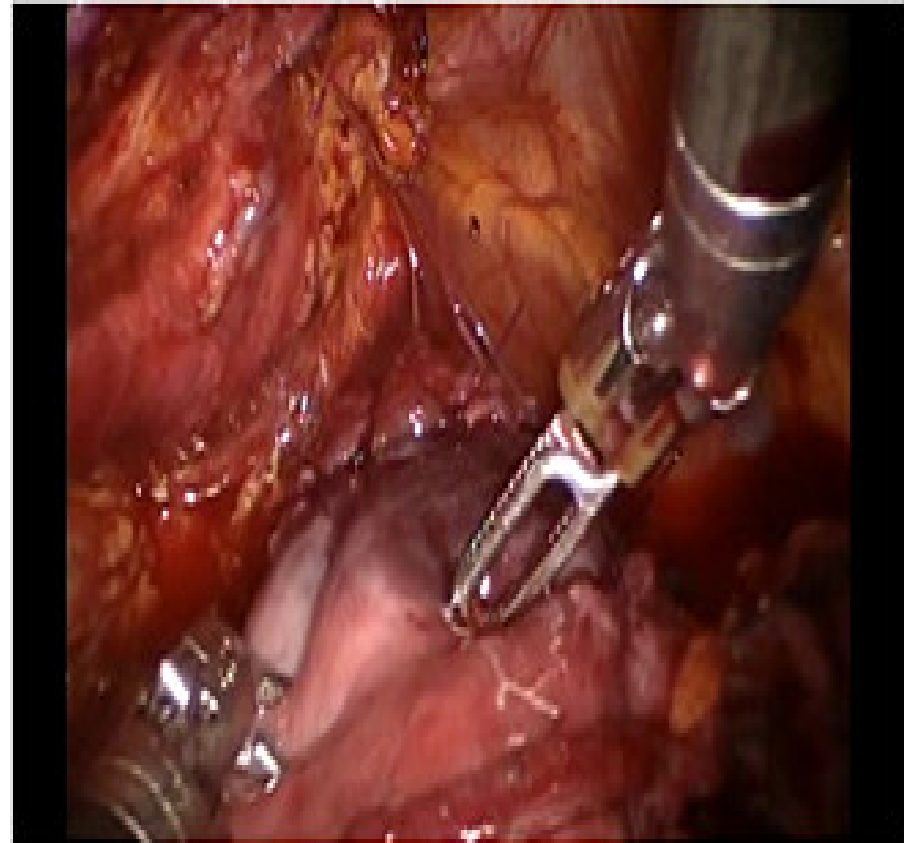
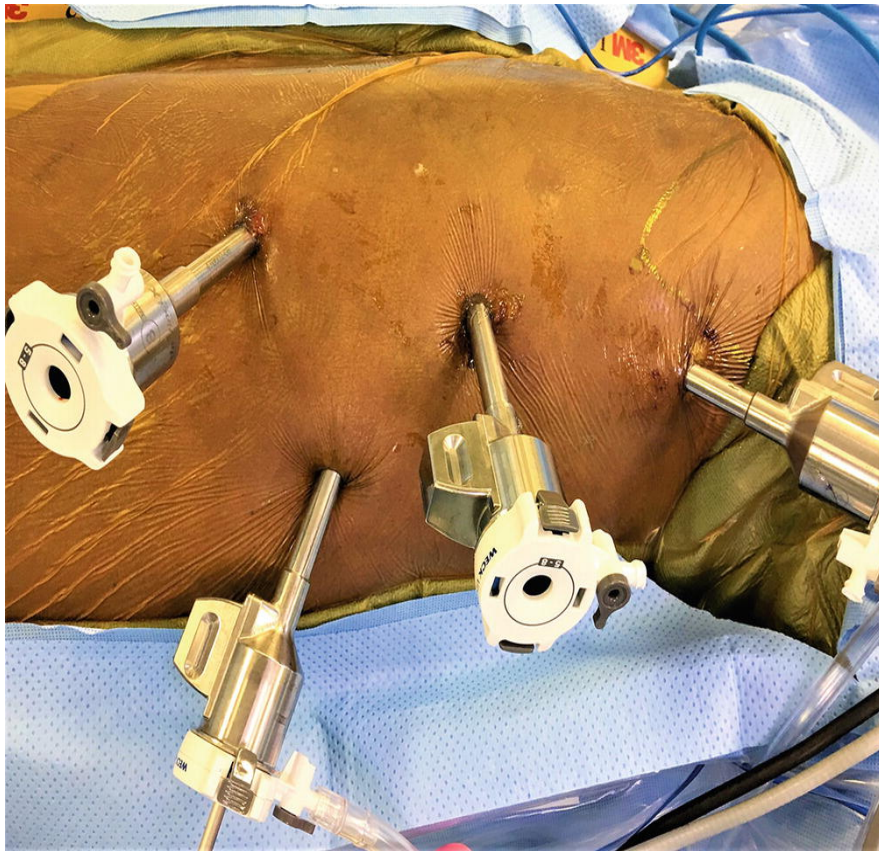


ROBOTIC ASSISTED MINIMALLY INVASIVE ESOPHAGECTOMY (RAMIE)





ROBOTIC ASSISTED MINIMALLY INVASIVE ESOPHAGECTOMY (RAMIE)





ΕΥΧΑΡΙΣΤΩ ΠΟΛΥ