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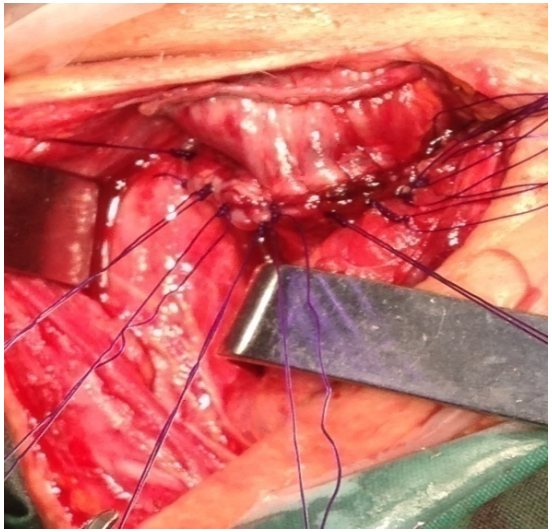
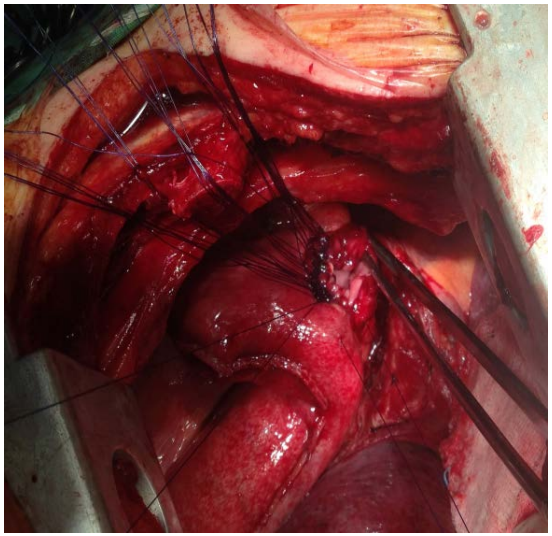




ΔΗΜΗΤΡΙΟΣ Α. ΣΧΙΖΑΣ

ΕΠΙΚΟΥΡΟΣ ΚΑΘΗΓΗΤΗΣ
Α΄ΧΕΙΡΟΥΡΓΙΚΗ ΚΛΙΝΙΚΗ ΕΚΠΑ
ΛΑΪΚΟ ΝΟΣΟΚΟΜΕΙΟ

ΑΝΑΤΟΜΙΑ/ΙΣΤΟΛΟΓΙΑ ΟΙΣΟΦΑΓΟΥ

ΑΝΑΤΟΜΙΑ ΟΙΣΟΦΑΓΟΥ

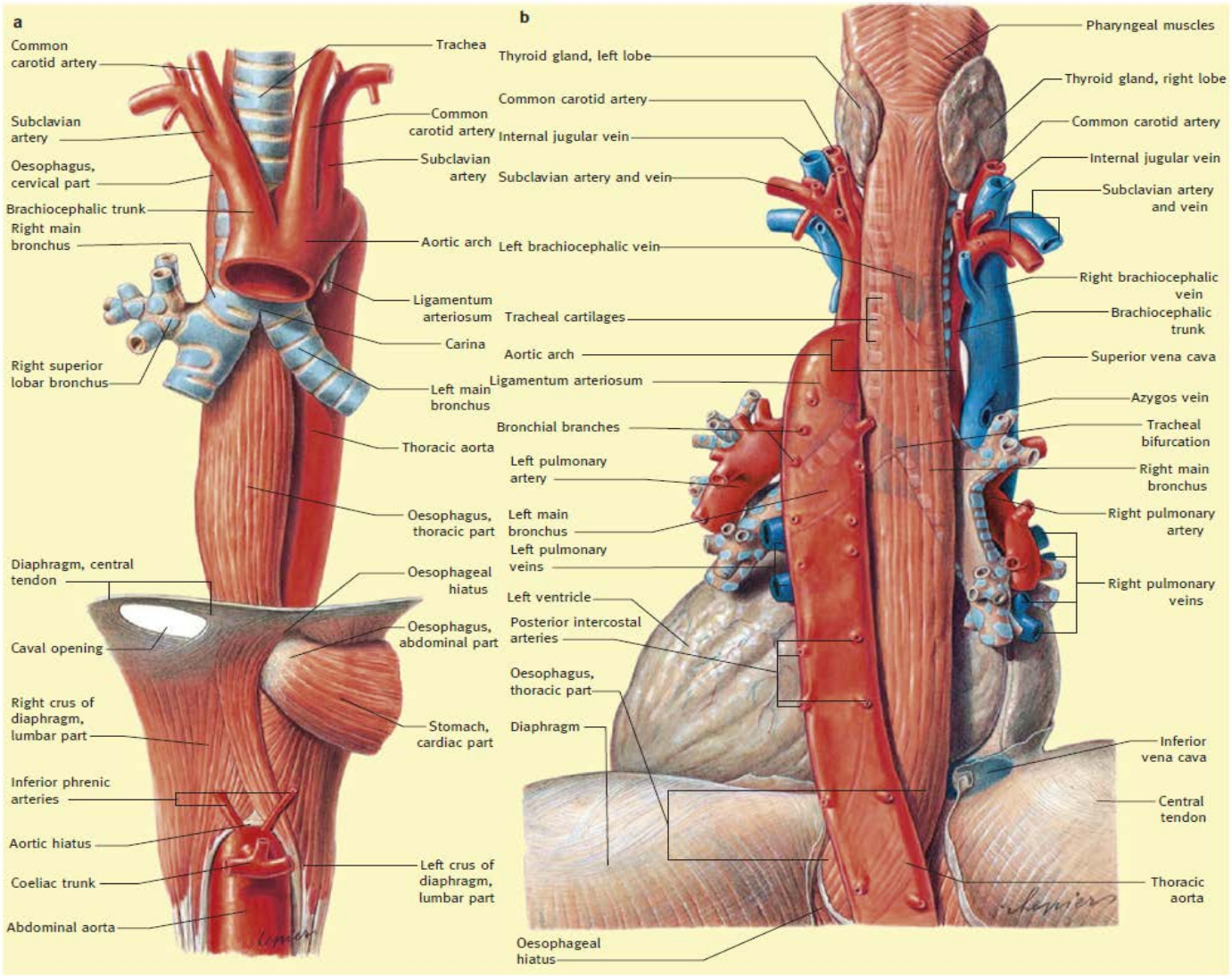
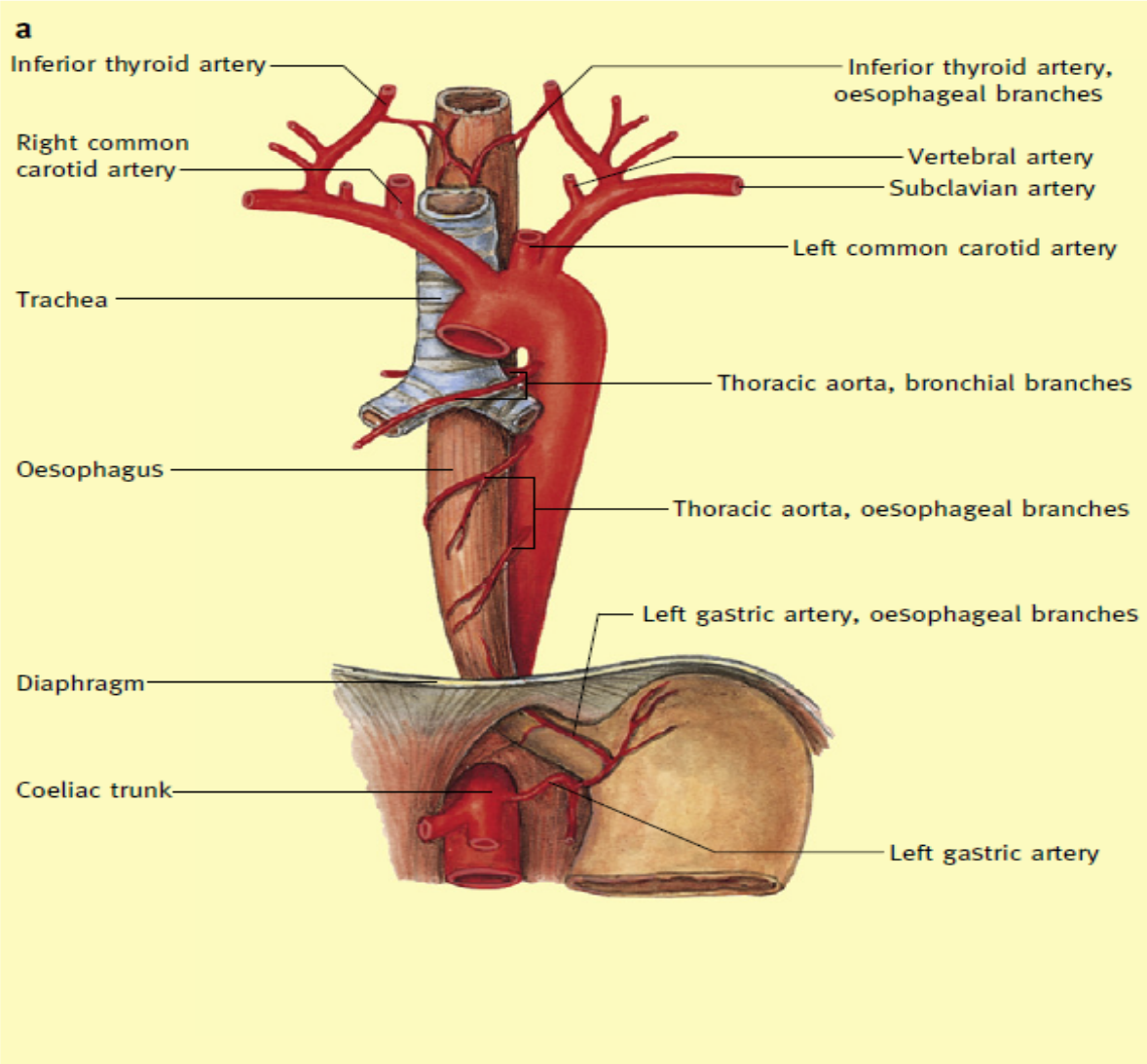


Figure 1 The oesophagus: anterior view (**a**), posterior view (**b**). (From Gray's Anatomy 40th edition).

ΠΑΘΗΣΕΙΣ ΟΙΣΟΦΑΓΟΥ

ΑΡΤΗΡΙΑΚΗ ΑΙΜΑΤΩΣΗ



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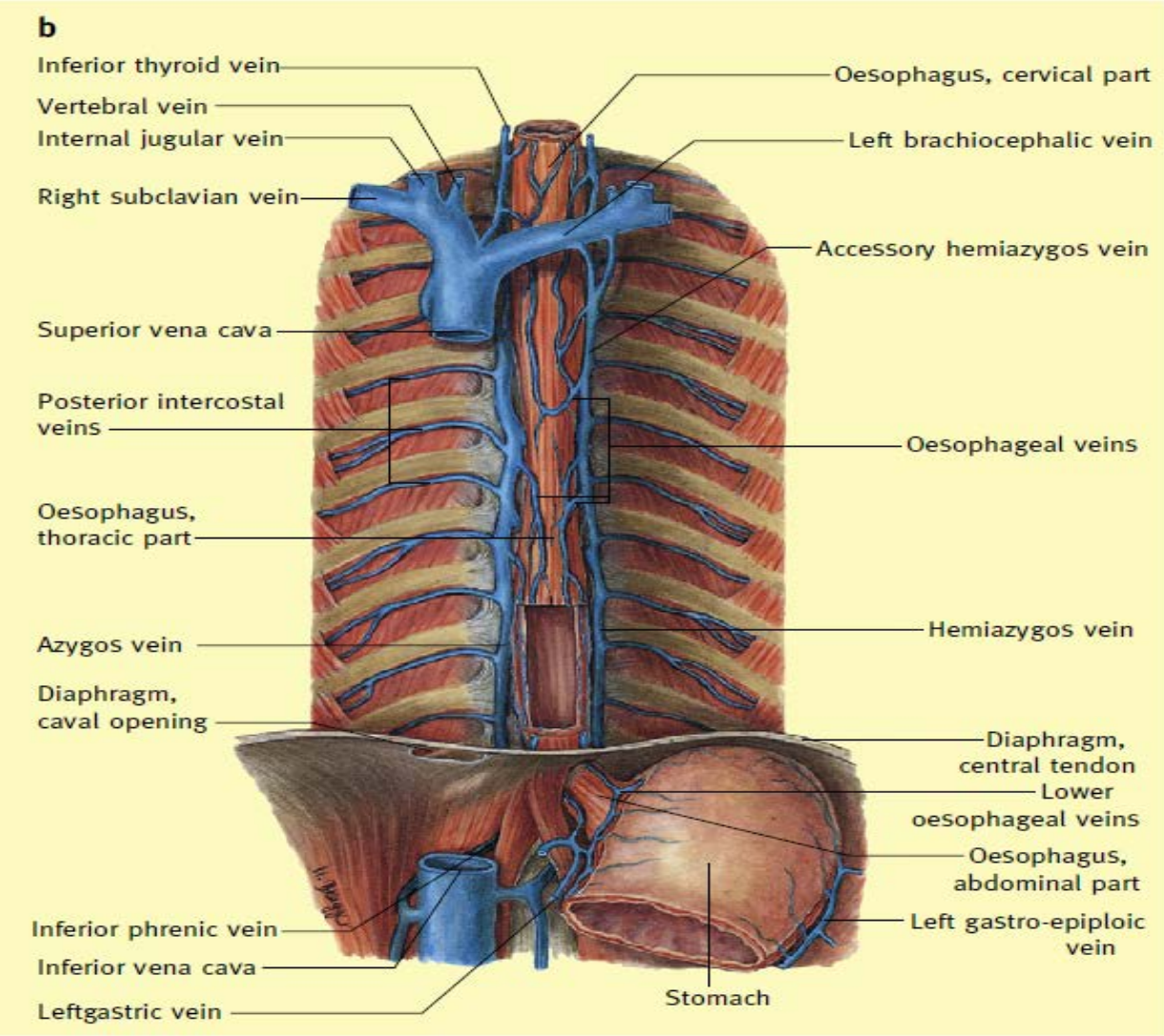

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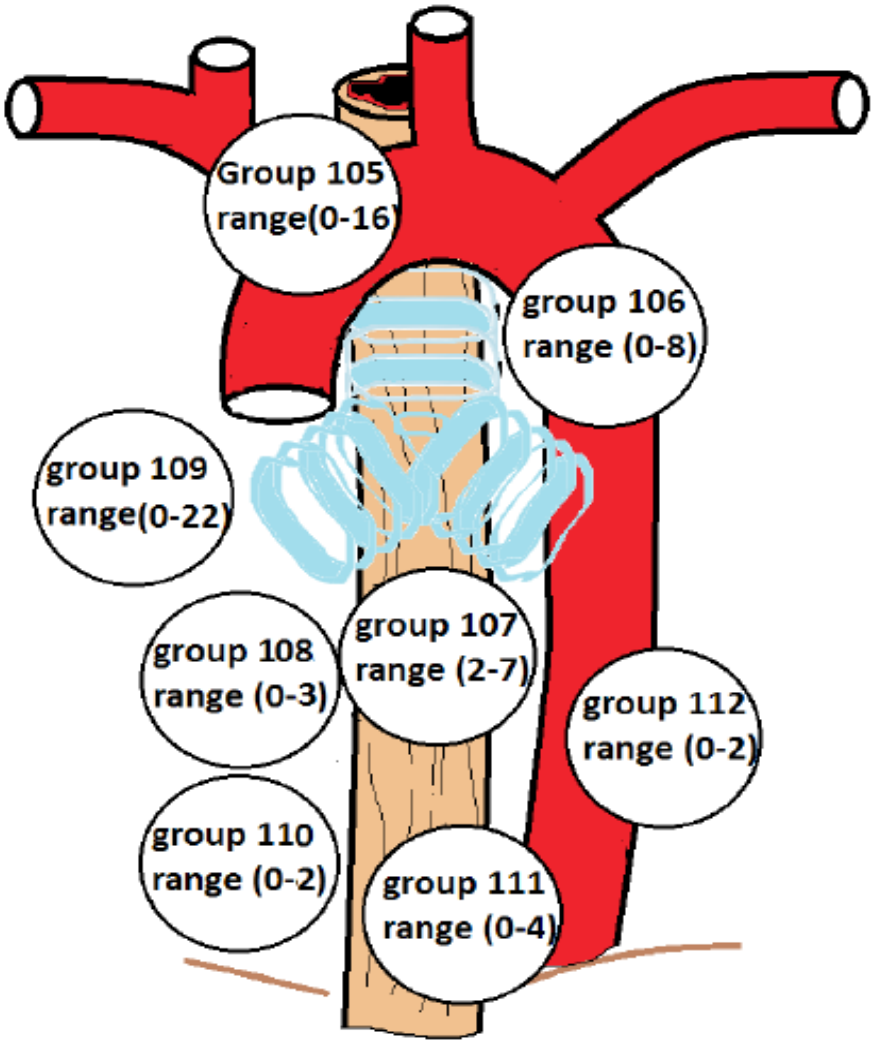

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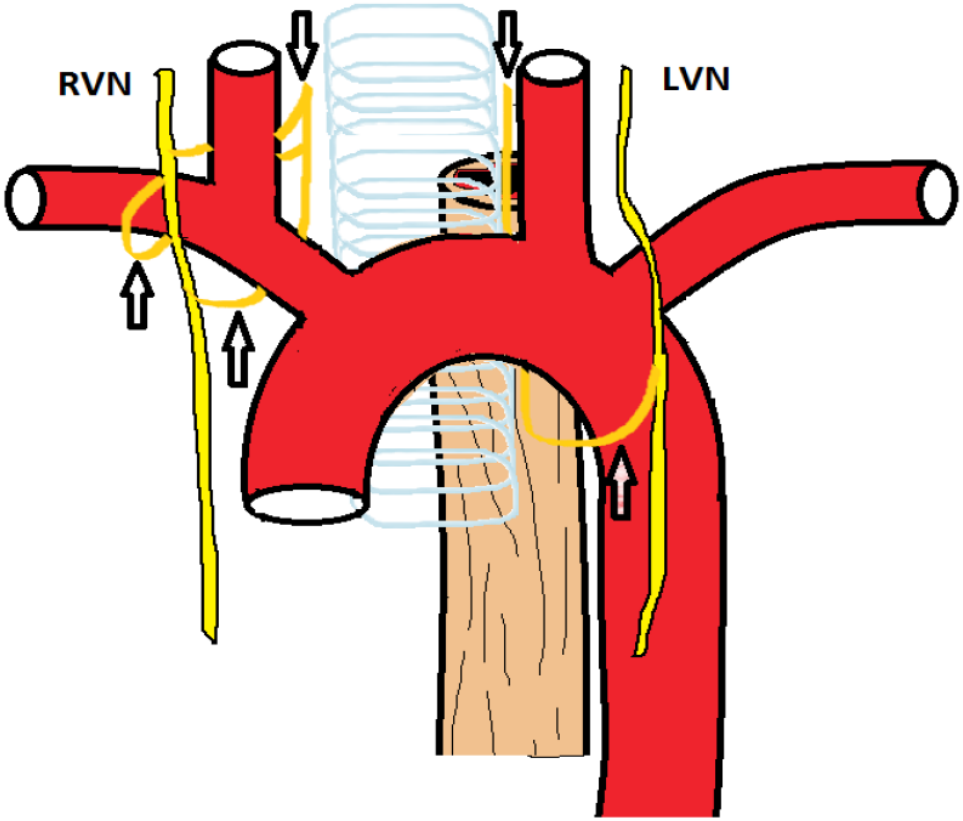
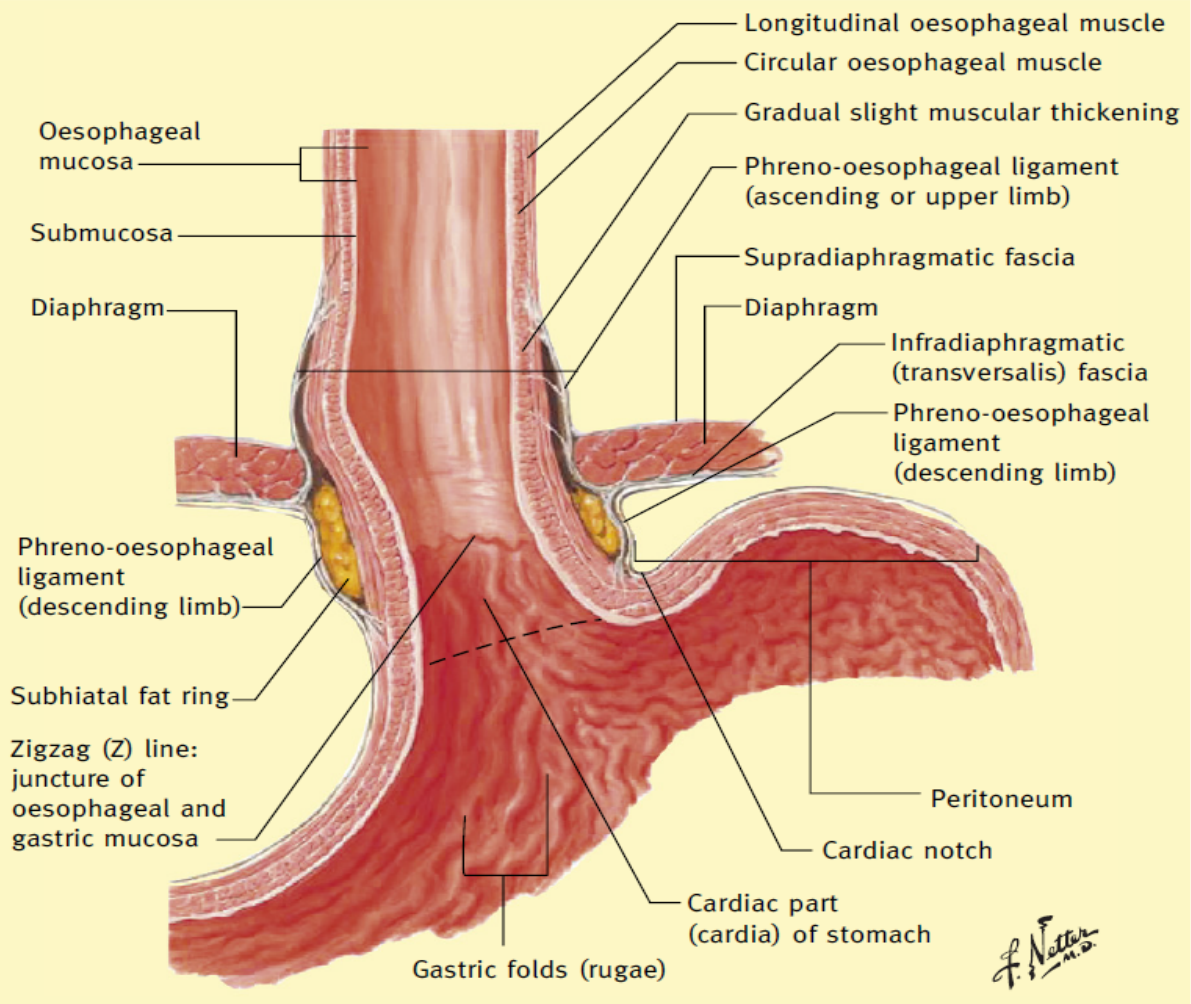
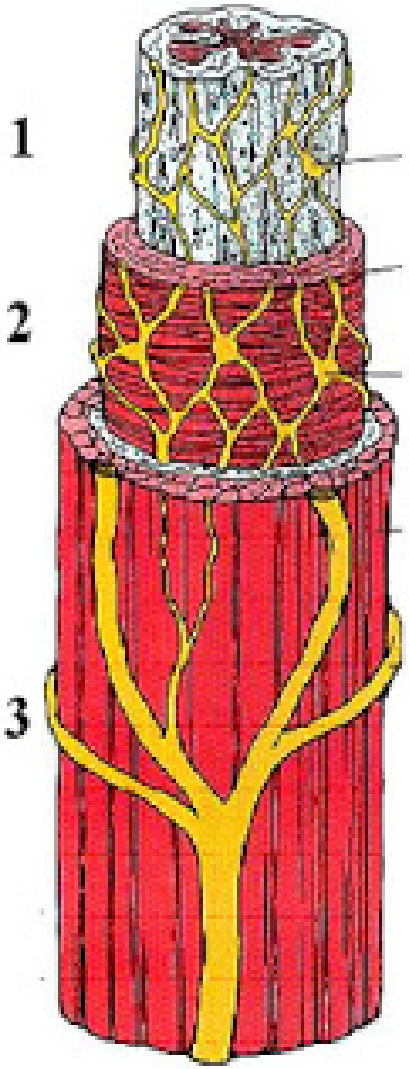


RVN: right vagus nerve. LVN: left vagus nerve. Arrow: recurrent laryngeal nerves

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

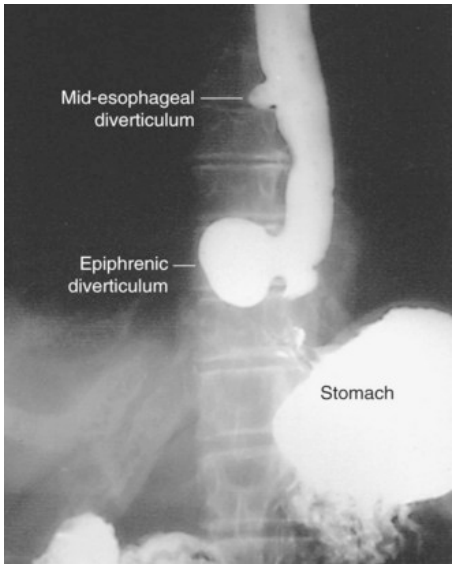
LONGITUDINAL MUSCLE: contraction causes shortening

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

ΕΚΚΟΛΠΩΜΑΤΑ ΟΙΣΟΦΑΓΟΥ

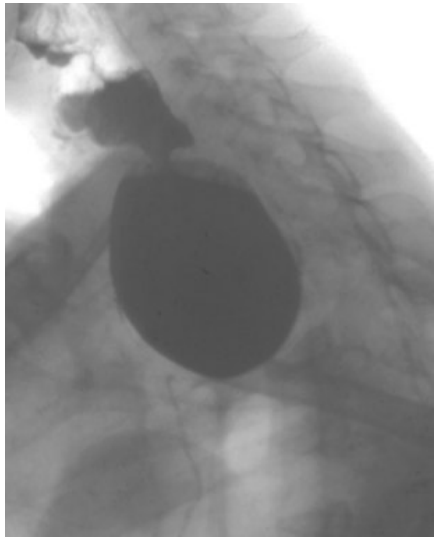
ΘΕΣΗ / ΜΗΧΑΝΙΣΜΟΣ

Zenker diverticulum	Immediately above the upper esophageal sphincter
Traction diverticulum	Near the midpoint of the esophagus
Epiphrenic diverticulum	Immediately above the lower esophageal sphincter

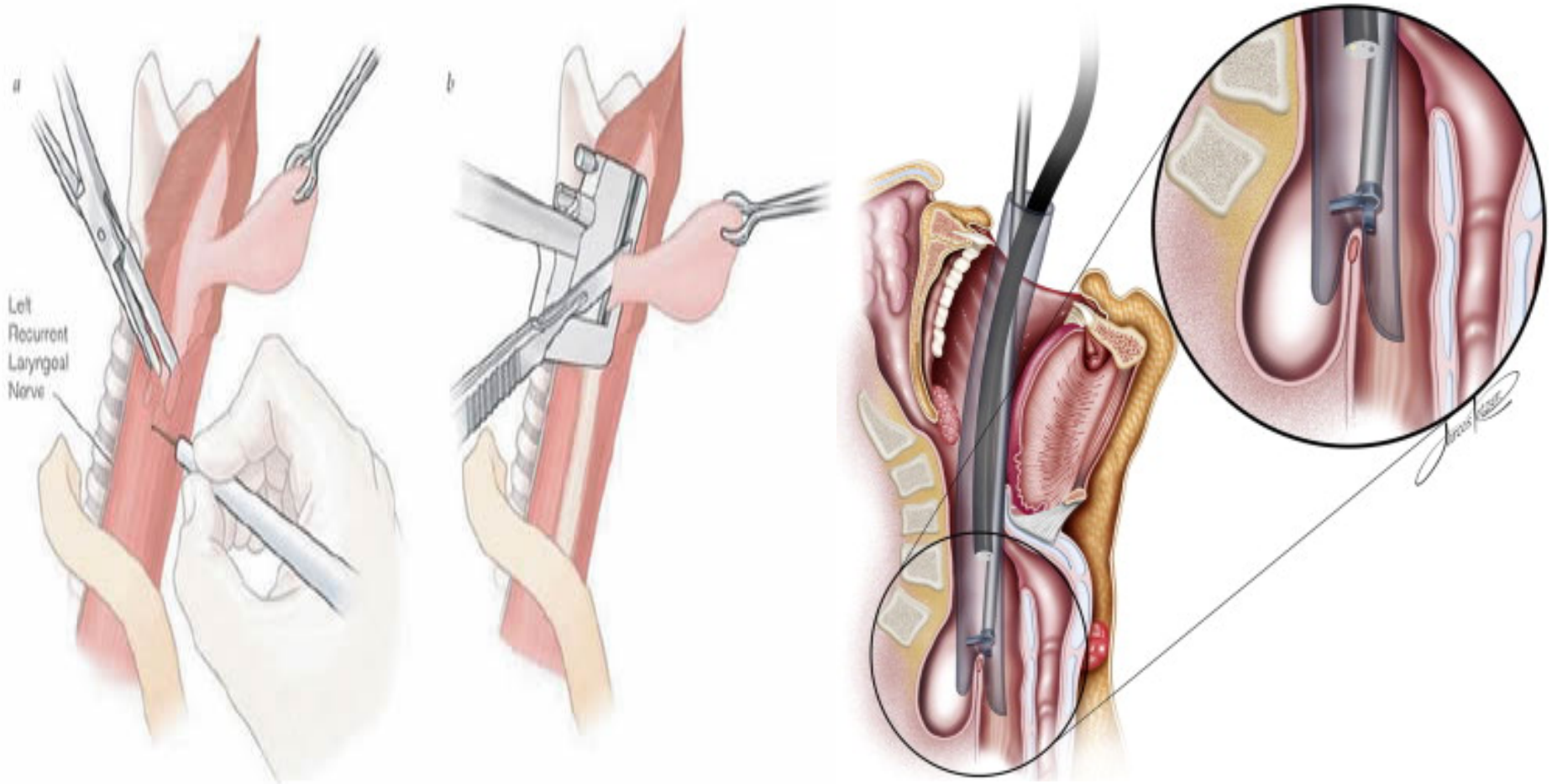


ΕΚΚΟΛΠΩΜΑ ZENKER

- 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 ZENKER – 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

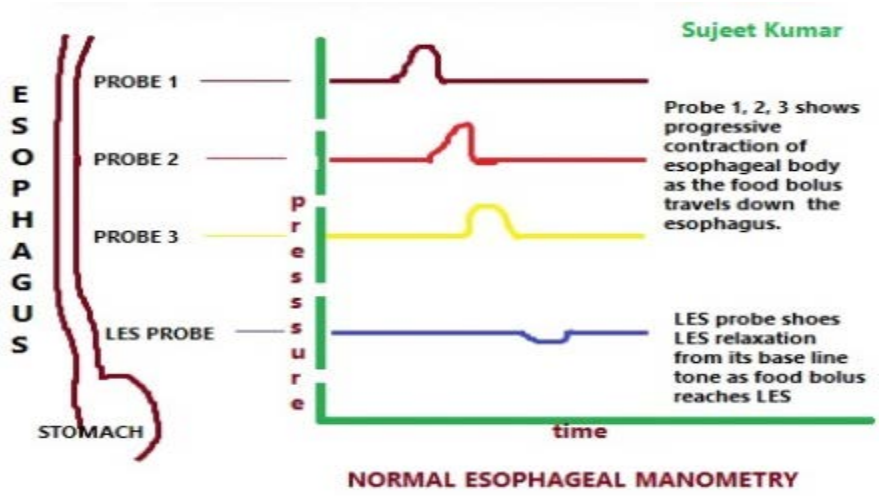
ΑΧΑΛΑΣΙΑ ΟΙΣΟΦΑΓΟΥ

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 Phμετρία
- **Μανομετρία**

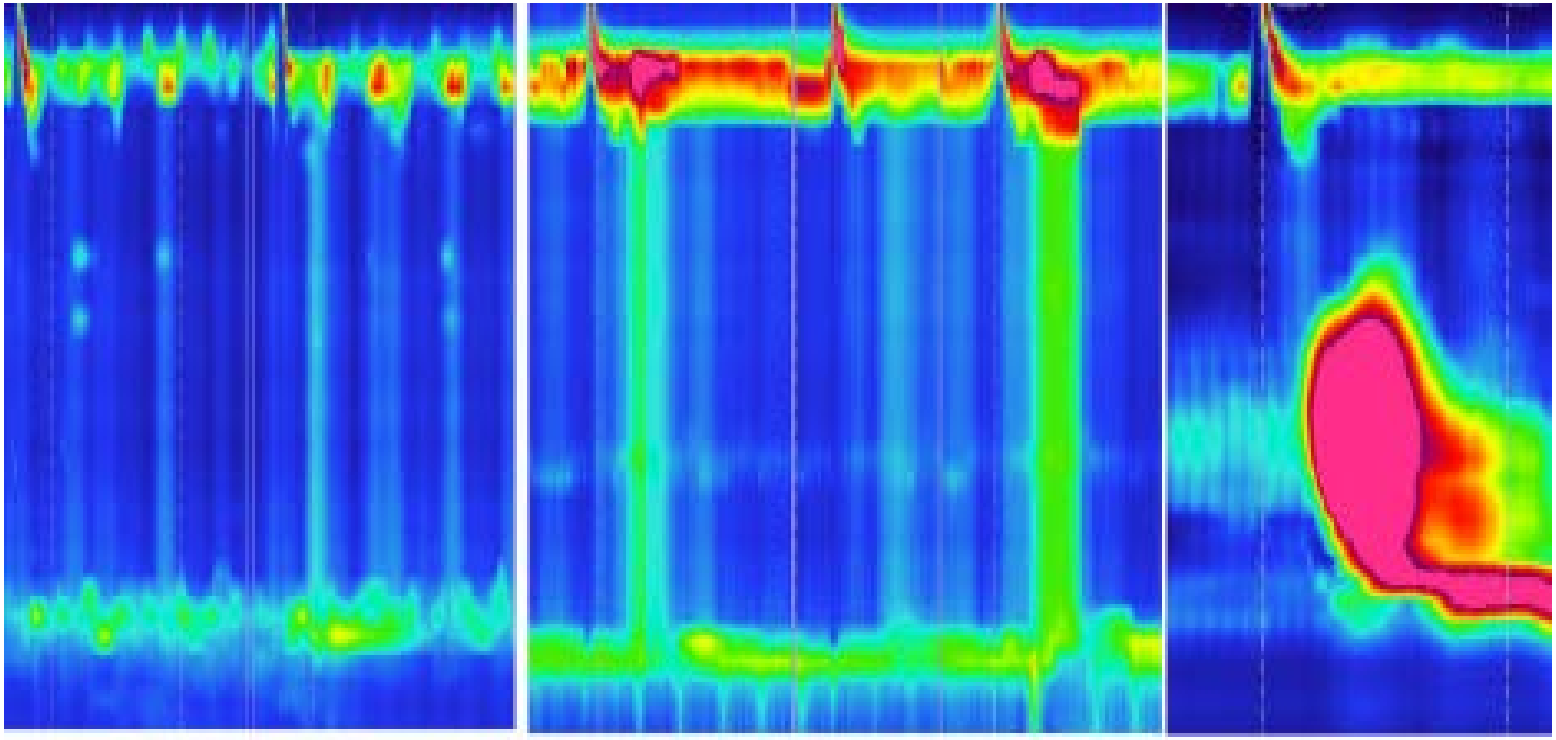


CHICAGO CLASSIFICATION

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 Panesophageal pressurization with ≥20% of swallows	
Type III achalasia	>15 mmHg	Absent peristalsis Premature contractions with ≥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 esophagealpressurization with swallows or MRS supports achalasia
Distal esophageal spasm	Normal or increased	≥20% premature contractions (DL < 4.5 s)	May be evolving type III achalasia
Jackhammer	Normal or increased	≥20% of swallows with DCI > 8000 mmHg s cm	May be evolving type III achalasia if DL < 4.5 s with ≥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

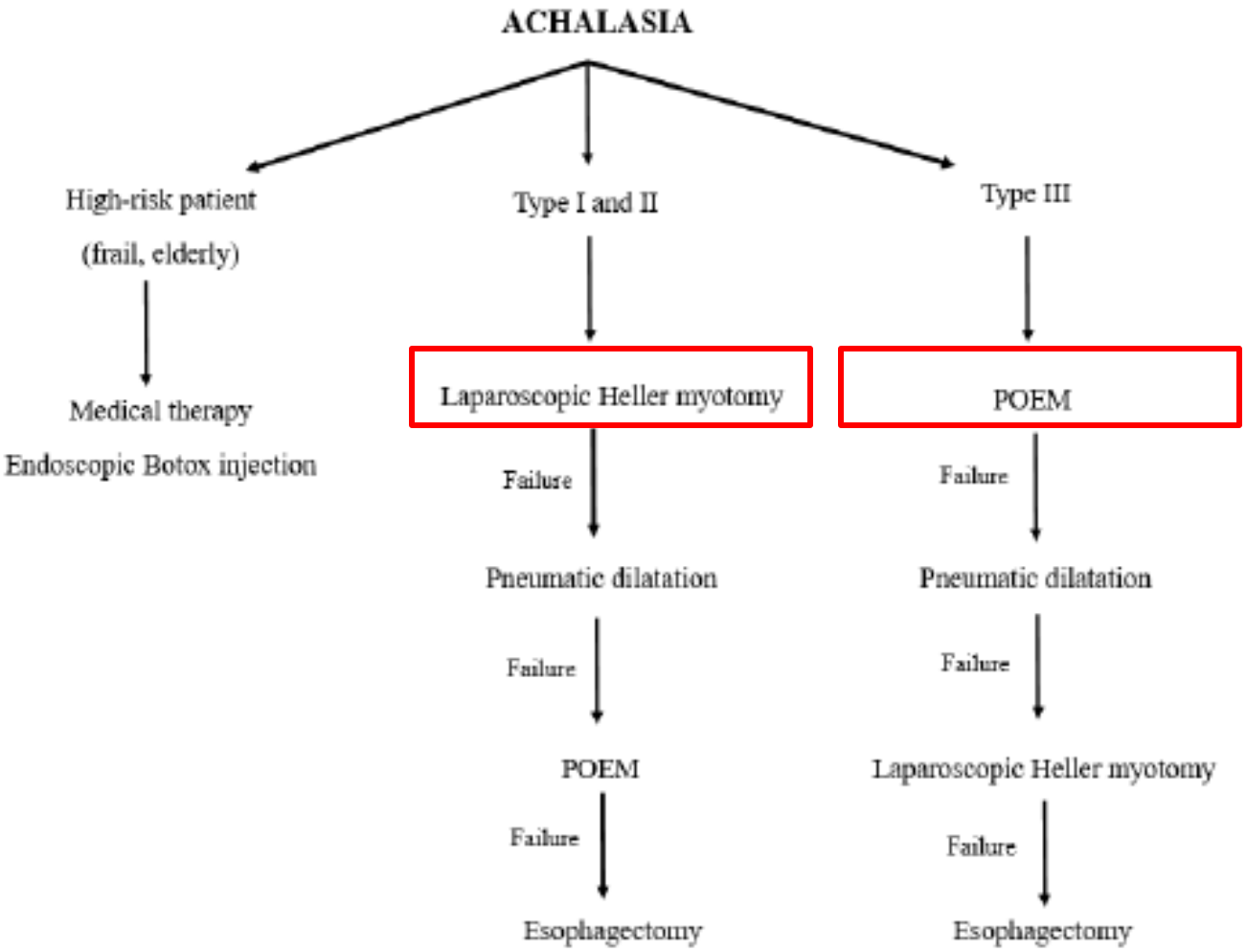


Type I

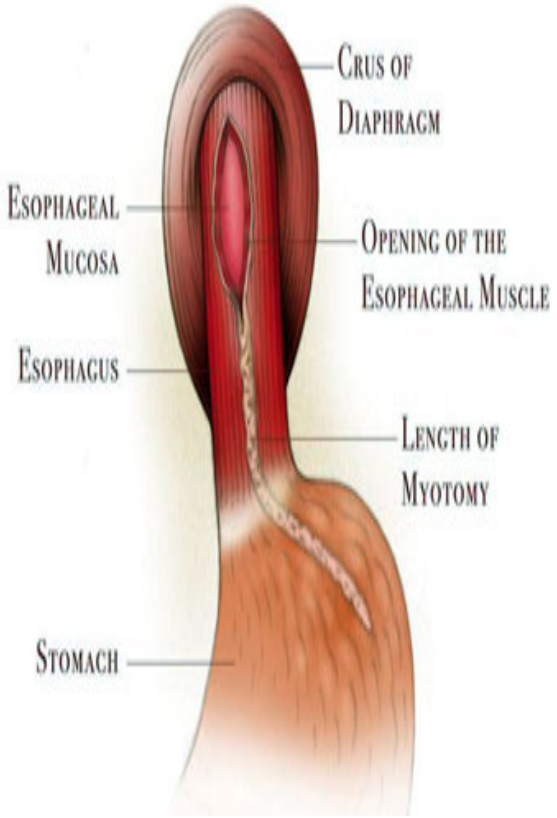
Type II

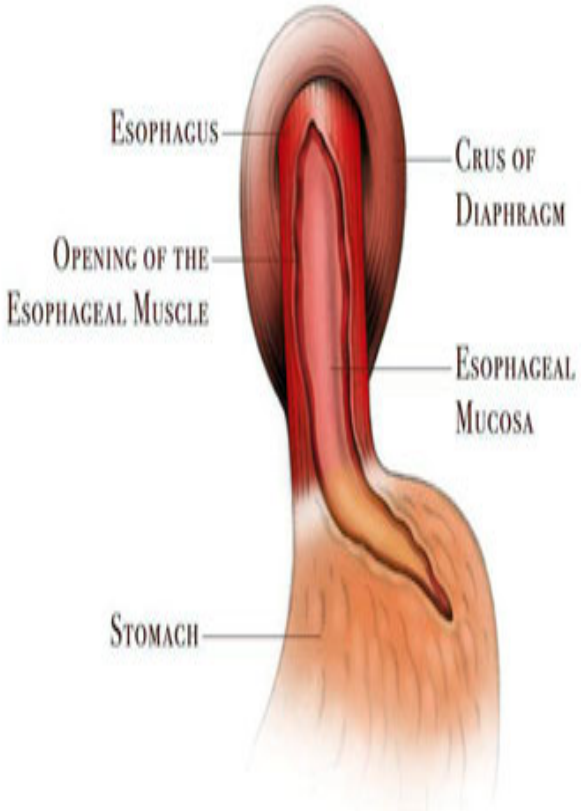
Type III

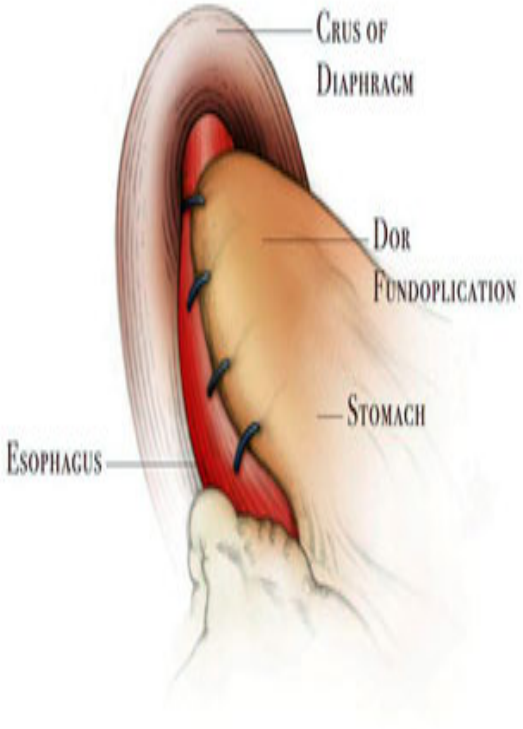
ΘΕΡΑΠΕΥΤΙΚΟΣ ΑΛΓΟΡΙΘΜΟΣ



HELLER MYOTOMY







ΓΑΣΤΡΟΟΙΣΟΦΑΓΙΚΗ ΠΑΛΙΝΔΡΟΜΙΚΗ ΝΟΣΟΣ

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)
- **PHμετρία (το gold-standard για τη διάγνωση)**

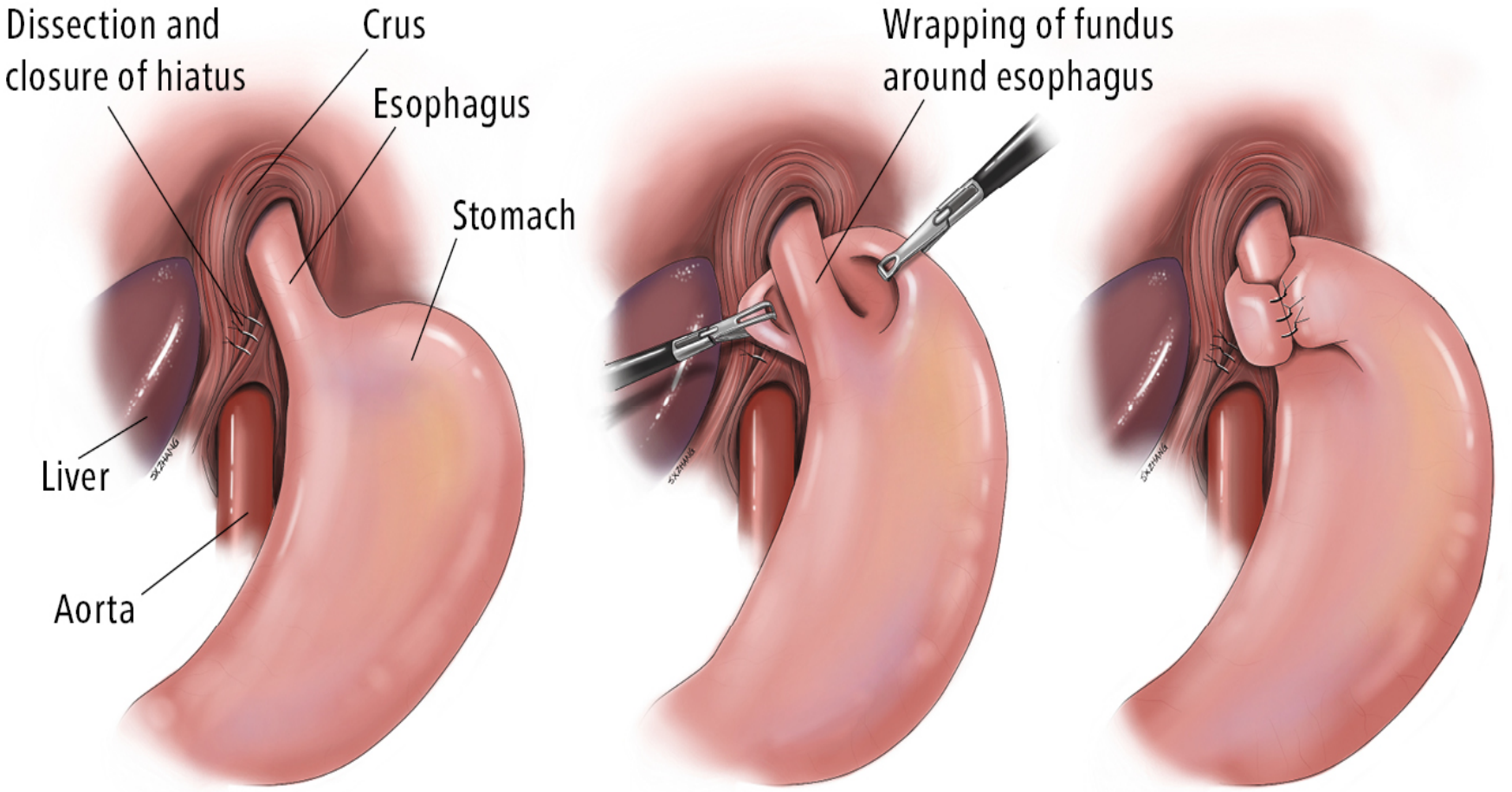
ΕΝΔΕΙΞΕΙΣ ΧΕΙΡΟΥΡΓΙΚΗΣ ΑΝΤΙΜΕΤΩΠΙΣΗΣ

- **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

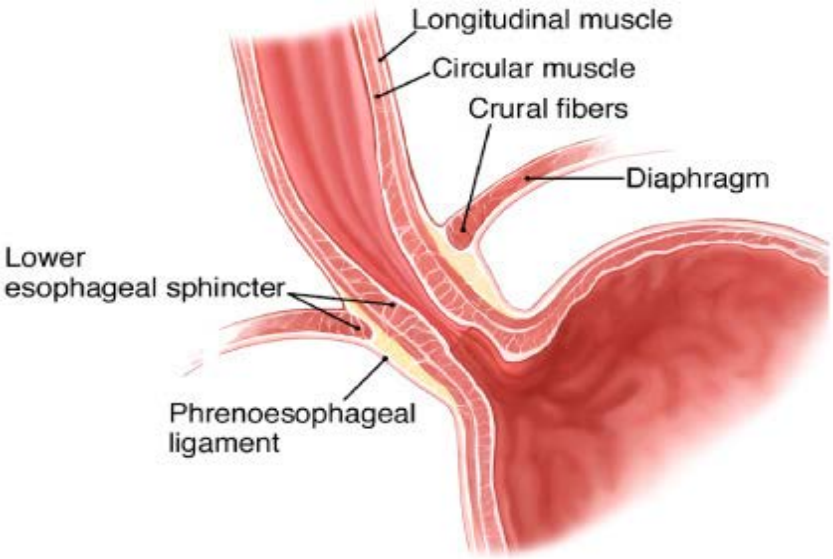
- Σήμερα αποτελεί **gold-standard η πλήρης (360°) θολοπλαστική κατά Nissen** σε όλους τους ασθενείς εκτός από αυτούς με αχαλασία ή σκληρόδερμα
- Η θολοπλαστική κατά Nissen προσφέρει καλύτερο έλεγχο της παλινδρόμησης από τις μερικές θολοπλαστικές, με τα ίδια ποσοστά μετεγχειρητικής δυσφαγίας
- Ειδικά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

ΘΟΛΟΠΛΑΣΤΙΚΗ ΚΑΤΑ NISSEN

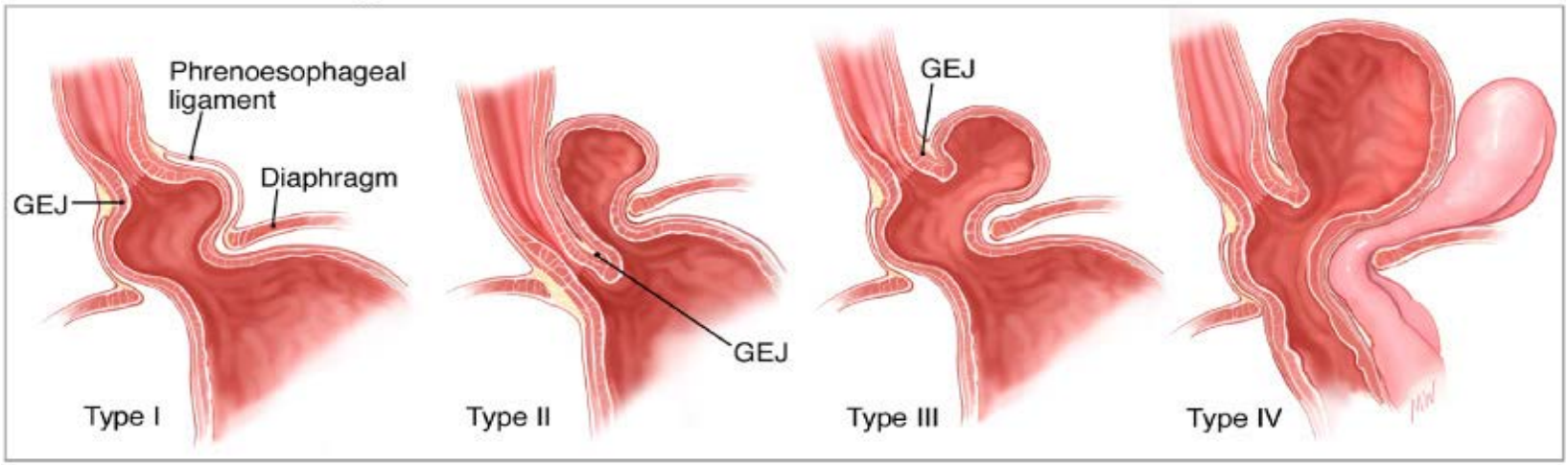


ΔΙΑΦΡΑΓΜΑΤΟΚΗΛΕΣ

ΤΥΠΟΙ ΔΙΑΦΡΑΓΜΑΤΟΚΗΛΗΣ



A



B

ΕΝΔΕΙΞΕΙΣ ΧΕΙΡΟΥΡΓΙΚΗΣ ΑΝΤΙΜΕΤΩΠΙΣΗΣ

- Η αποκατάσταση **κατ'επολίσθησιν διαφραγματοκήλης (Τύπου Ι)** απουσία ΓΟΠΝ δεν ενδείκνυται

- Όλες οι **συμπτωματικές παραοισοφαγικές διαφραγματοκήλης (Τύπου ΙΙ,ΙΙΙ,ΙV)** πρέπει να χειρουργούνται

- Στις **ασυμπτωματικές παραοισοφαγικές διαφραγματοκήλες (Τύπου ΙΙ,ΙΙΙ,ΙV)** 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

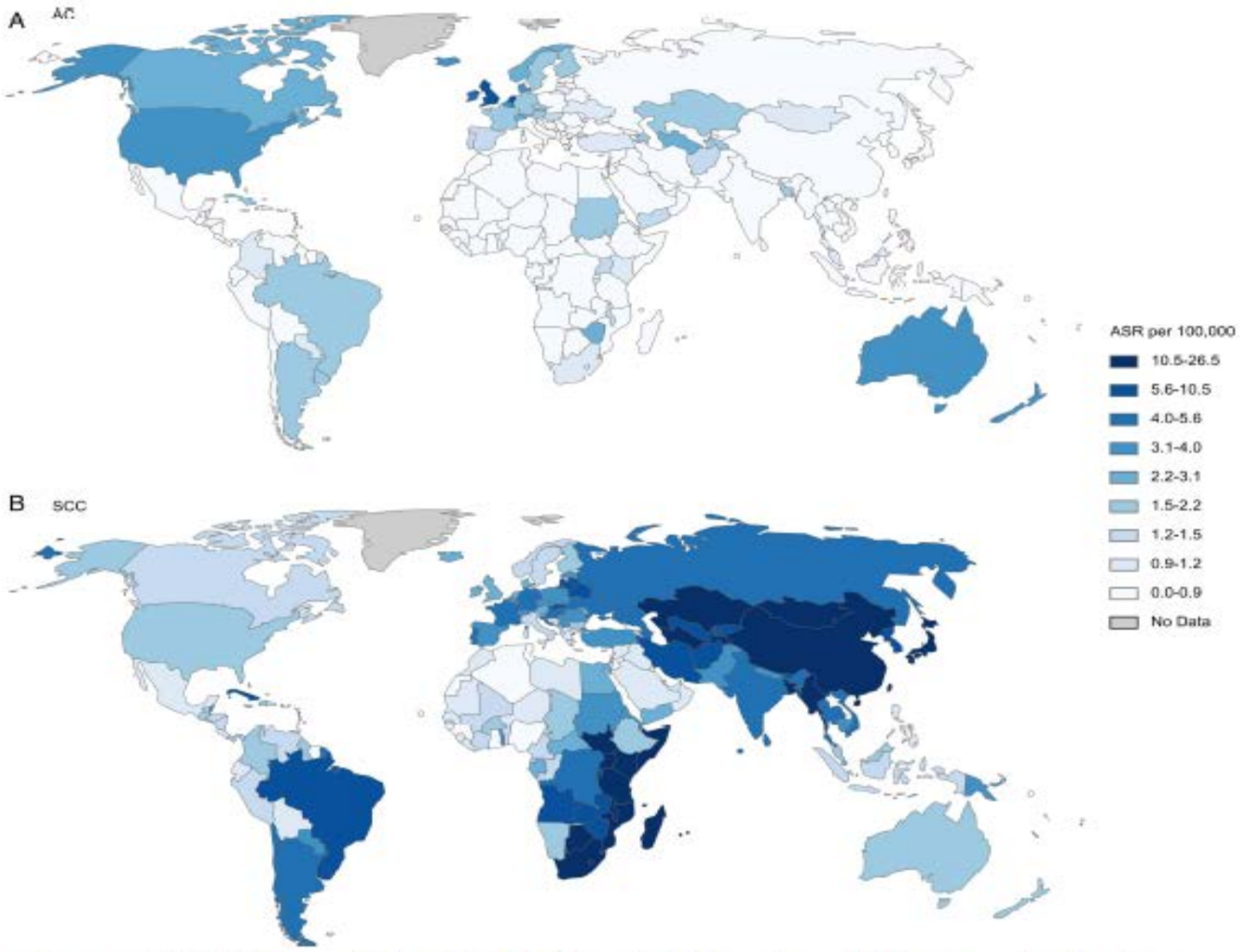


Figure 1 Age-standardised incidence rate (ASR) per 100 000 of (A) oesophageal adenocarcinoma and (B) squamous cell carcinoma in men. AC, adenocarcinoma; SCC, squamous cell carcinoma.

ΠΑΓΚΟΣΜΙΑ ΕΠΙΠΤΩΣΗ - ΓΥΝΑΙΚΕΣ

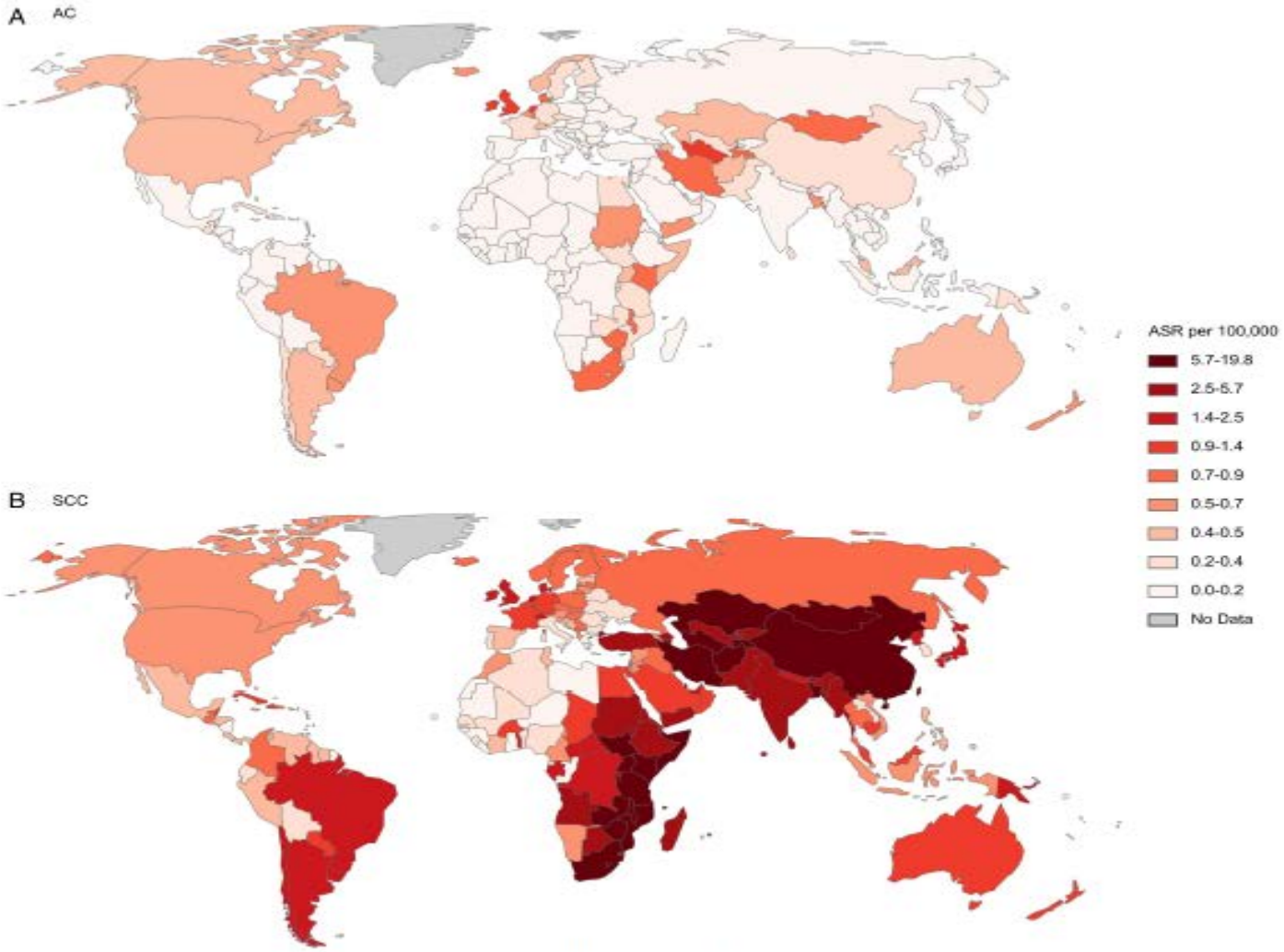


Figure 2 Age-standardised incidence rate (ASR) per 100 000 of (A) oesophageal adenocarcinoma and (B) squamous cell carcinoma in women. AC, adenocarcinoma; SCC, squamous cell carcinoma.

ΠΑΓΚΟΣΜΙΑ ΕΠΙΠΤΩΣΗ ΑΝΑ ΗΠΕΙΡΟ

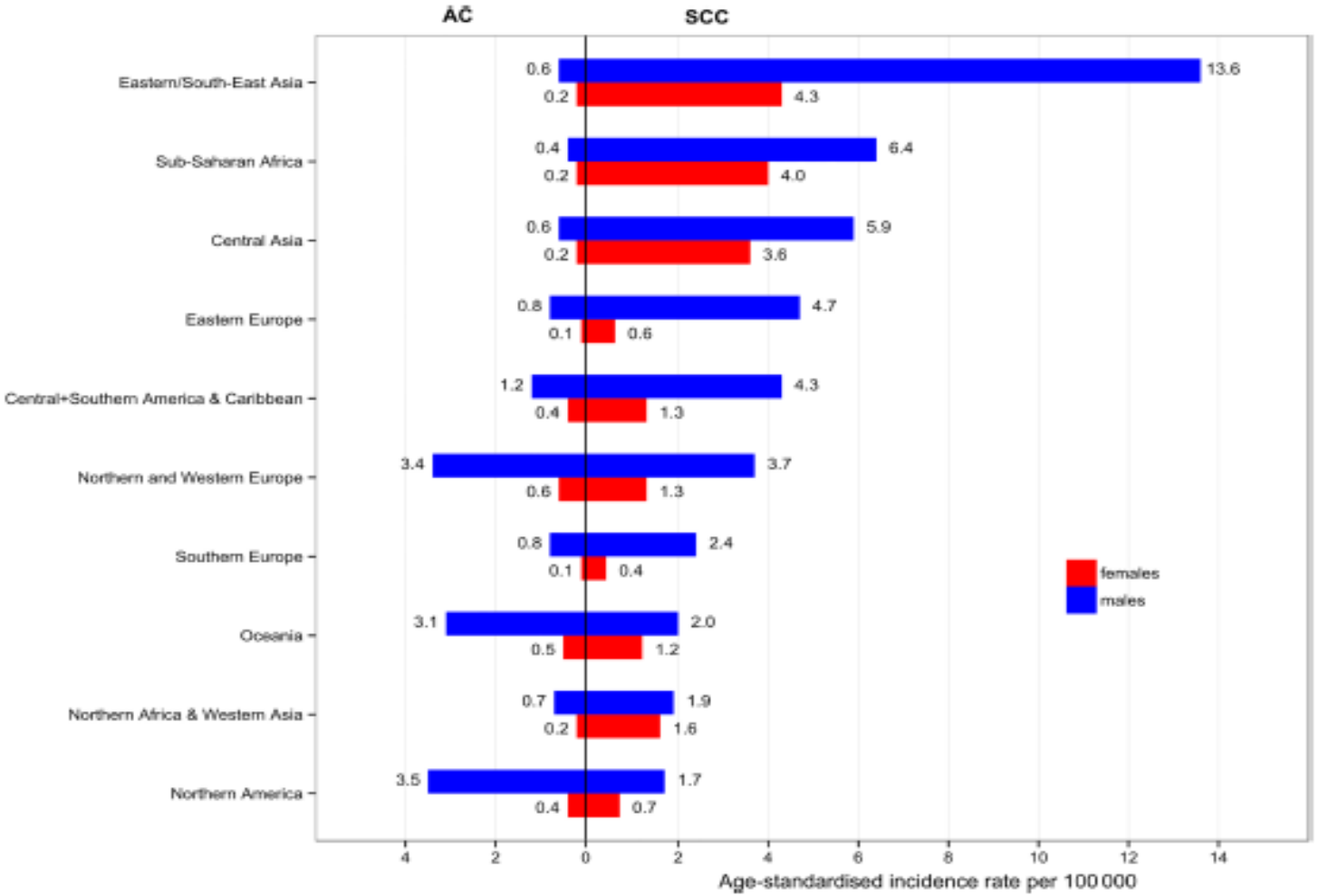


Figure 3 Age-standardised incidence rate per 100 000 of oesophageal cancer by histological subtype, region and sex. AC, adenocarcinoma; SCC, squamous cell carcinoma.

ΜΕΤΑΒΟΛΕΣ ΕΠΙΠΤΩΣΗΣ – USA - SWEDEN

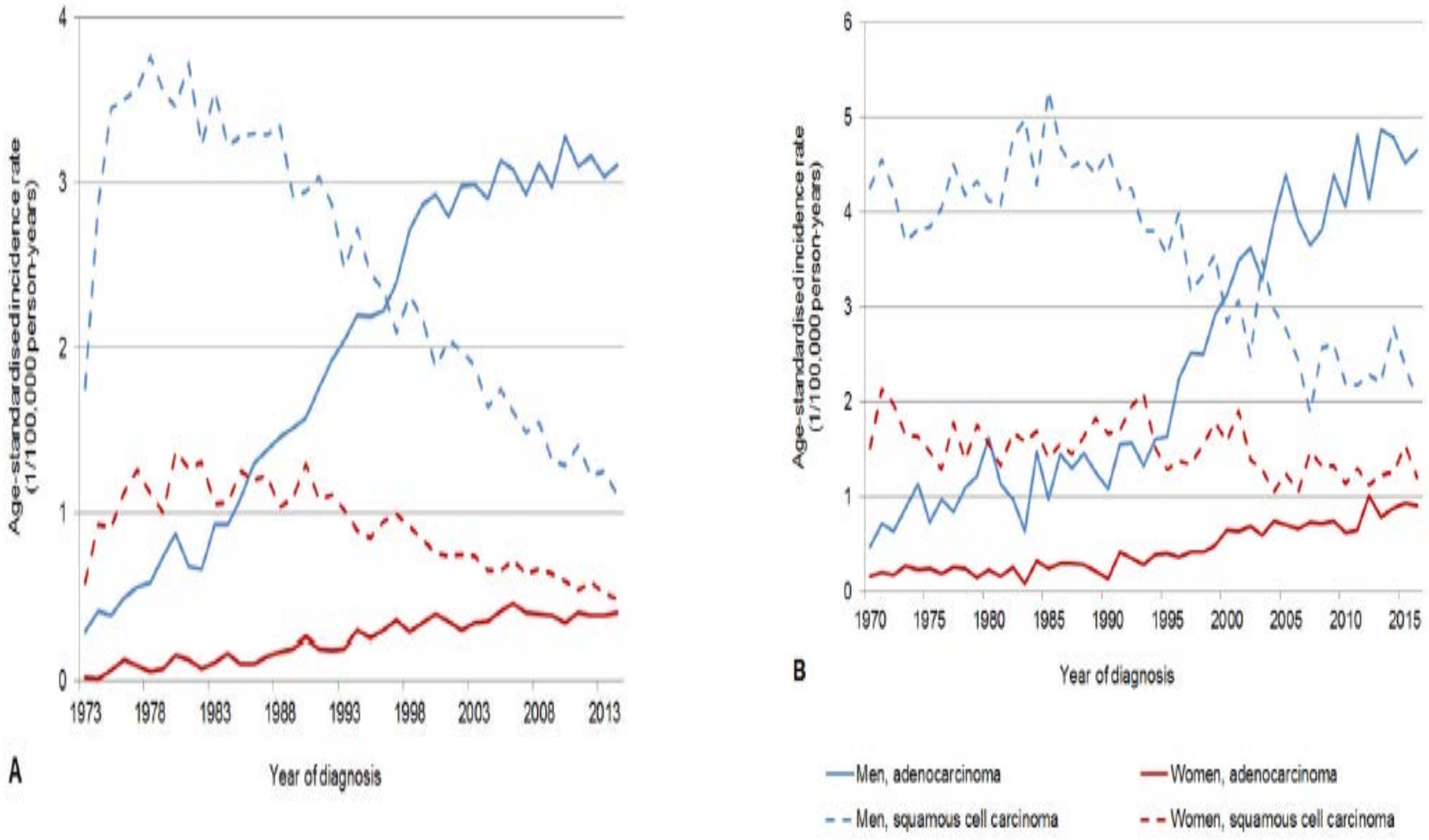


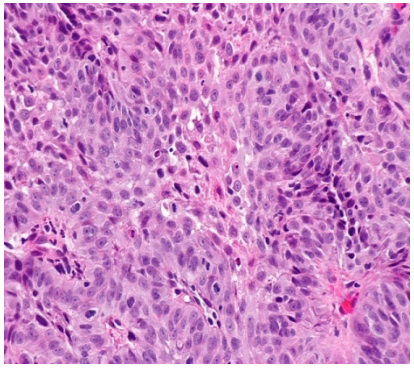
Fig. 1. Incidence trends of oesophageal squamous cell carcinoma and oesophageal adenocarcinoma in the United States (A) and in Sweden (B). Data sources: the Surveillance, Epidemiology, and End Results (SEER), SEER 9 registries research data, November 2017 submission; and the Swedish Cancer Registry.

ΚΑΡΚΙΝΟΣ ΟΙΣΟΦΑΓΟΥ

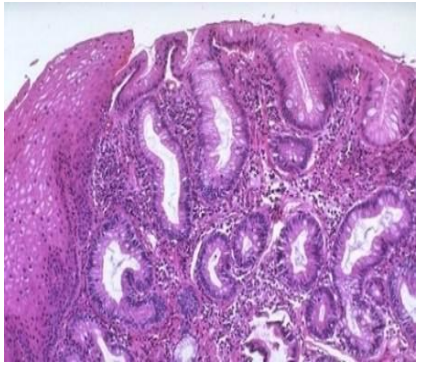
ΠΑΡΑΓΟΝΤΕΣ ΚΙΝΔΥΝΟΥ

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 Nyrén,[1] and Jesper Lagergren[1,2]

Departments of [1]Medical Epidemiology and Biostatistics and [2]Surgical 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[1]	(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)
P[5]		0.01		0.02	
Education (y)					
High (≥13)	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	

ΠΑΘΗΣΕΙΣ ΟΙΣΟΦΑΓΟΥ

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Review

REVIEW

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

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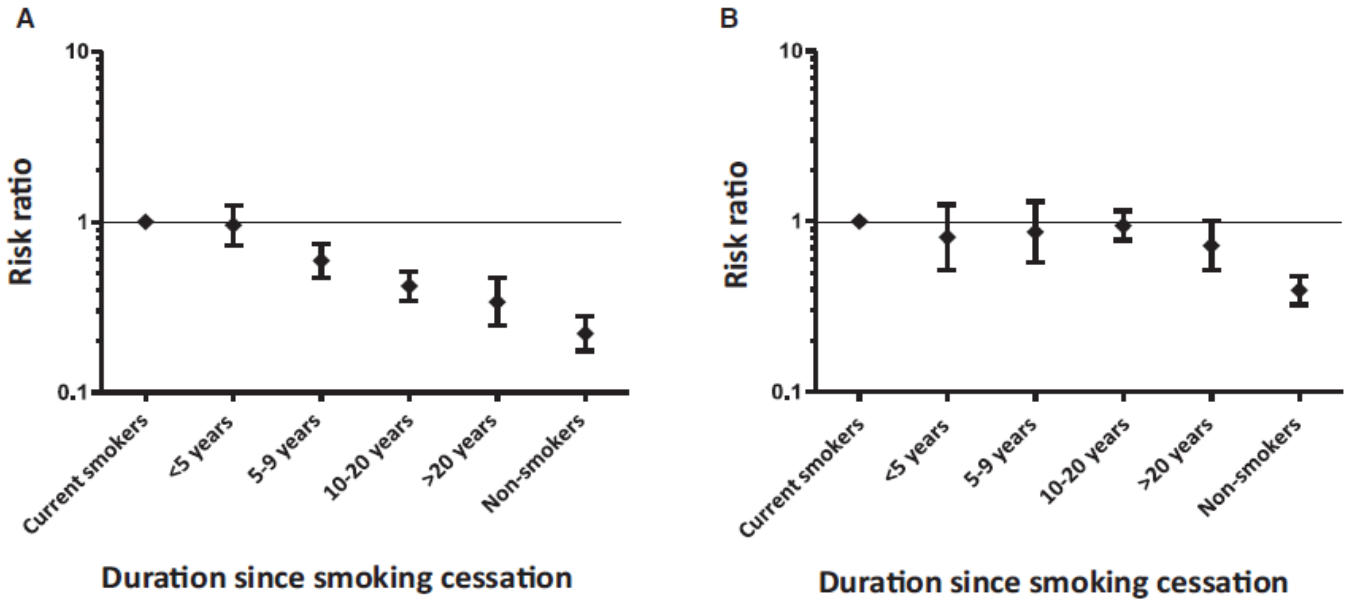

Figure 3. Risk ratio of esophageal squamous cell carcinoma and adenocarcinoma by duration since smoking cessation, using current smokers as reference. **A)** Effect sizes for esophageal squamous cell carcinoma. **B)** Effect sizes for esophageal adenocarcinoma. **Error bars** = 95% confidence interval.

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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 Fedirko[1], Irene Tramacere[4], Vincenzo Bagnardi[5,6], Mazda Jenab[1], Lorenza Scotti[5], Matteo Rota[5,7], Giovanni Corrao[5], Werner Garavello[4,8], Joachim Schüz[1], Kurt Straif[1], Eva Negri[4], 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)

[1]Amount 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. [2]Only 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. [3]Studies that provided risk estimates for esophageal squamous cell carcinoma only. [4]Adjusted at least for age, sex, and tobacco use.

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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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SYMPTOMATIC GASTROESOPHAGEAL REFLUX AS A RISK FACTOR FOR ESOPHAGEAL ADENOCARCINOMA

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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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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 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)

ΠΑΘΗΣΕΙΣ ΟΙΣΟΦΑΓΟΥ

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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 Chow[2] and Marilie D Gammon[16]

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)

	OA						OGJA						All adenocarcinomas					
BMI (kg/m^2)	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	10481	1897	1.09	1.06–1.12	76	12	10295	1822	1.07	1.05–1.09	54	10	10481	3719	1.08	1.06–1.10	75	12

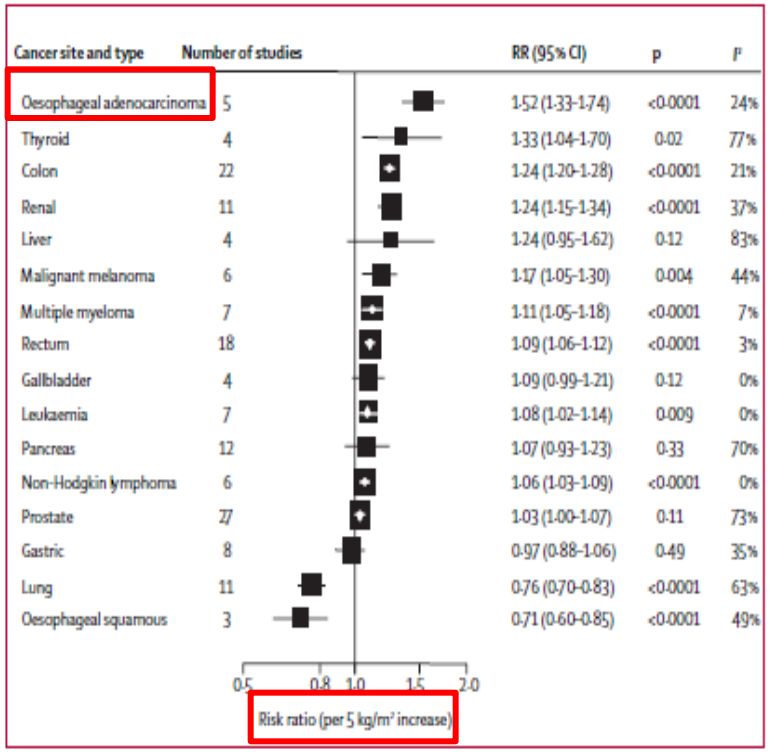
Results were adjusted for age (categorical: <50, 50–59, 60–69, ⩾70 years), gender, pack-years of smoking (categorical: <15, 15 to <30, 30 to <45, ⩾45), education (study-specific) and other study-specific adjustment variables (e.g. study centre, where applicable). Summary odds ratios and 95% confidence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

I^2 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.

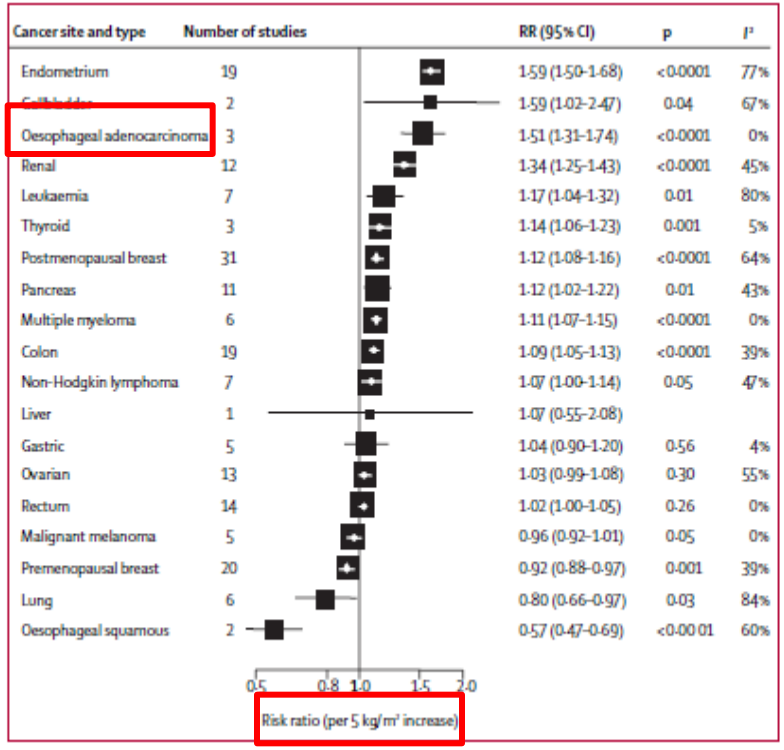
Body-mass index and incidence of cancer: a systematic review and meta-analysis of prospective observational studies

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



Cancer site and type	Number of studies	RR (95% CI)	p	I^2
Oesophageal adenocarcinoma	5	1·52 (1·33–1·74)	<0·0001	24%
Thyroid	4	1·33 (1·04–1·70)	0·02	77%
Colon	22	1·24 (1·20–1·28)	<0·0001	21%
Renal	11	1·24 (1·15–1·34)	<0·0001	37%
Liver	4	1·24 (0·95–1·62)	0·12	83%
Malignant melanoma	6	1·17 (1·05–1·30)	0·004	44%
Multiple myeloma	7	1·11 (1·05–1·18)	<0·0001	7%
Rectum	18	1·09 (1·06–1·12)	<0·0001	3%
Gallbladder	4	1·09 (0·99–1·21)	0·12	0%
Leukaemia	7	1·08 (1·02–1·14)	0·009	0%
Pancreas	12	1·07 (0·93–1·23)	0·33	70%
Non-Hodgkin lymphoma	6	1·06 (1·03–1·09)	<0·0001	0%
Prostate	27	1·03 (1·00–1·07)	0·11	73%
Gastric	8	0·97 (0·88–1·06)	0·49	35%
Lung	11	0·76 (0·70–0·83)	<0·0001	63%
Oesophageal squamous	3	0·71 (0·60–0·85)	<0·0001	49%

Figure 3: Summary risk estimates by cancer sites in men



Cancer site and type	Number of studies	RR (95% CI)	p	I^2
Endometrium	19	1·59 (1·50–1·68)	<0·0001	77%
Gallbladder	2	1·59 (1·02–2·47)	0·04	67%
Oesophageal adenocarcinoma	3	1·51 (1·31–1·74)	<0·0001	0%
Renal	12	1·34 (1·25–1·43)	<0·0001	45%
Leukaemia	7	1·17 (1·04–1·32)	0·01	80%
Thyroid	3	1·14 (1·06–1·23)	0·001	5%
Postmenopausal breast	31	1·12 (1·08–1·16)	<0·0001	64%
Pancreas	11	1·12 (1·02–1·22)	0·01	43%
Multiple myeloma	6	1·11 (1·07–1·15)	<0·0001	0%
Colon	19	1·09 (1·05–1·13)	<0·0001	39%
Non-Hodgkin lymphoma	7	1·07 (1·00–1·14)	0·05	47%
Liver	1	1·07 (0·55–2·08)		
Gastric	5	1·04 (0·90–1·20)	0·56	4%
Ovarian	13	1·03 (0·99–1·08)	0·30	55%
Rectum	14	1·02 (1·00–1·05)	0·26	0%
Malignant melanoma	5	0·96 (0·92–1·01)	0·05	0%
Premenopausal breast	20	0·92 (0·88–0·97)	0·001	39%
Lung	6	0·80 (0·66–0·97)	0·03	84%
Oesophageal squamous	2	0·57 (0·47–0·69)	<0·00 01	60%

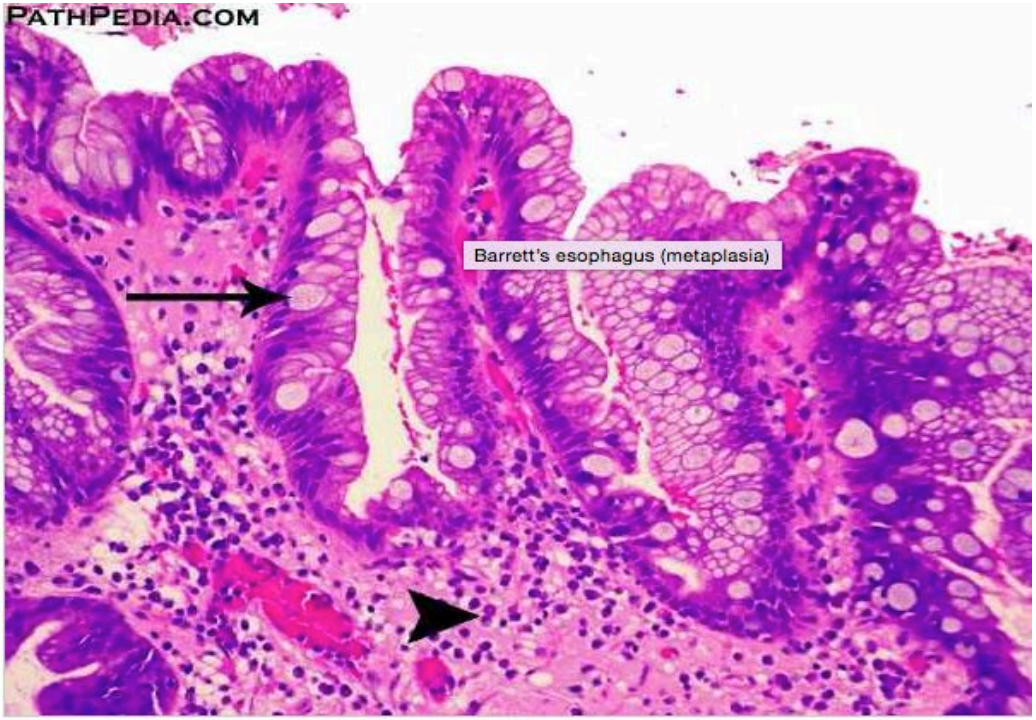
Figure 4: Summary risk estimates by cancer sites in women

ΠΑΘΗΣΕΙΣ ΟΙΣΟΦΑΓΟΥ

ΚΑΡΚΙΝΟΣ ΟΙΣΟΦΑΓΟΥ

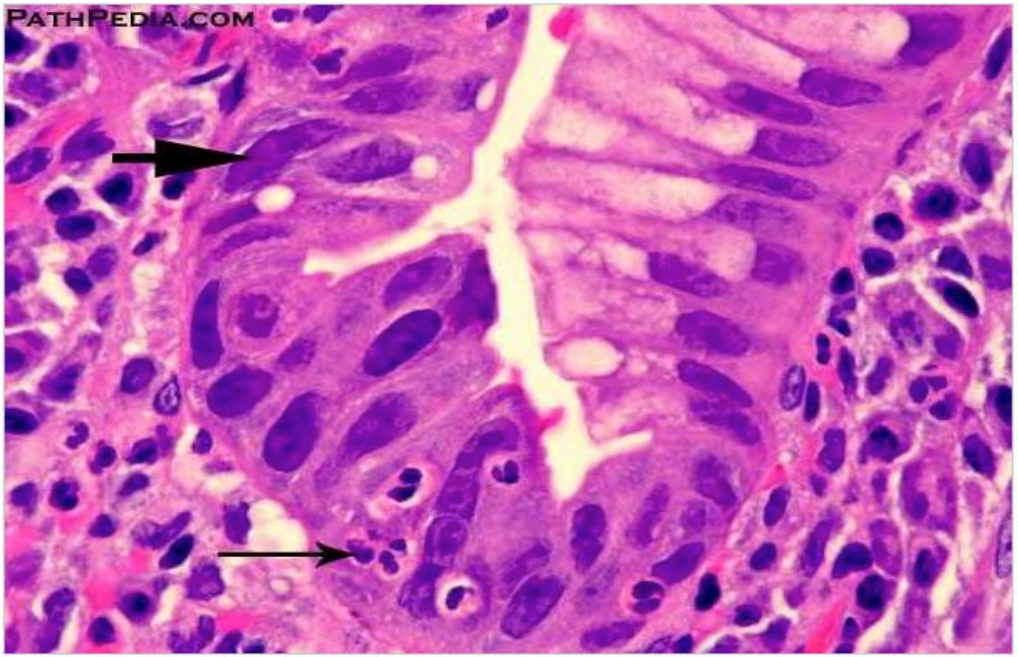
ΟΙΣΟΦΑΓΟΣ BARRETT

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

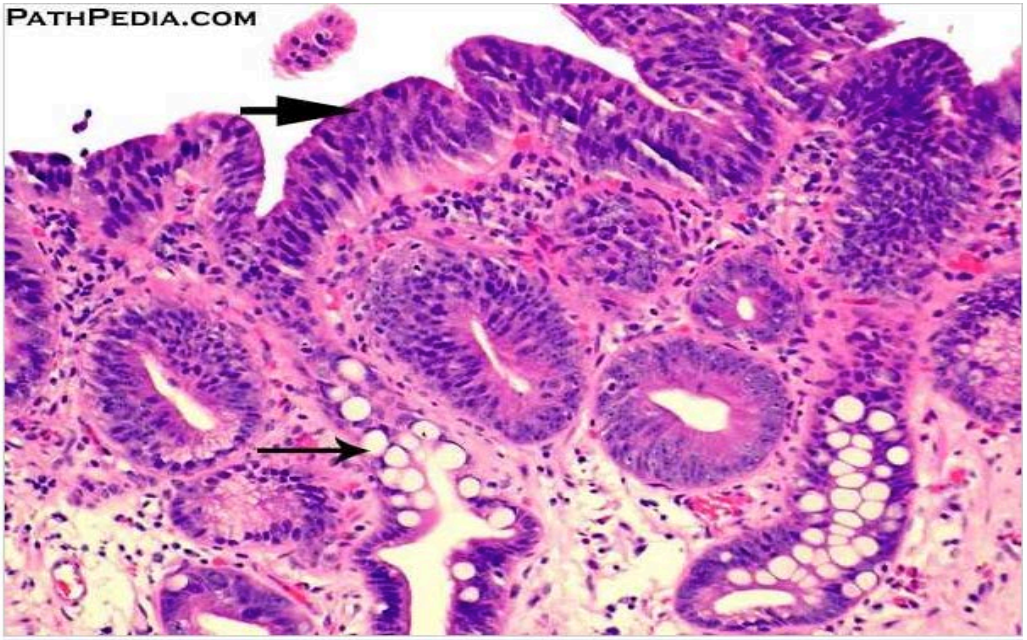


[BARRETT'S METAPLASIA]. Inflammation present in Barrett's metaplasia may lead to nuclear atypia of varying degrees. In some cases the nuclear atypia simulates dysplasia but histologic features diagnostic of dysplasia are not seen. Also, this is one area in diagnostic histopathology where consensus among 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

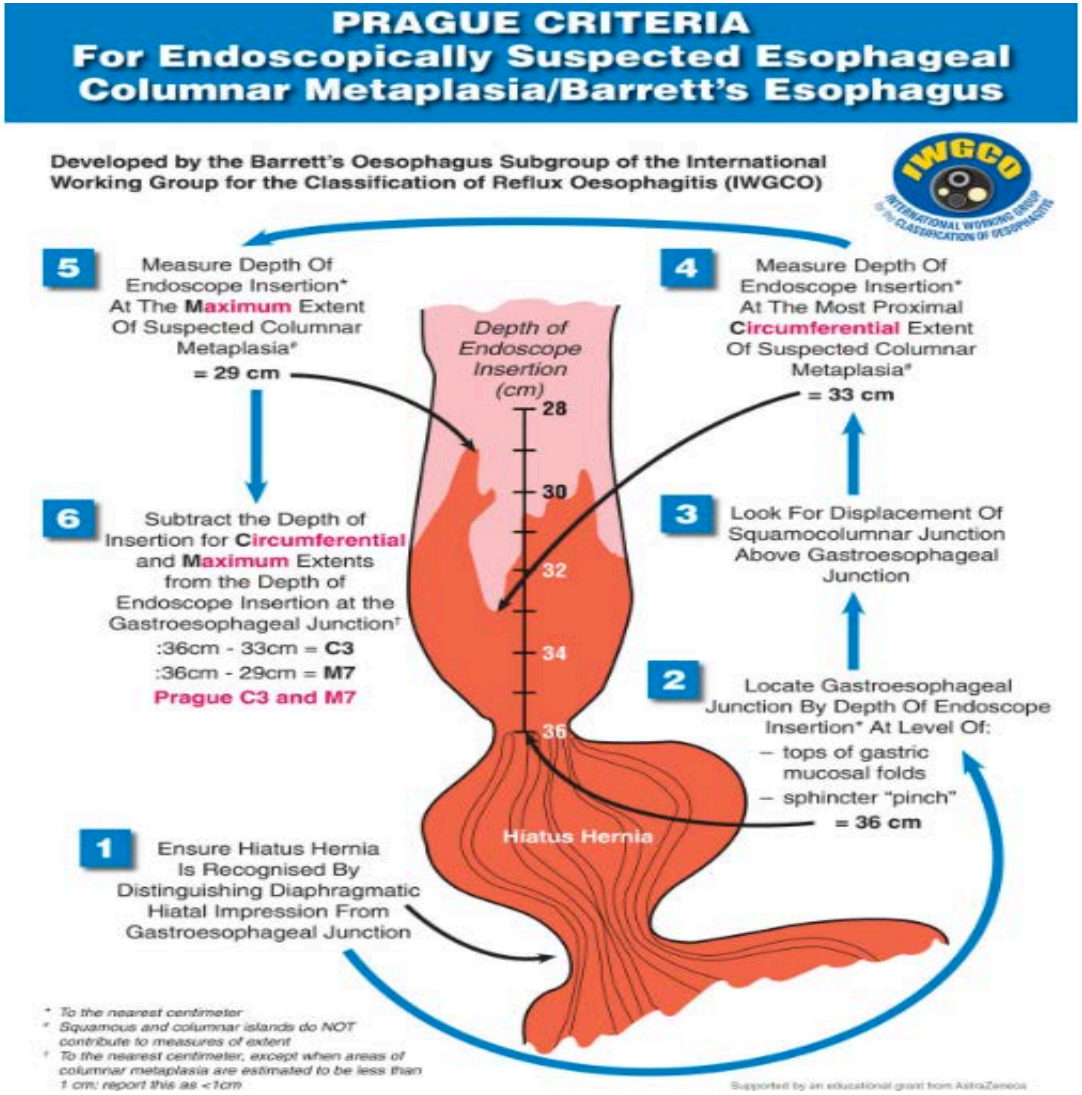
[BARRETT'S METAPLASIA]. A low-grade dysplasia in Barrett's esophagus, at a minimum, requires nuclear enlargement (right arrow), nuclear hyperchromasia, and nuclear contour irregularity. Prominent nucleoli and mitoses are common but reactive epithelium may also show mitoses and prominent nucleoli. Generally, 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

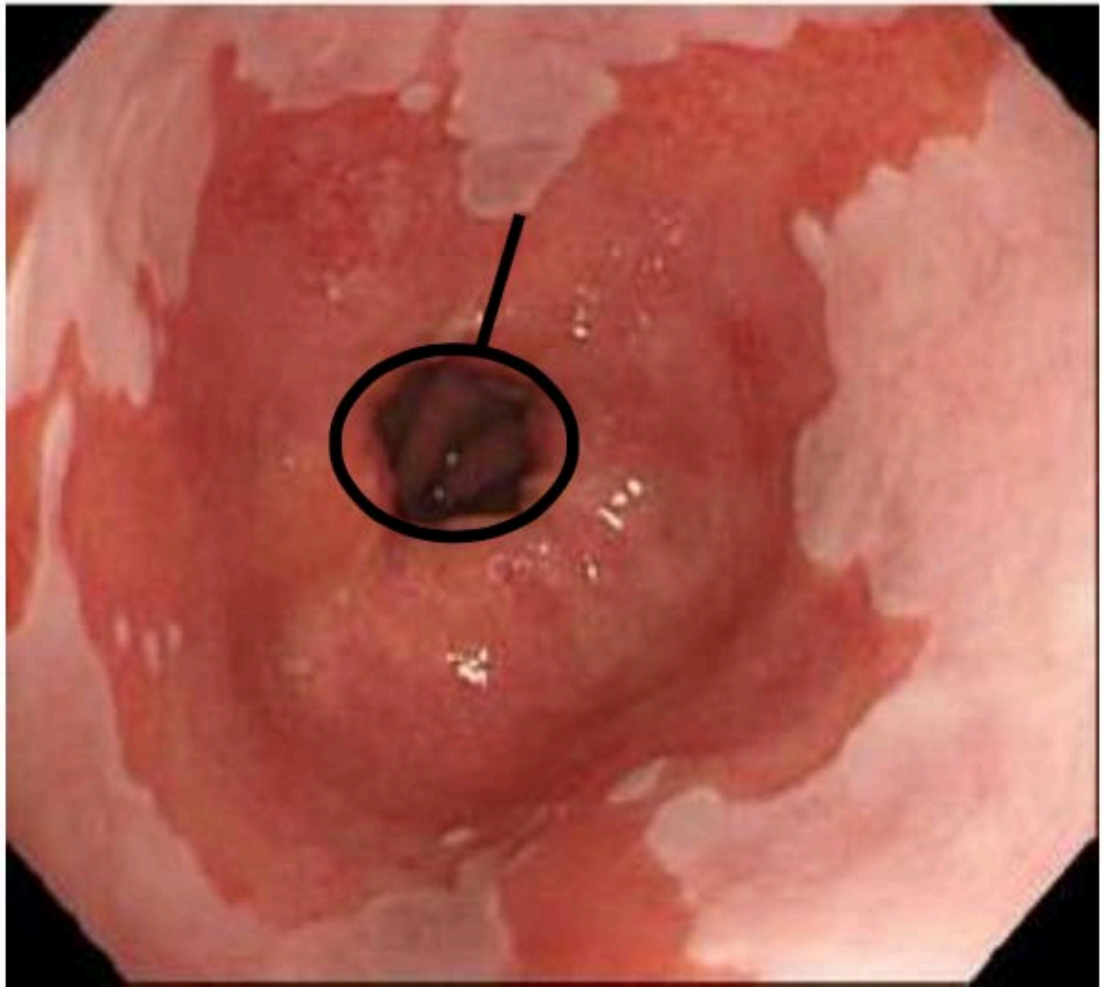


[BARRETT'S METAPLASIA]. High-grade dysplasia in Barrett's is difficult to miss because the changes stand out at low magnifications. The diagnosis of high-grade dysplasia should be made when surface epithelium shows all features of high-grade dysplasia (top arrow) such as enlarged hyperchromatic nuclei with little cytoplasm left in cells. This requirement of surface epithelium involvement help ensure that neither low grade dysplasia not 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 CLASSIFICATION - C



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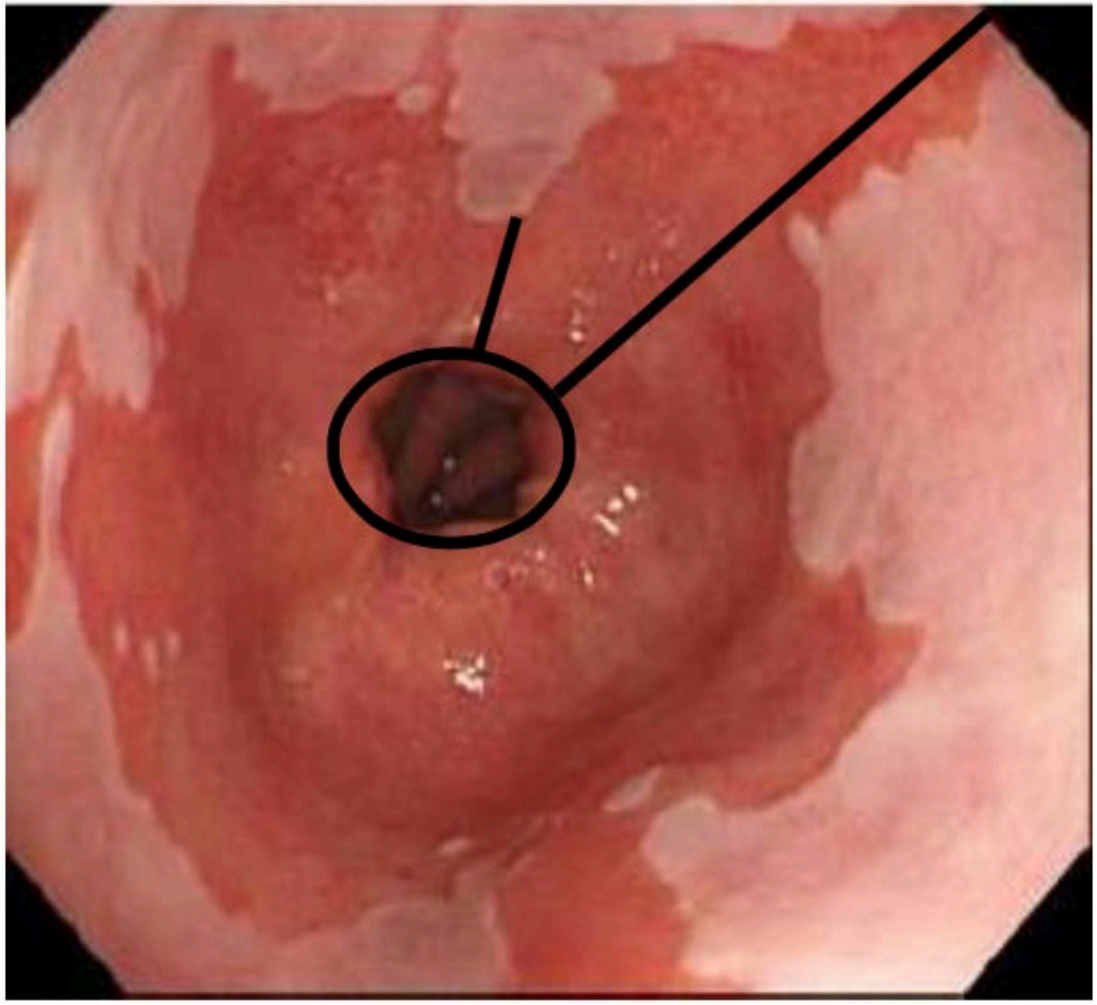


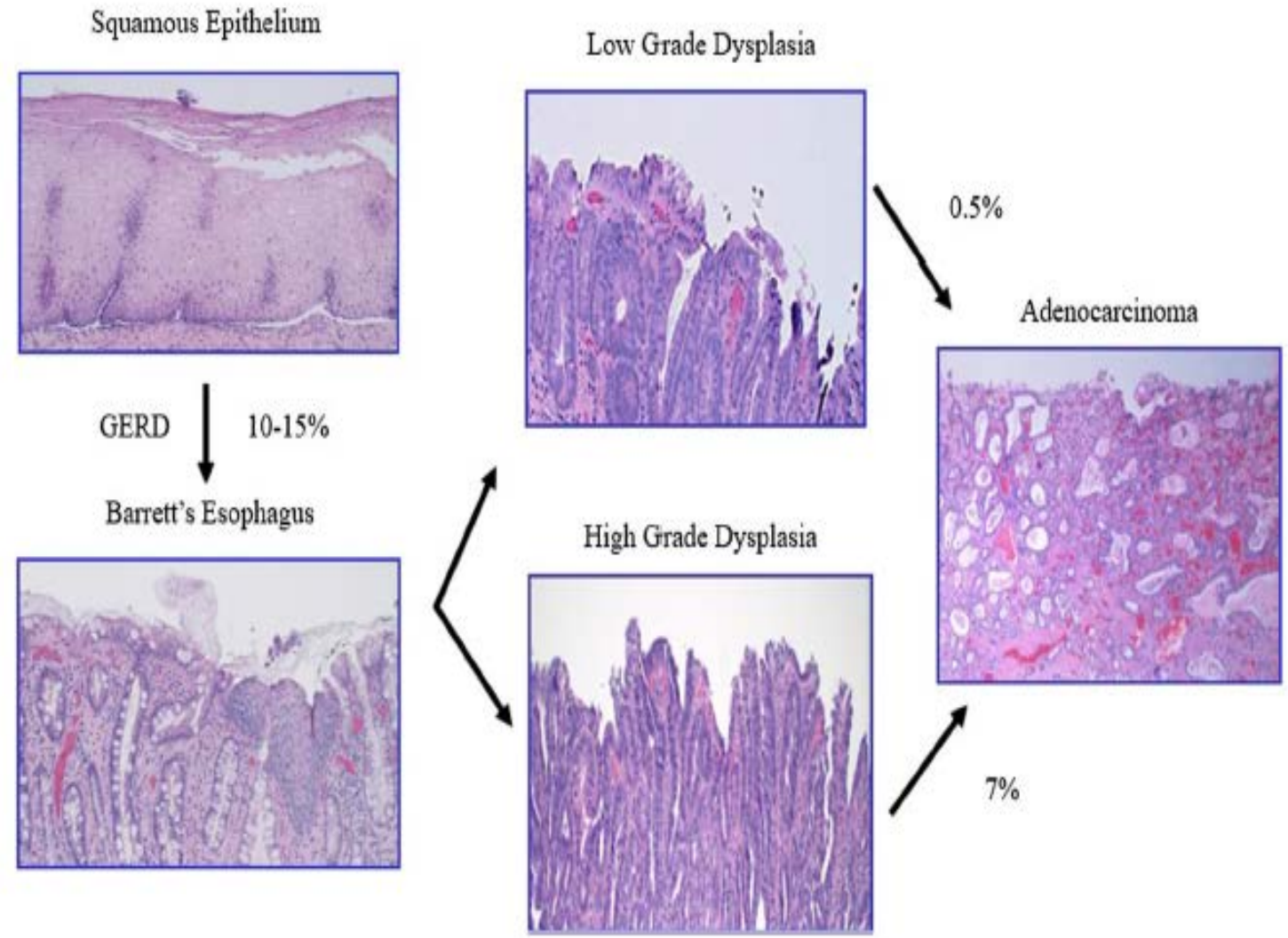


Fig. 1 Malignant transformation of Barrett's esophagus

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*

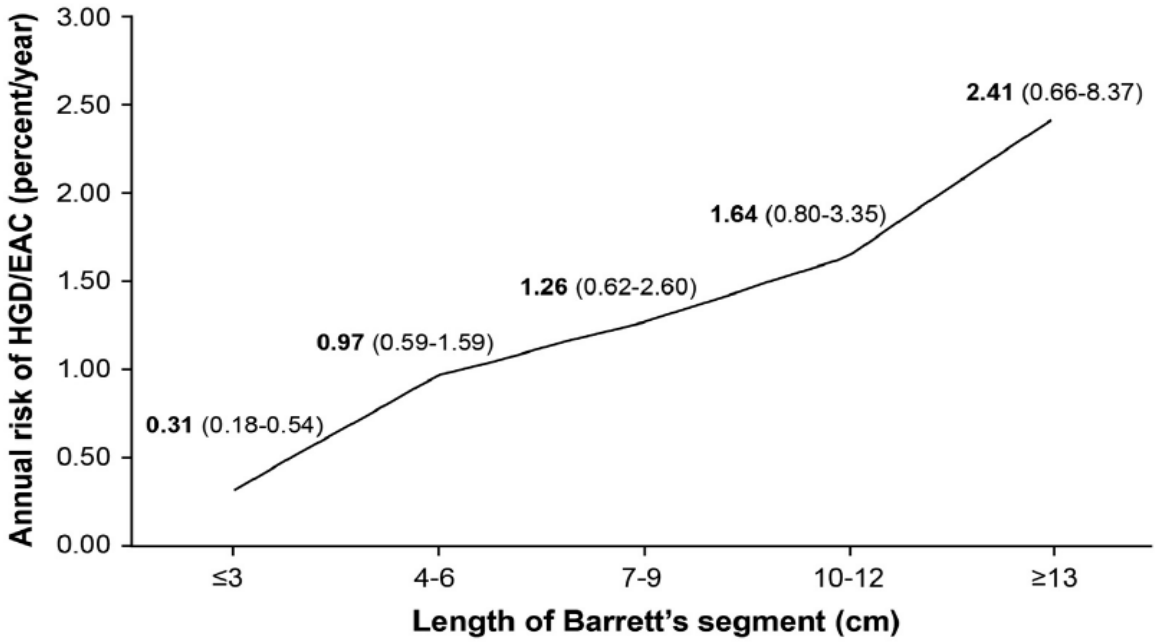


Figure 2. Annual risk of HGD/EAC (%/year) in NDBE patients. Incident rates of HGD/EAC with 95% CI (in parentheses)

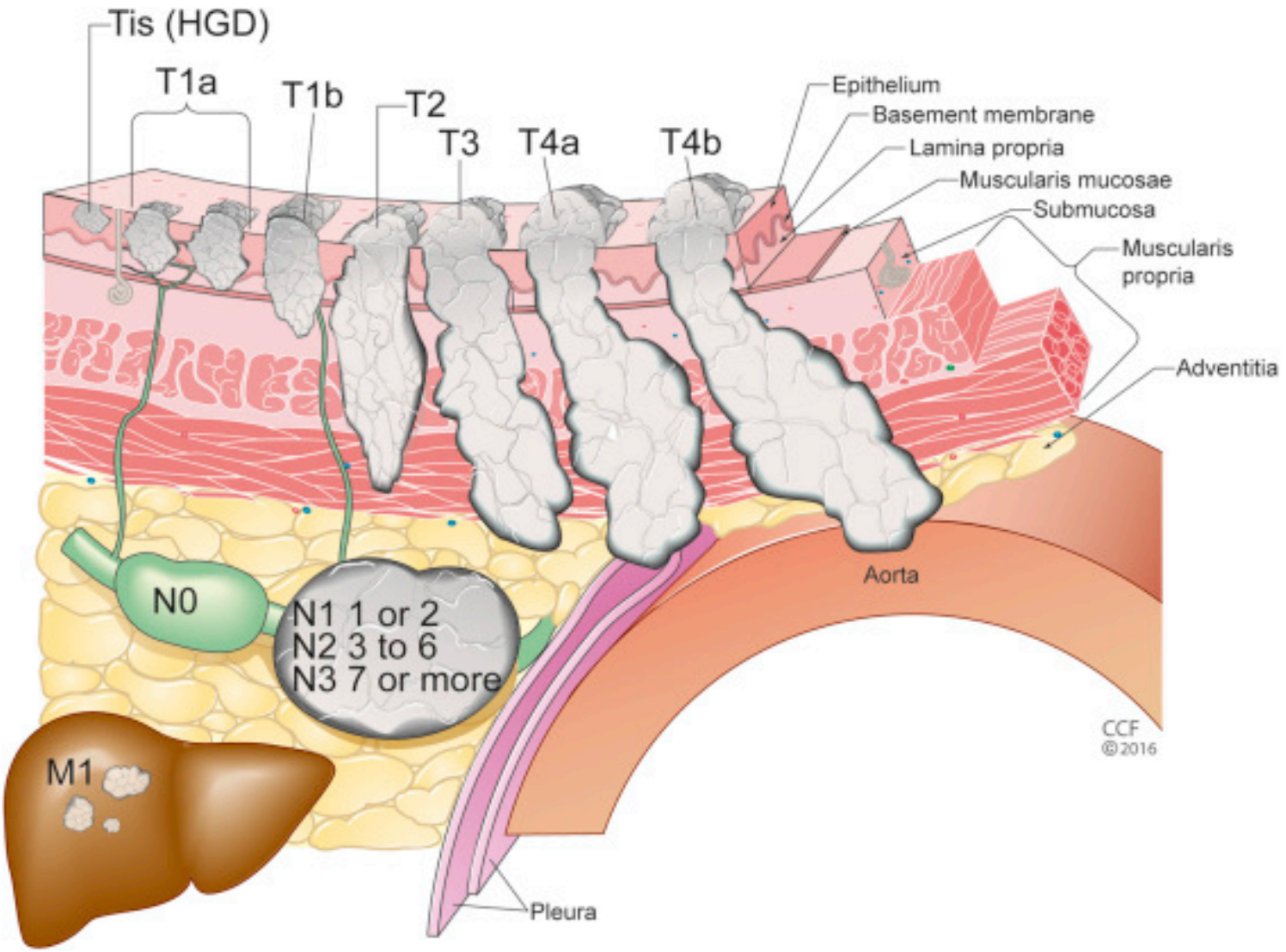
ΚΛΙΝΙΚΗ ΕΙΚΟΝΑ ΚΑΡΚΙΝΟΥ ΟΙΣΟΦΑΓΟΥ

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

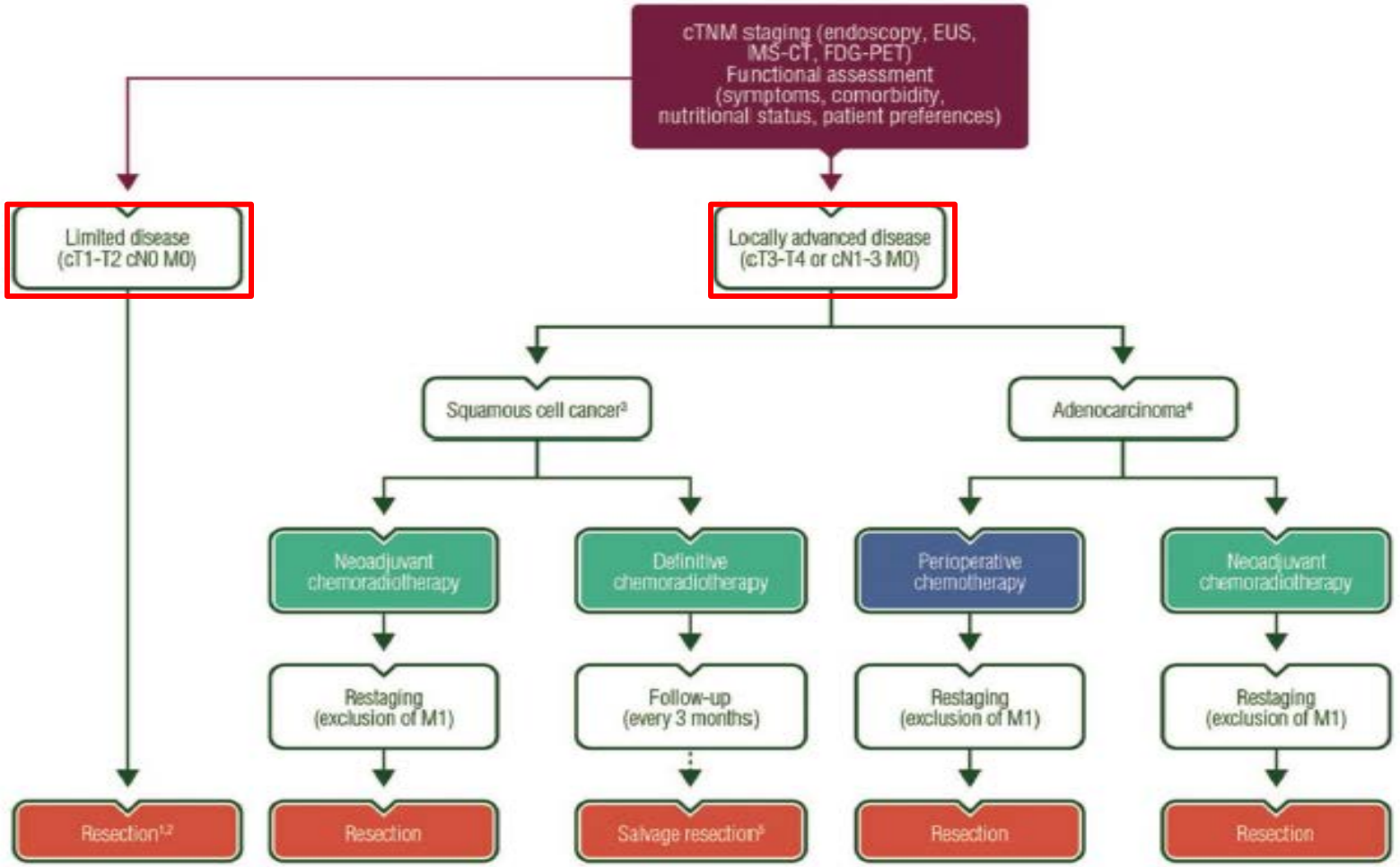
ΚΛΙΝΙΚΗ ΣΤΑΔΙΟΠΟΙΗΣΗ ΚΑΡΚΙΝΟΥ ΟΙΣΟΦΑΓΟΥ

- **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

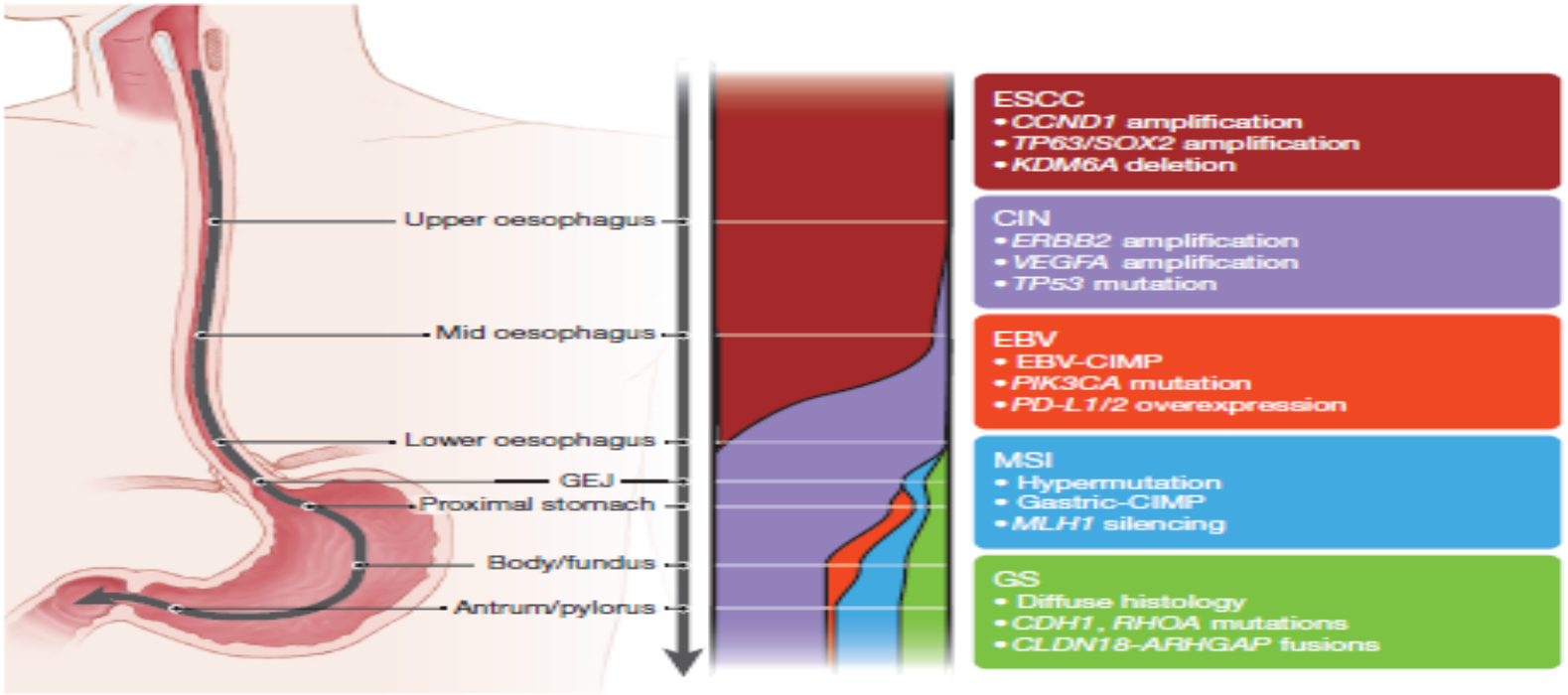
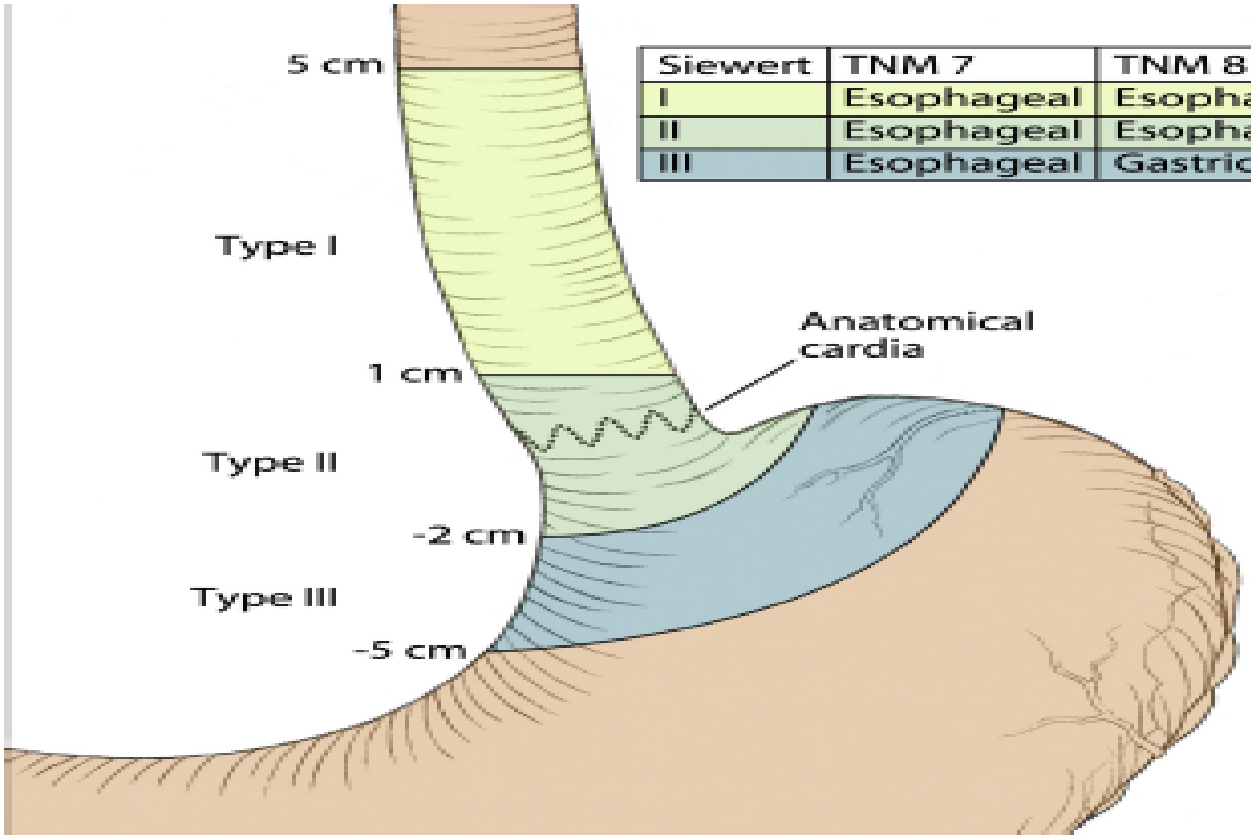


Figure 6 | Gradations of molecular subclasses of gastroesophageal carcinoma. Schematic representing shifting proportion of subtypes of gastroesophageal carcinoma from the proximal oesophagus to the distal stomach. The widths of the colour bands represent the proportion of the subtypes present within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

ΤΑΞΙΝΟΜΗΣΗ ΚΑΤΑ SIEWERT



Siewert	TNM 7	TNM 8
I	Esophageal	Esophageal
II	Esophageal	Esophageal
III	Esophageal	Gastric

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

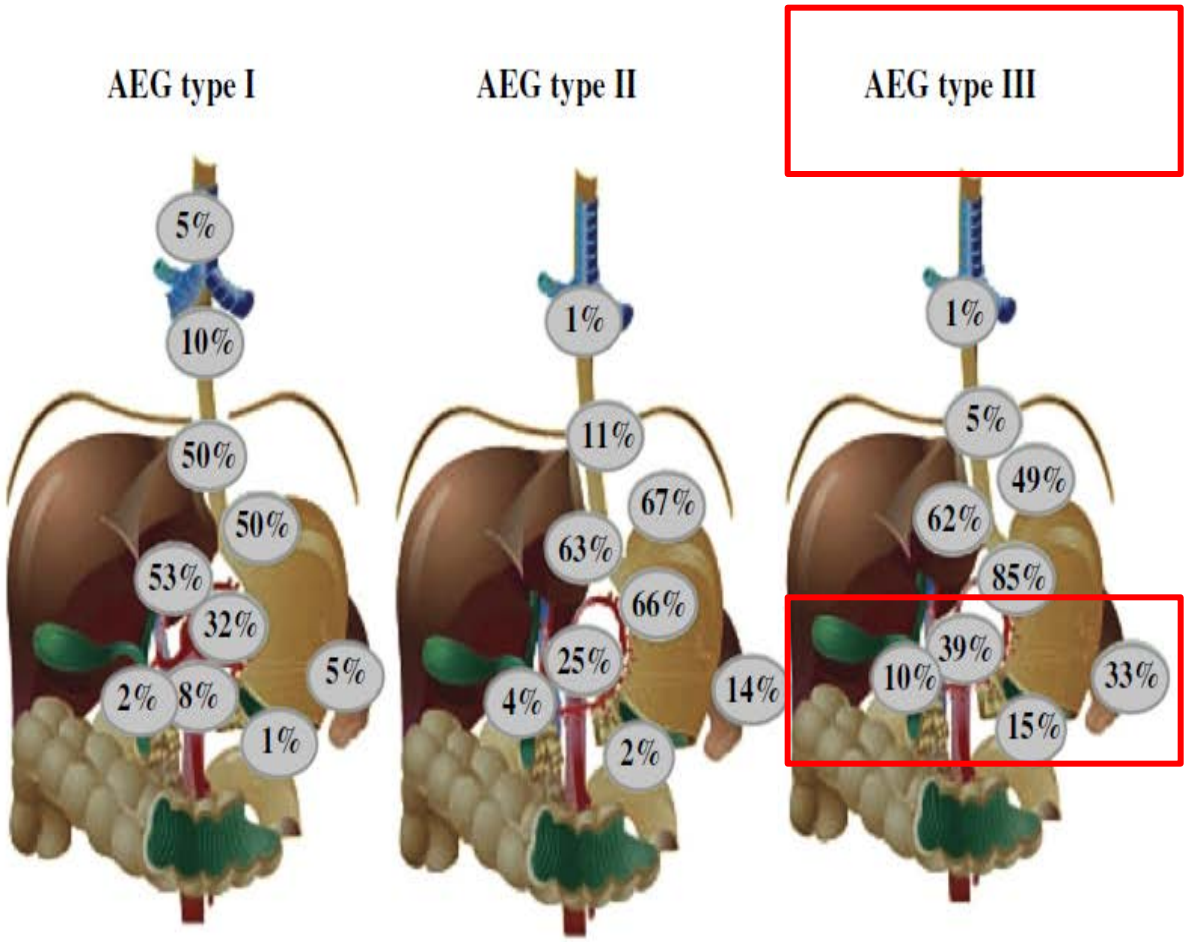


FIG. 2 Distribution of lymph node metastases in patients with resected adenocarcinoma of the esophagogastric junction (AEG) types I, II, and III. [*Adapted from*: Siewert JR, Stein HJ, Feith M. Adenocarcinoma of the esophago-gastric junction. *Scand J Surg*. 2006;95:260–69.]

SIEWERT III

Lancet Oncol 2011; 12: 296–305

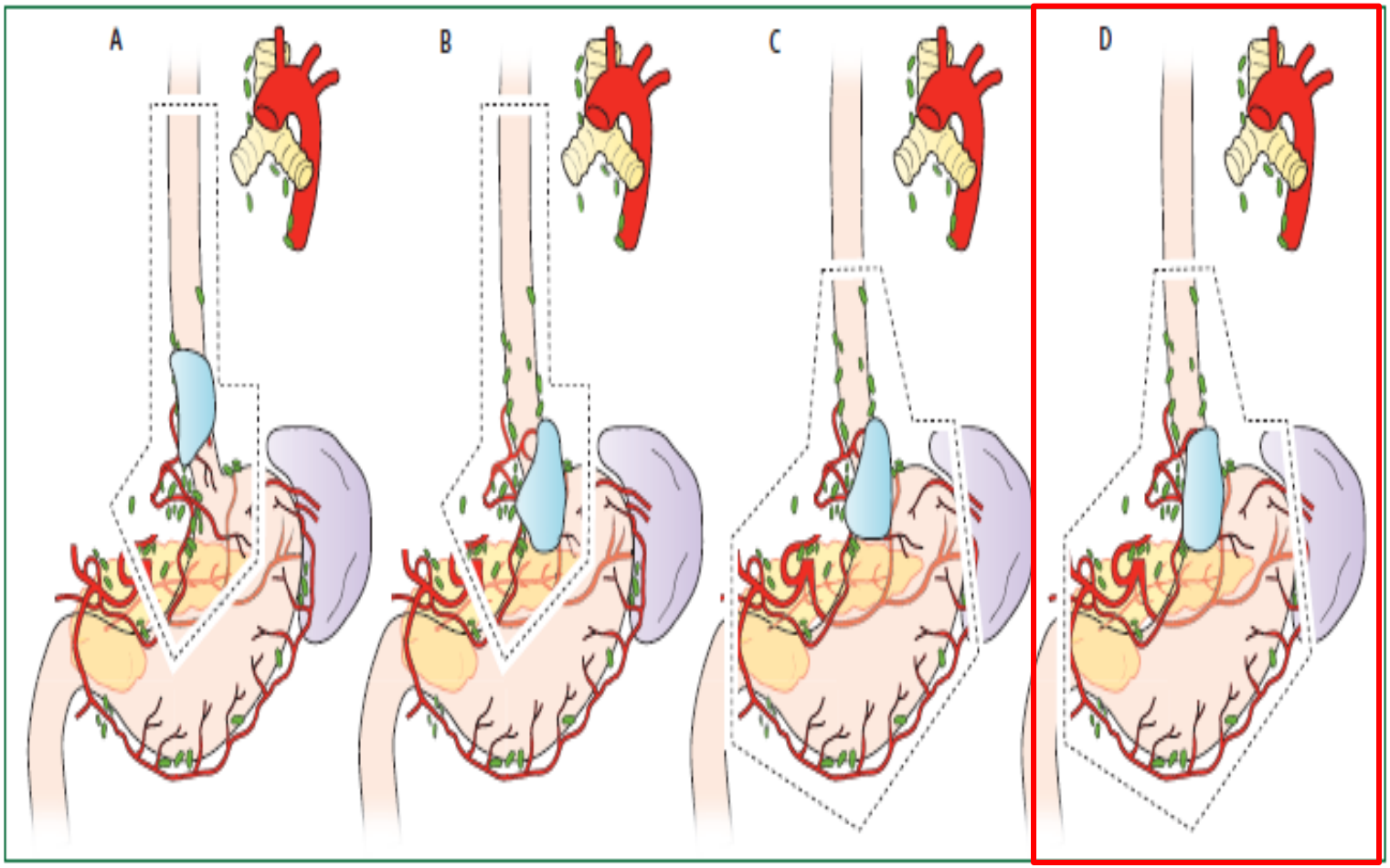
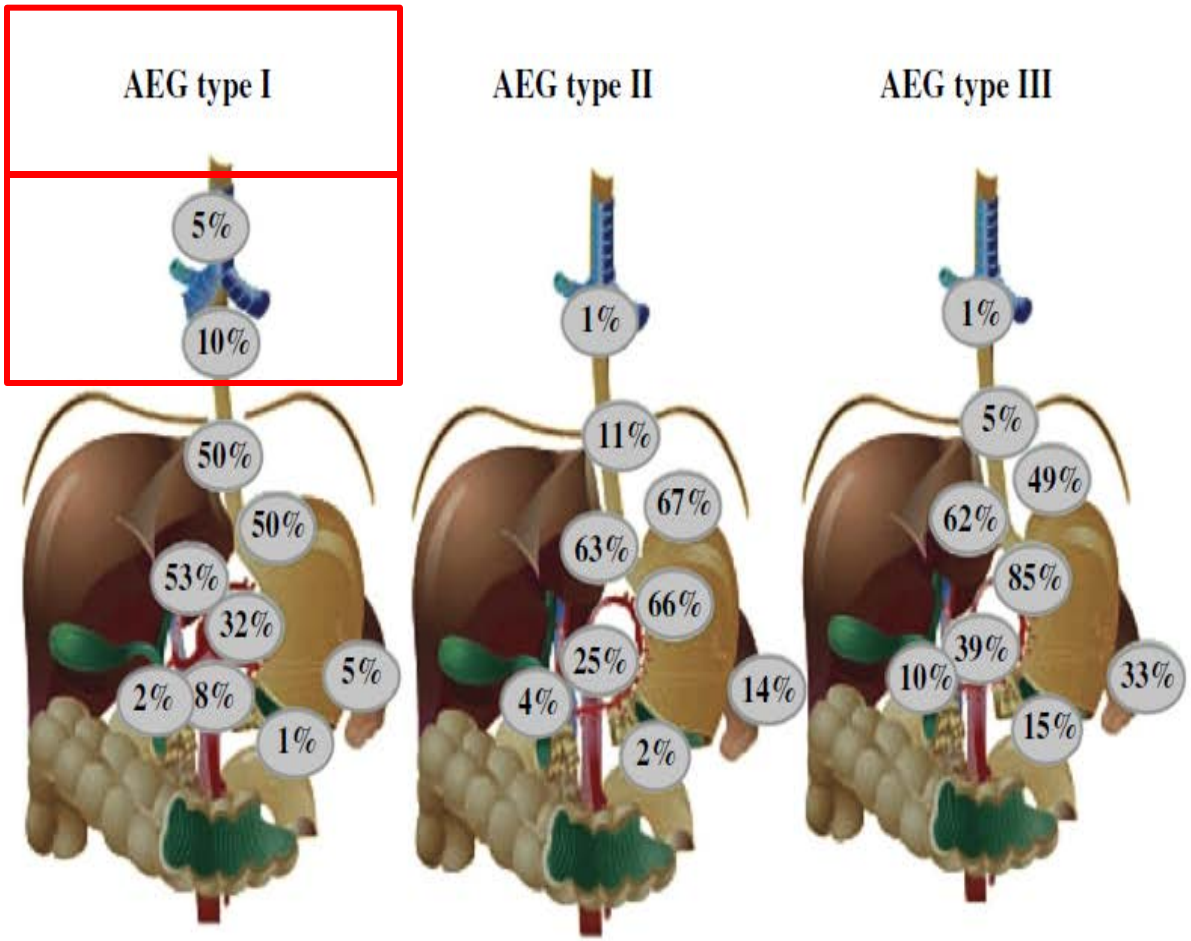


Figure 4: Schematic representation of recommended extent of surgical resection for oesophagogastric junction adenocarcinomas
Type I (A; subtotal oesophagectomy with superior polar gastrectomy), type II (subtotal oesophagectomy with superior polar gastrectomy [B] or total gastrectomy with inferior oesophagectomy [C]), and type III (D; total gastrectomy). Blue region is tumour site.

ΛΕΜΦΑΔΕΝΙΚΗ ΔΙΑΣΠΟΡΑ



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SIEWERT I

Lancet Oncol 2011; 12: 296–305

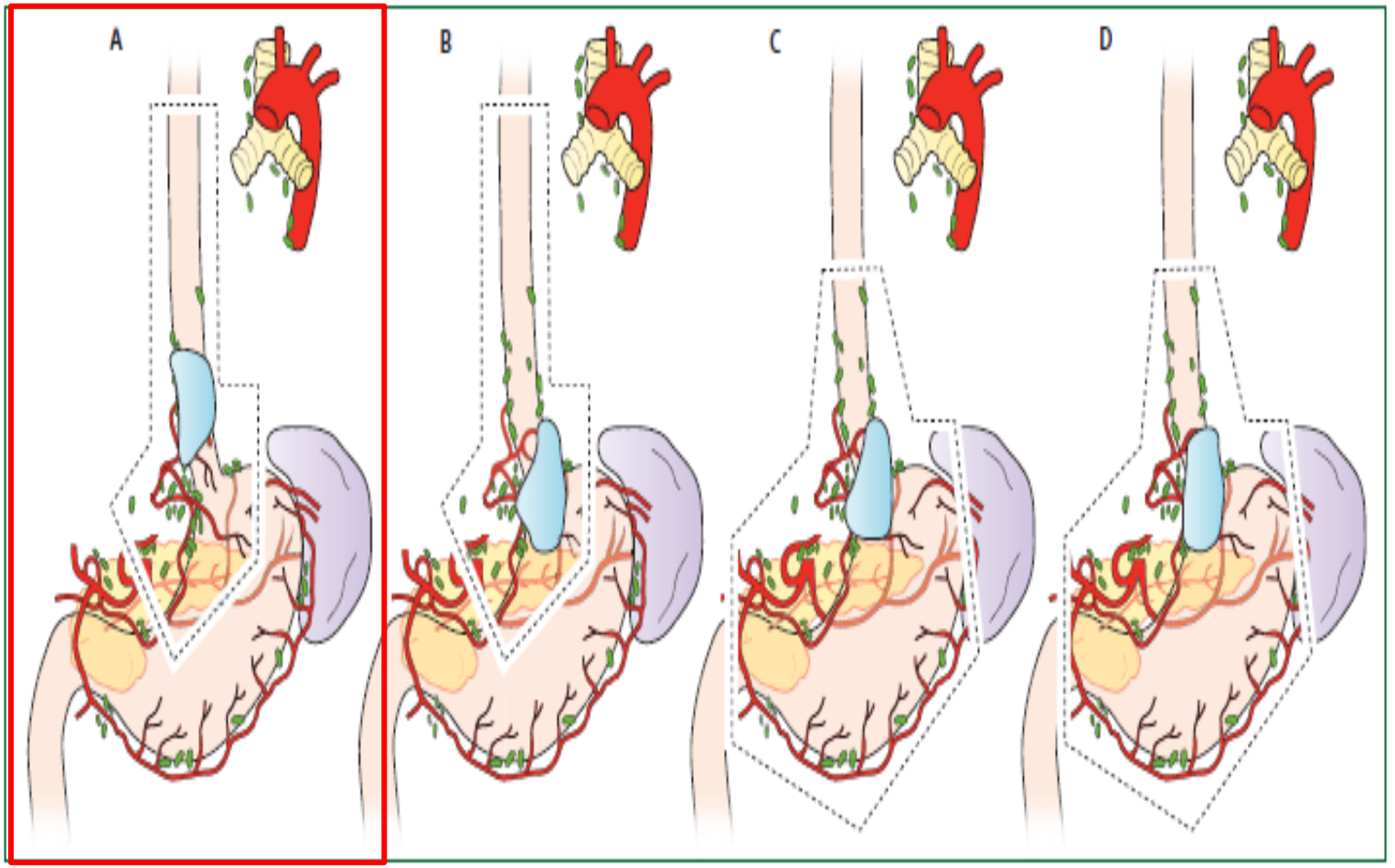
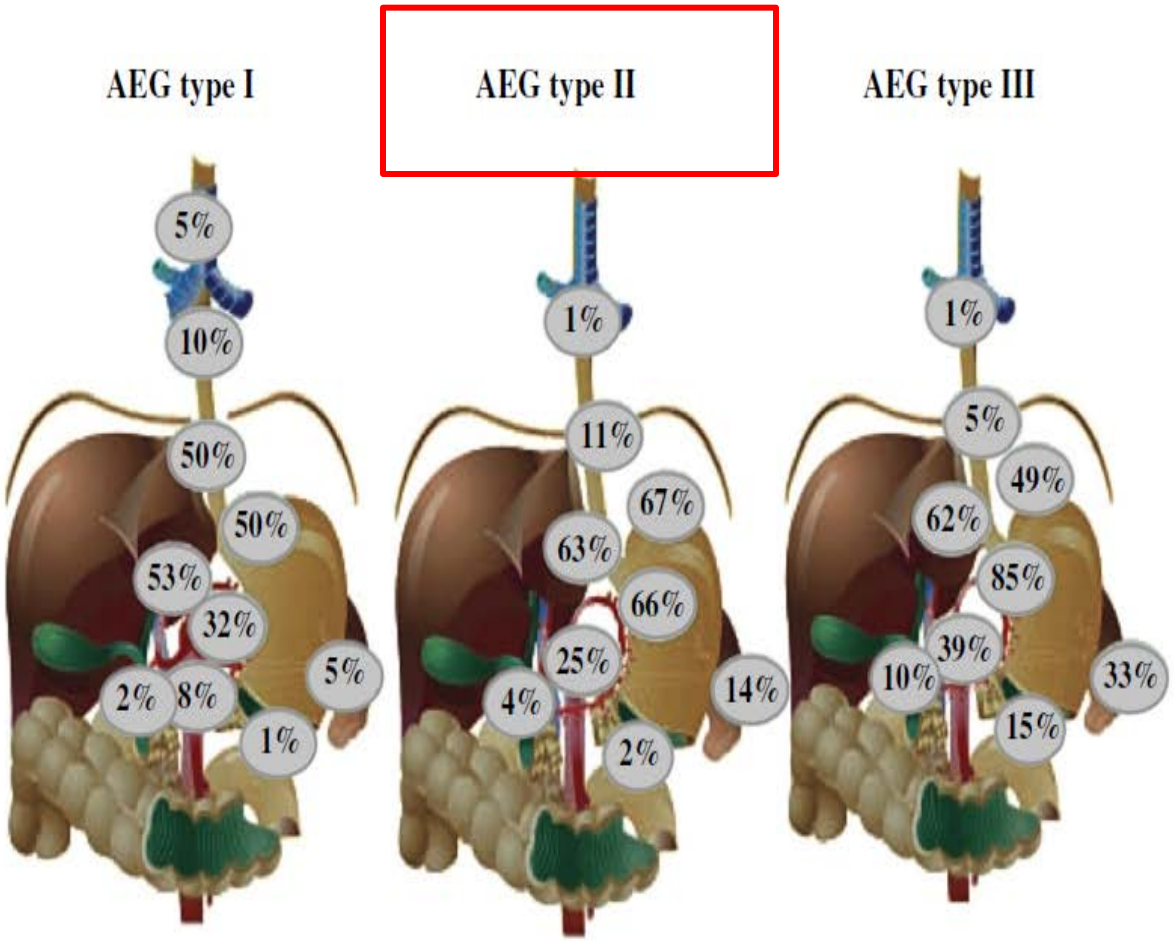


Figure 4: Schematic representation of recommended extent of surgical resection for oesophagogastric junction adenocarcinomas
Type I (A; subtotal oesophagectomy with superior polar gastrectomy), type II (subtotal oesophagectomy with superior polar gastrectomy [B] or total gastrectomy with inferior oesophagectomy [C]), and type III (D; total gastrectomy). Blue region is tumour site.

ΛΕΜΦΑΔΕΝΙΚΗ ΔΙΑΣΠΟΡΑ



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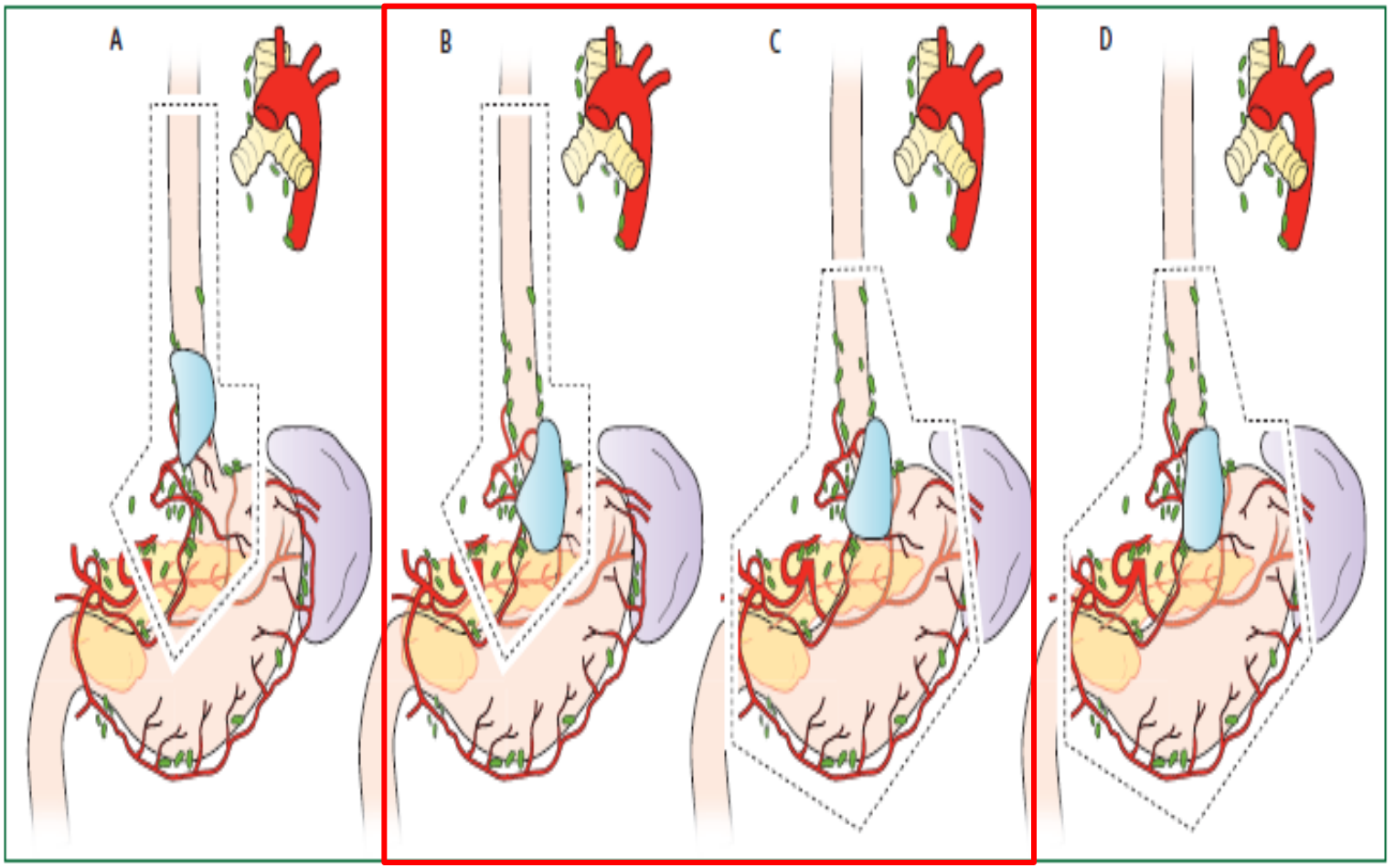
SIEWERT II

Lancet Oncol 2011; 12: 296–305



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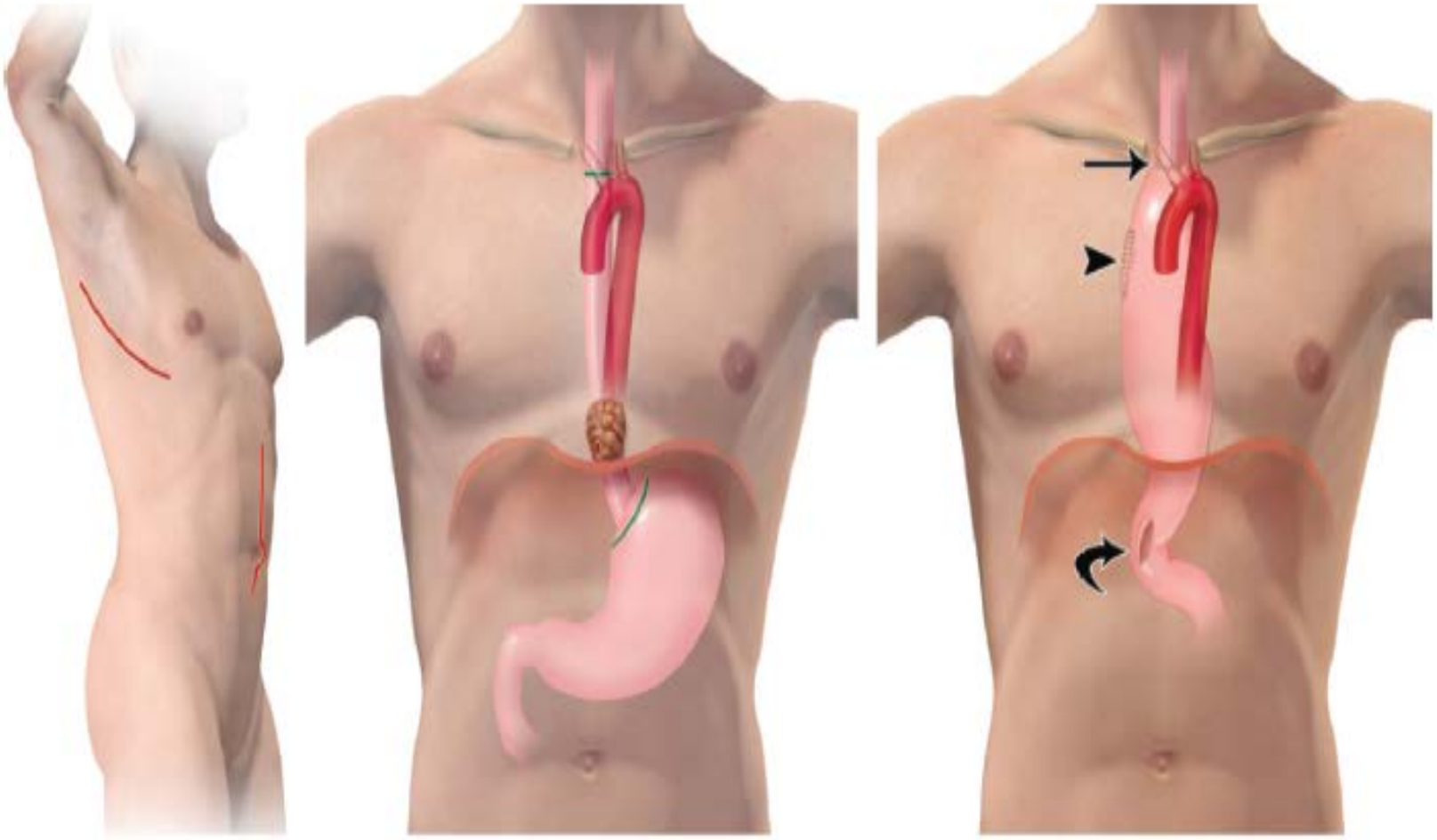
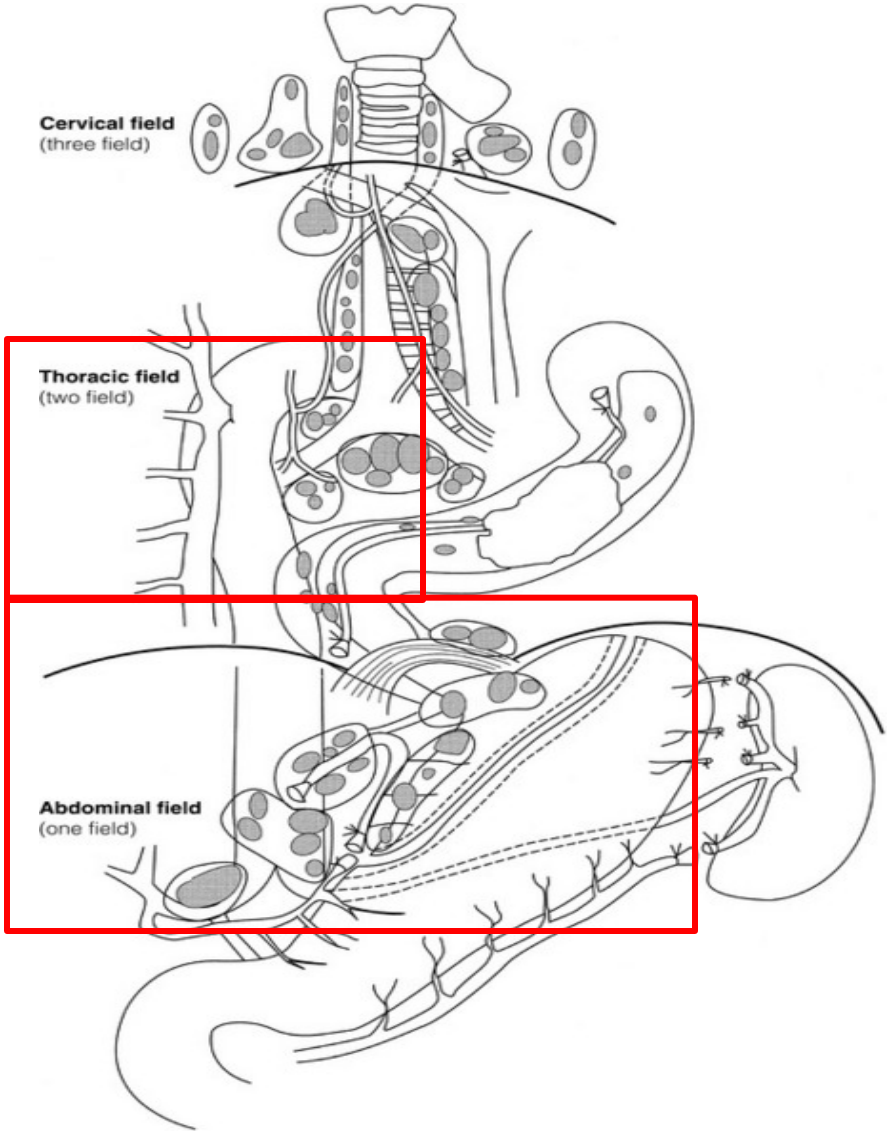
ΤΥΠΟΙ ΟΙΣΟΦΑΓΕΚΤΟΜΩΝ

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

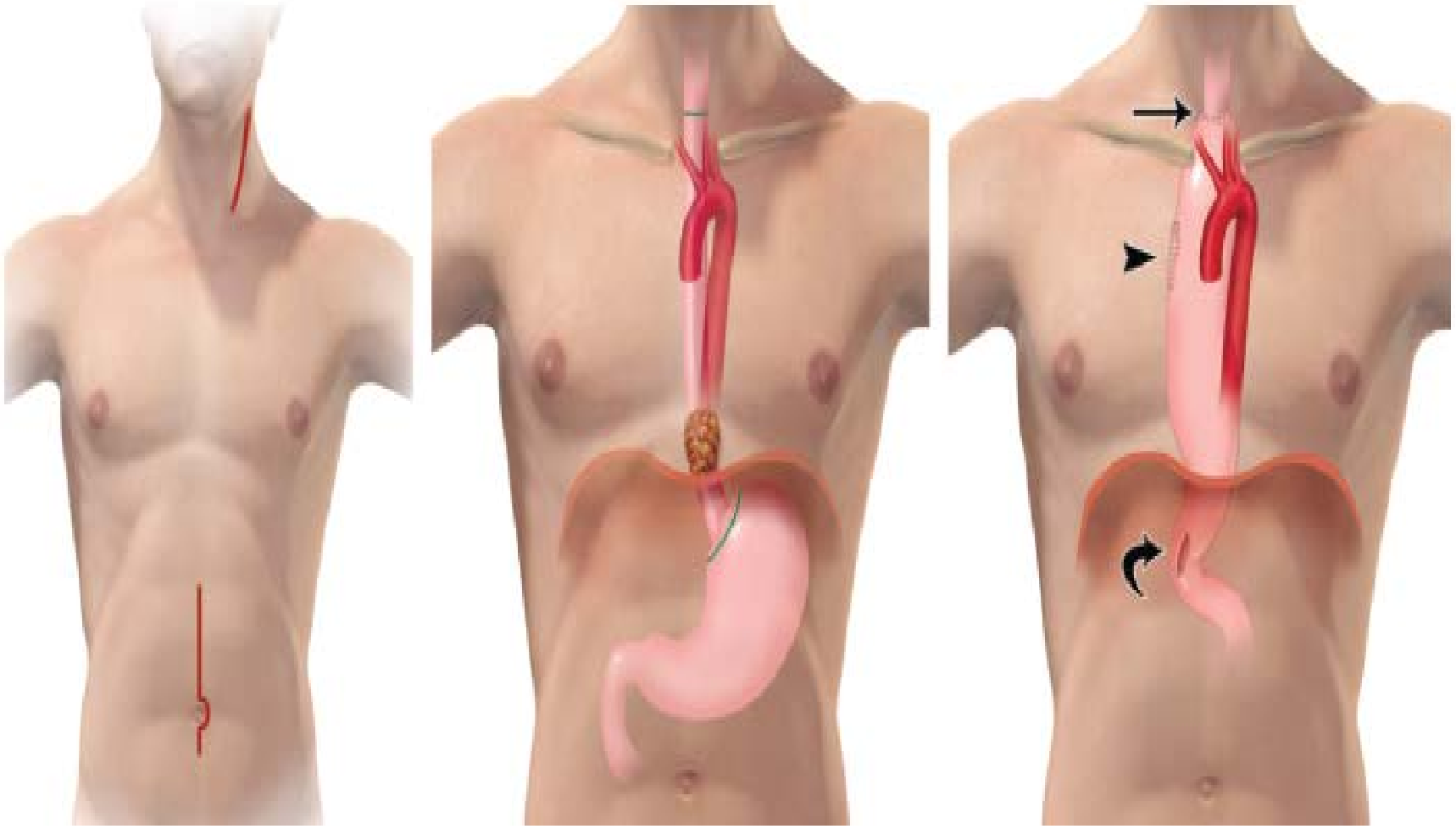
IVOR-LEWIS ΟΙΣΟΦΑΓΕΚΤΟΜΗ



ΛΕΜΦΑΔΕΝΙΚΟΣ ΚΑΘΑΡΙΣΜΟΣ - IVOR-LEWIS



TRANSHIATAL ΟΙΣΟΦΑΓΕΚΤΟΜΗ



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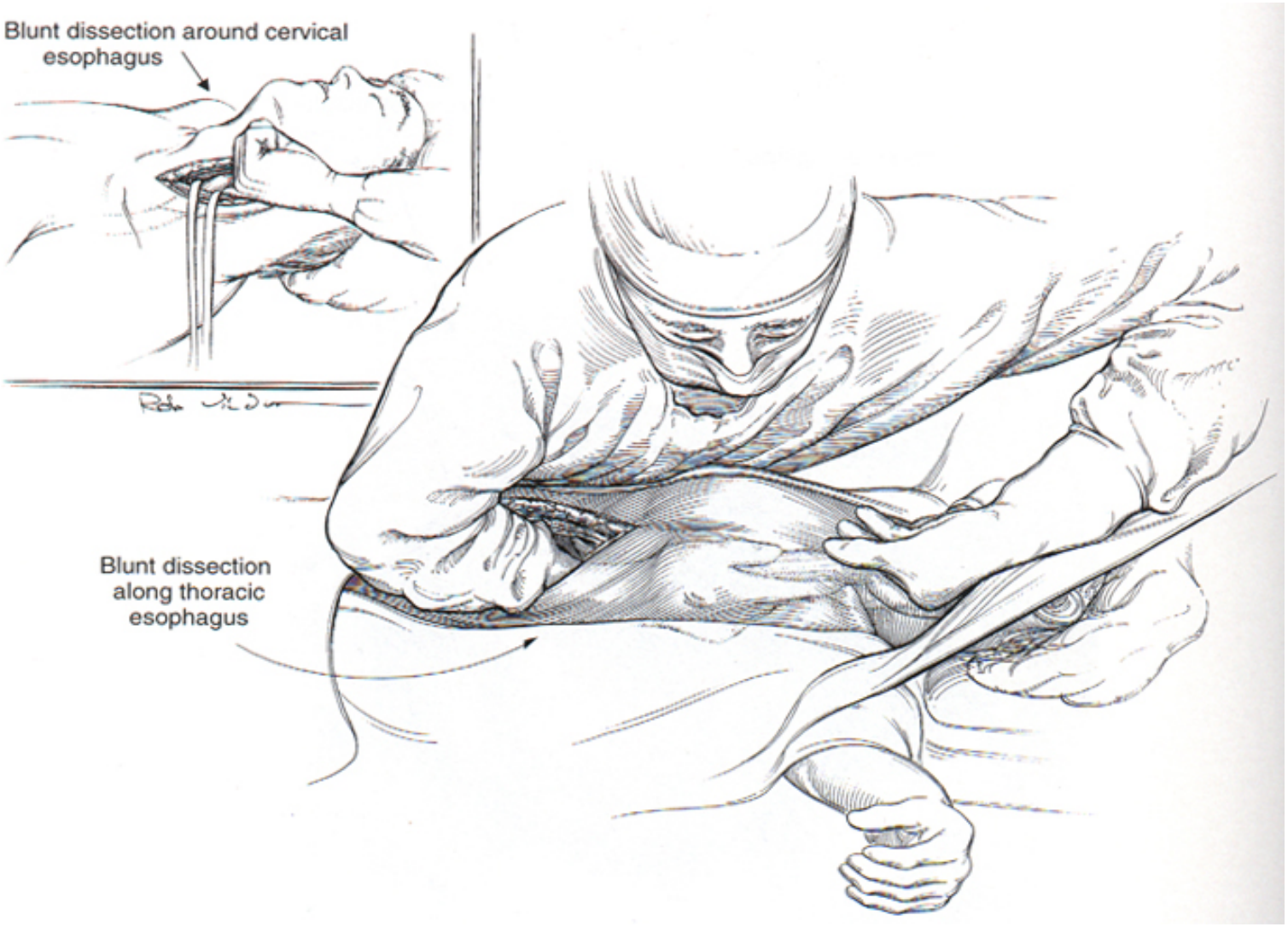

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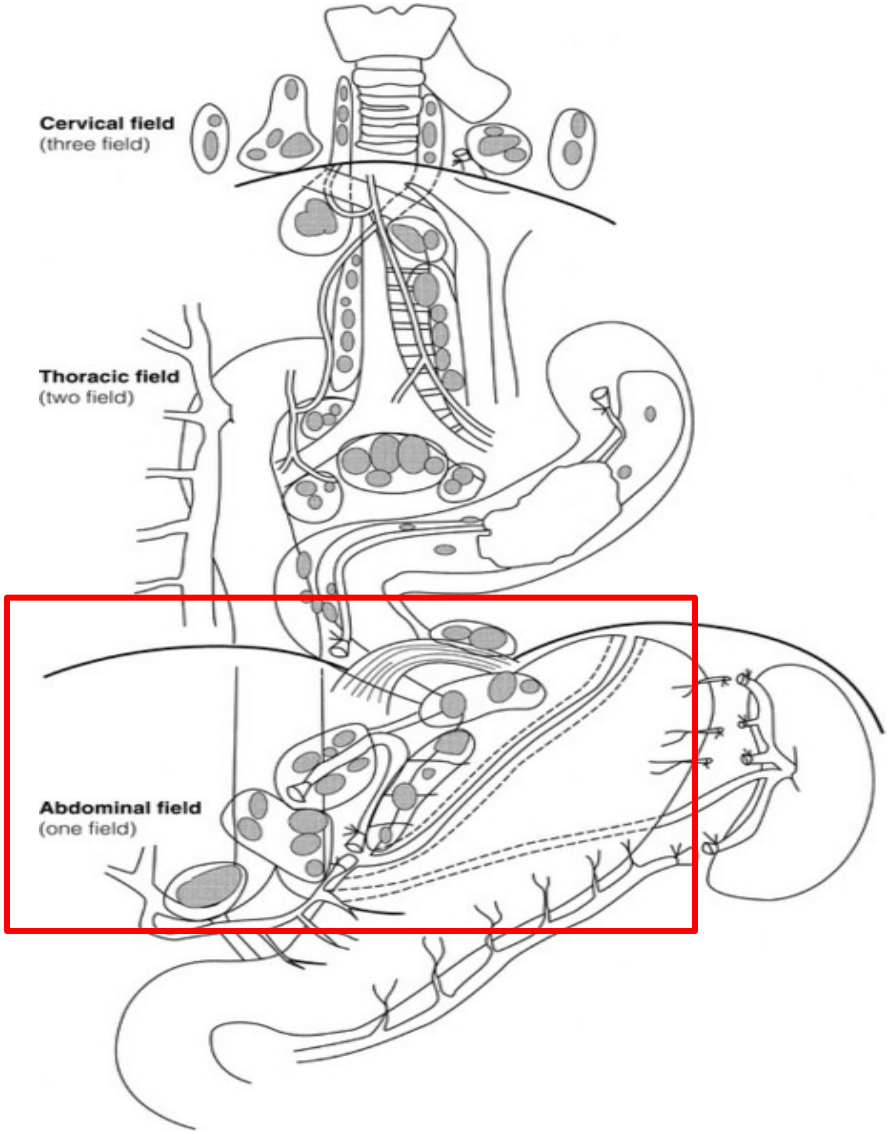
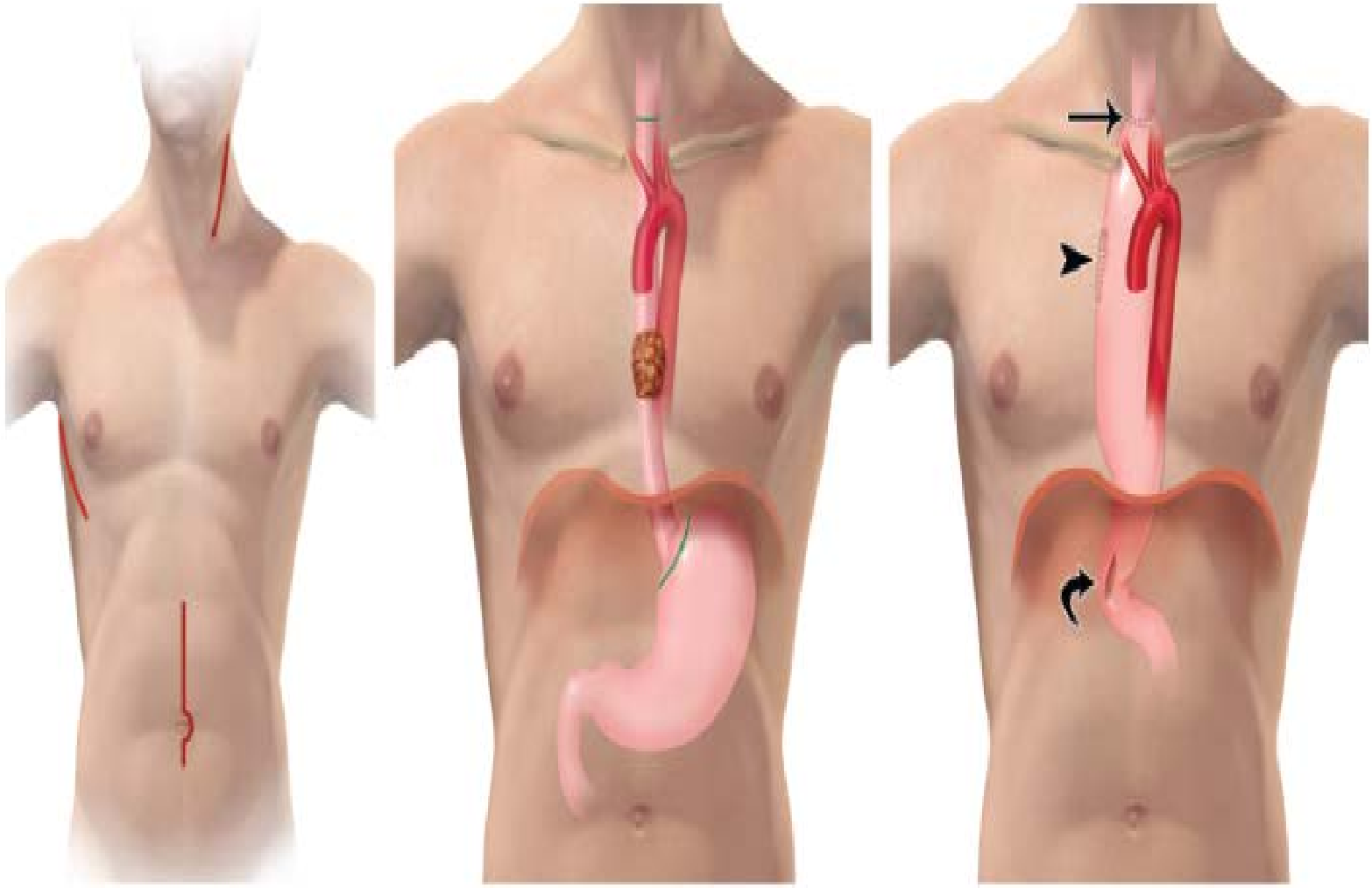


McKEOWN ΟΙΣΟΦΑΓΕΚΤΟΜΗ



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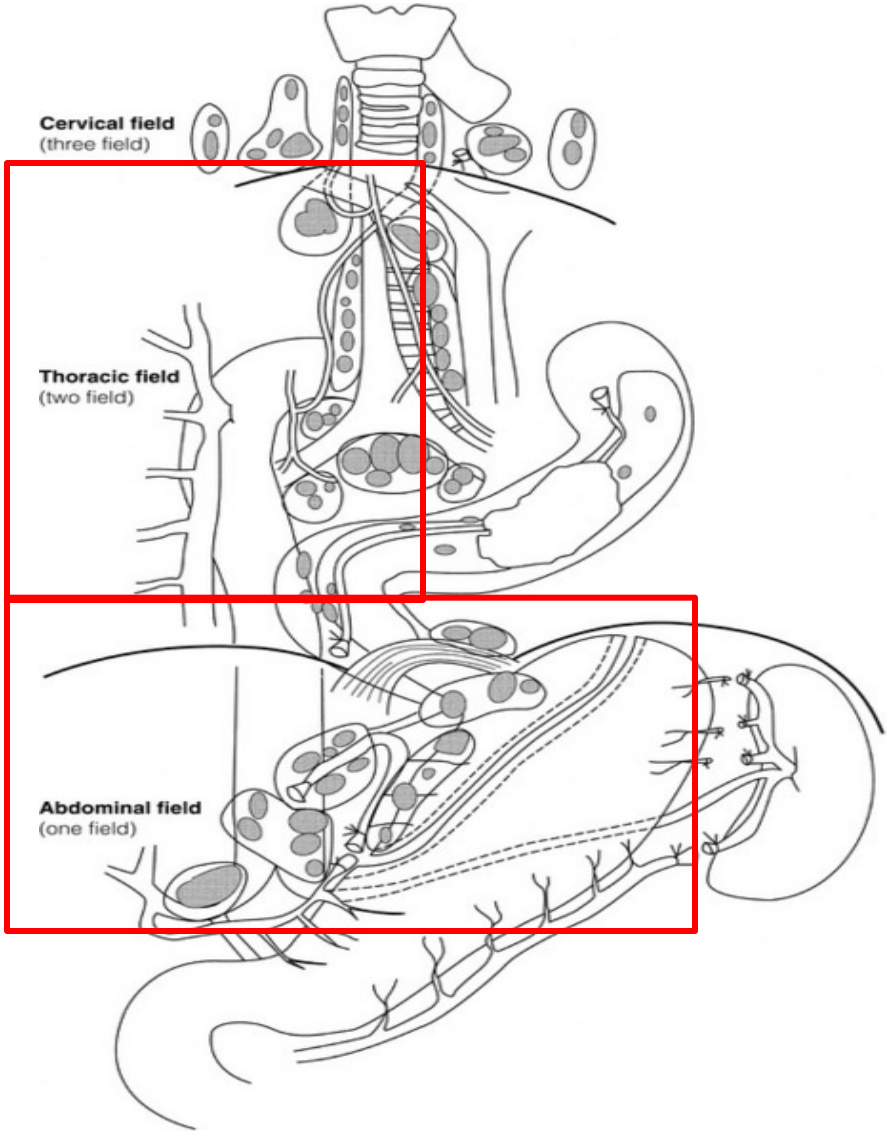
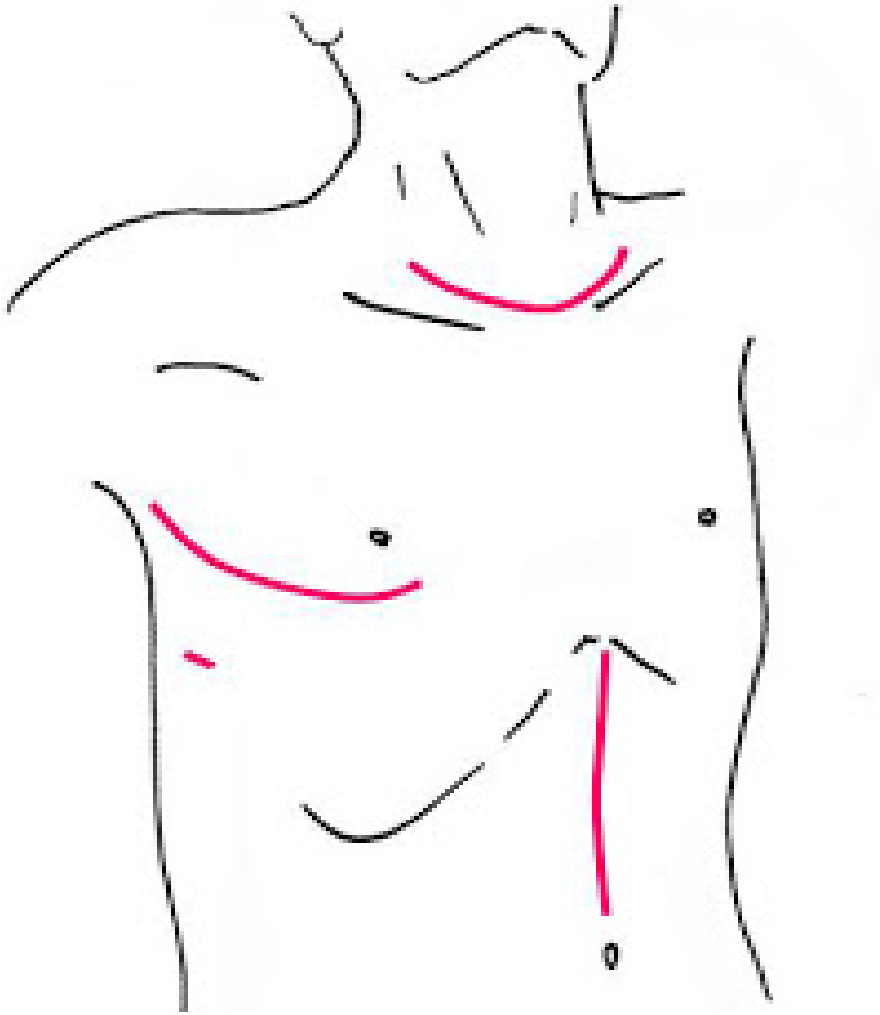
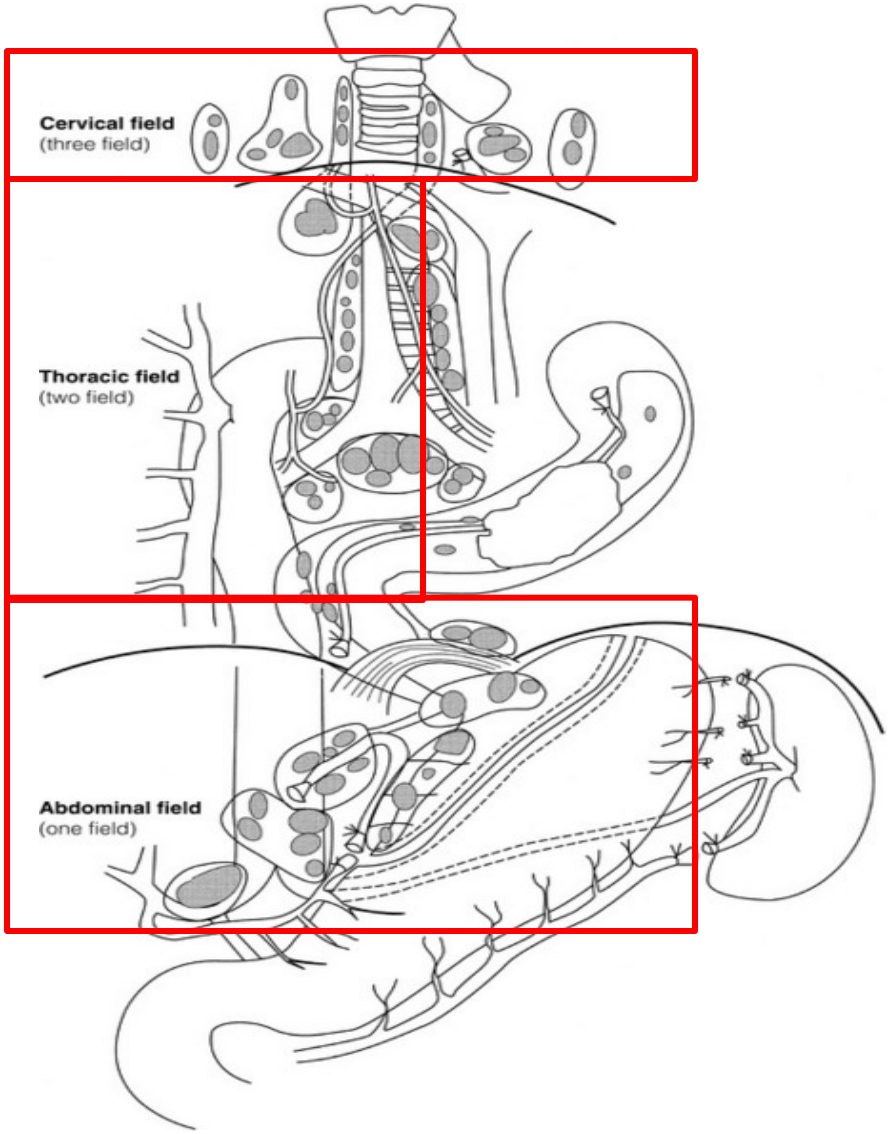


THREE FIELD ΟΙΣΟΦΑΓΕΚΤΟΜΗ



ΛΕΜΦΑΔΕΝΙΚΟΣ ΚΑΘΑΡΙΣΜΟΣ – THREE FIELD



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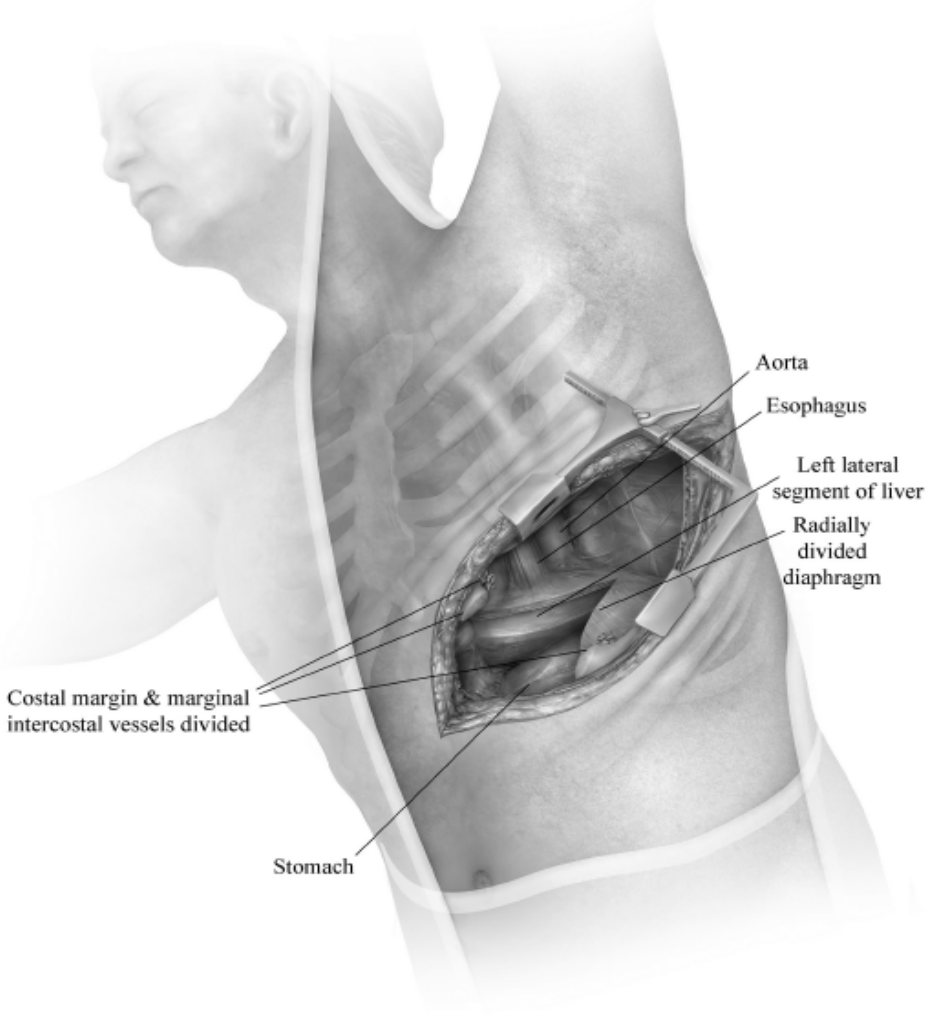
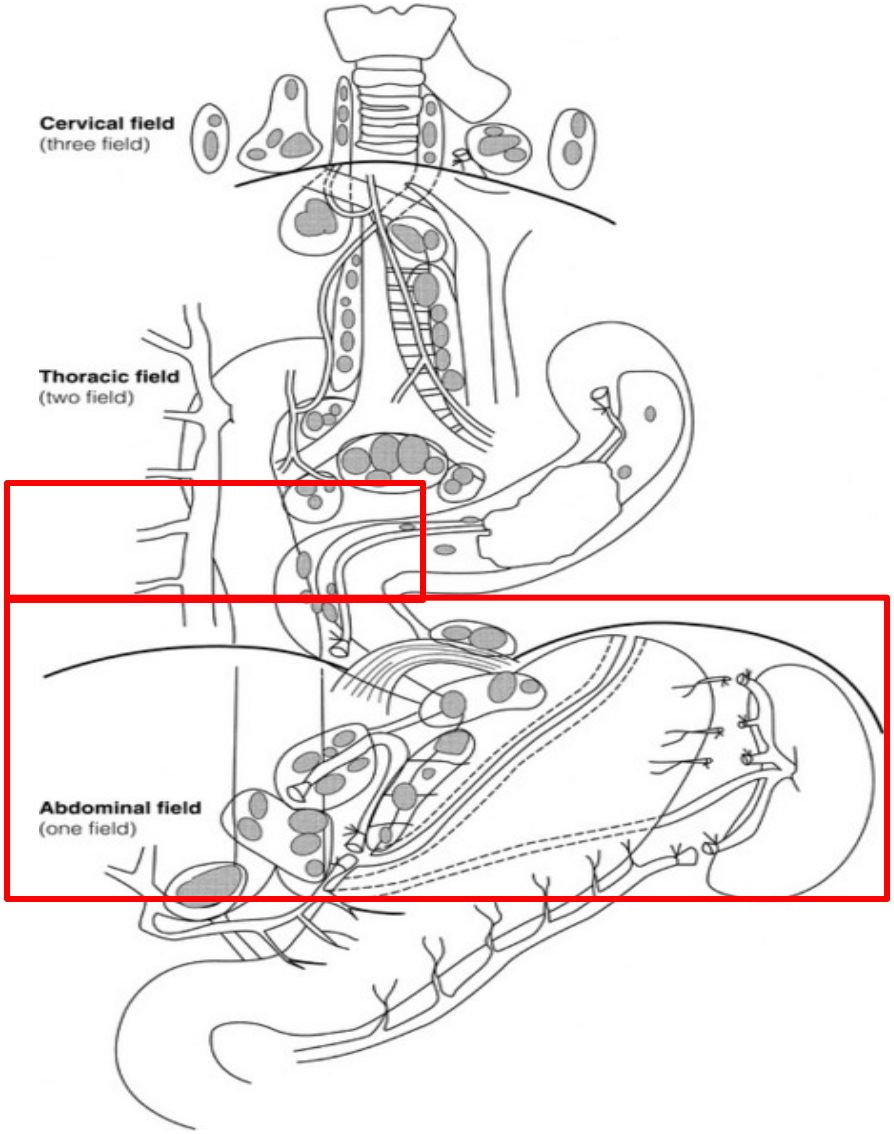
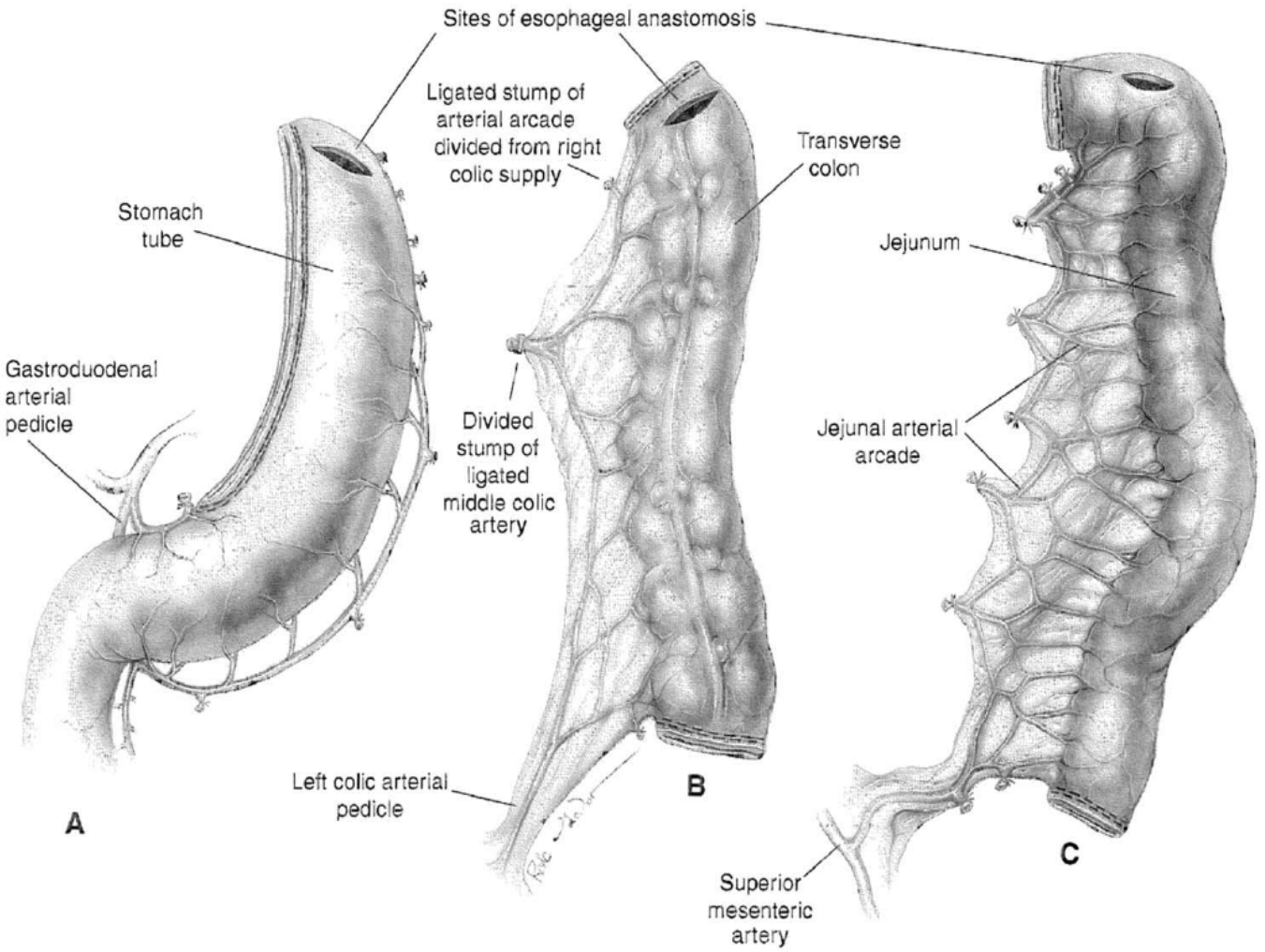


ΛΕΜΦΑΔΕΝΙΚΟΣ ΚΑΘΑΡΙΣΜΟΣ - ΑΡΙΣΤΕΡΗ ΘΩΡΑΚΟΚΟΙΛΙΑΚΗ

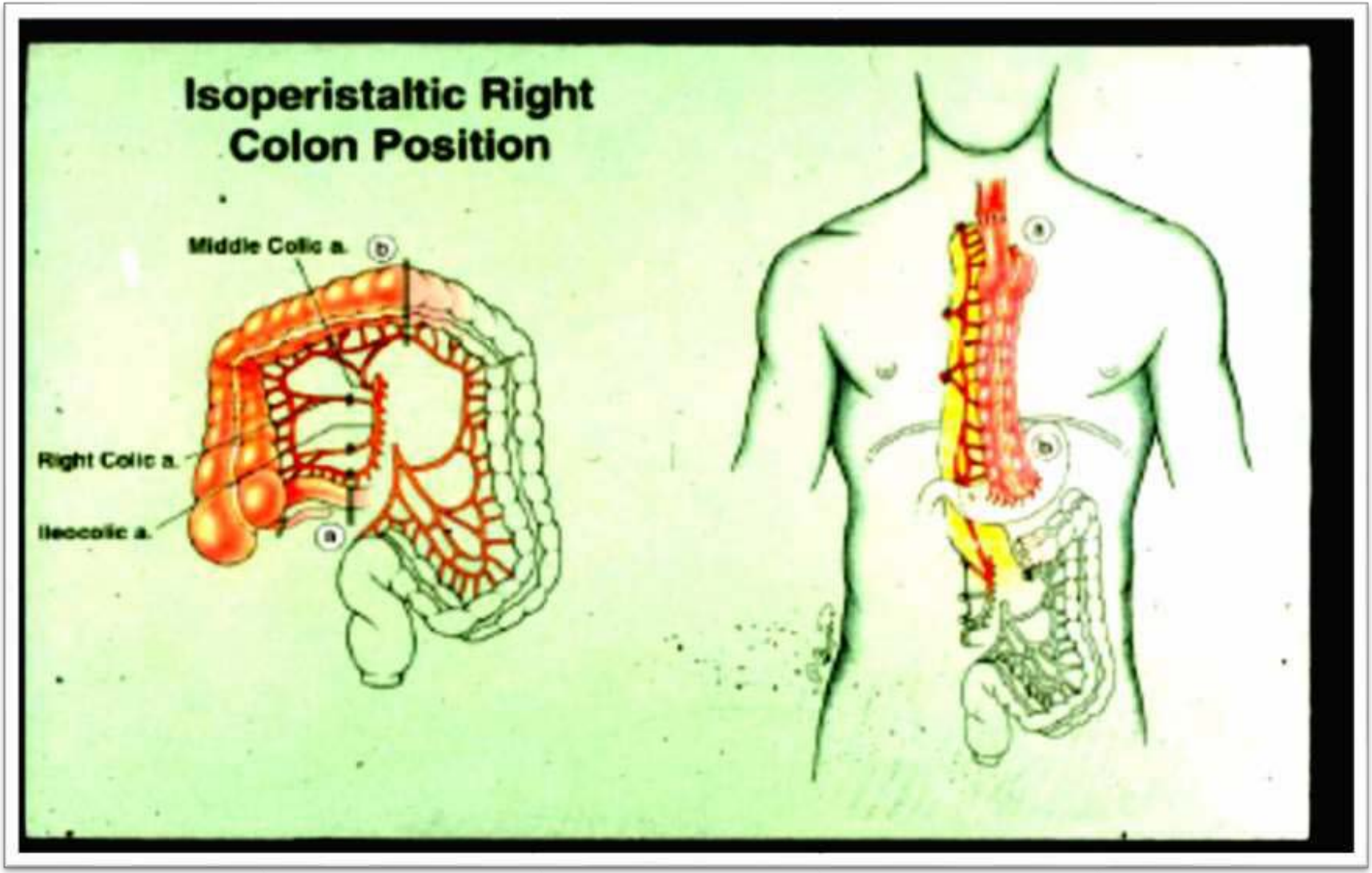


ΑΠΟΚΑΤΑΣΤΑΣΗ ΣΥΝΕΧΕΙΑΣ ΠΕΠΤΙΚΟΥ

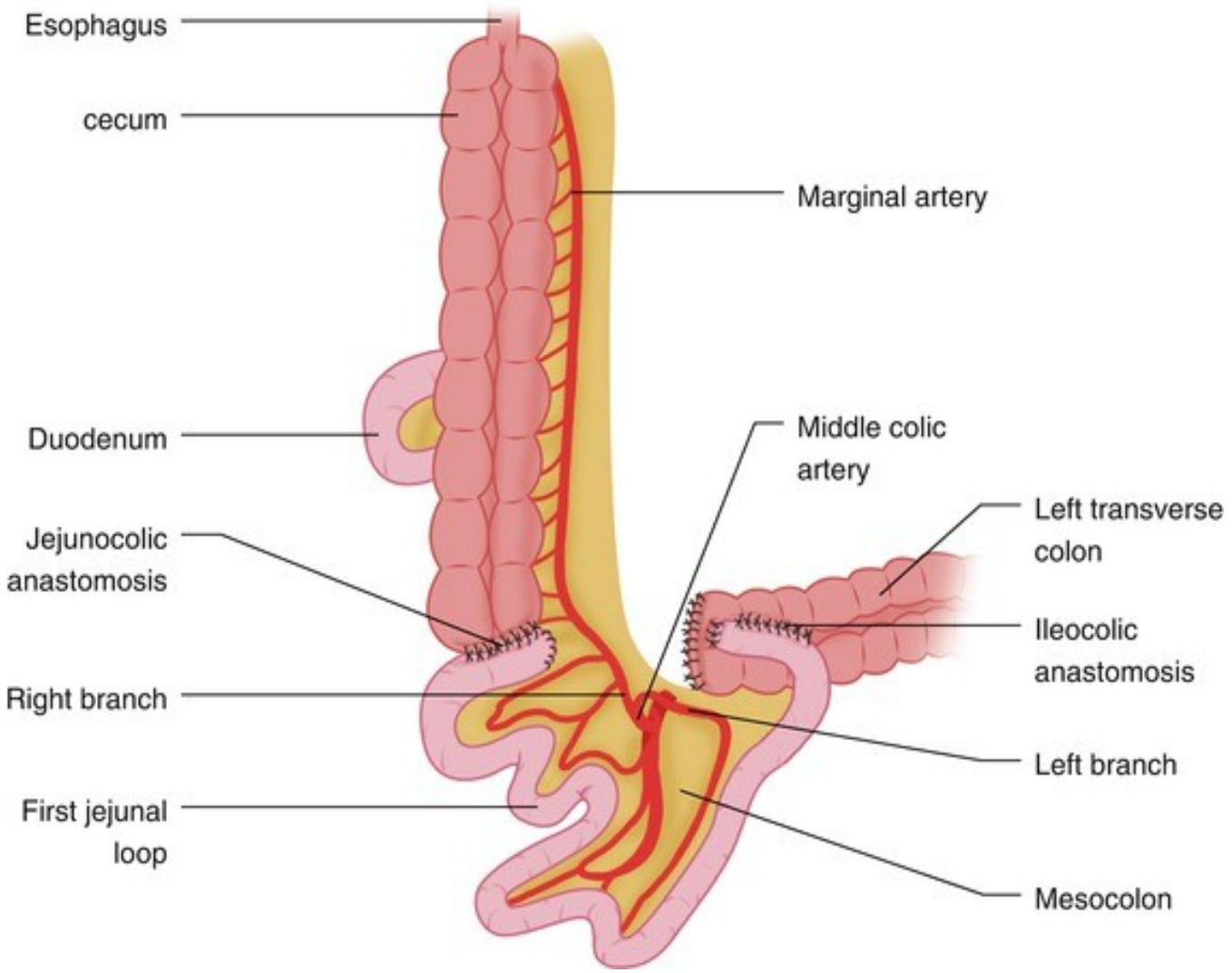
ΑΠΟΚΑΤΑΣΤΑΣΗ ΣΥΝΕΧΕΙΑΣ ΠΕΠΤΙΚΟΥ



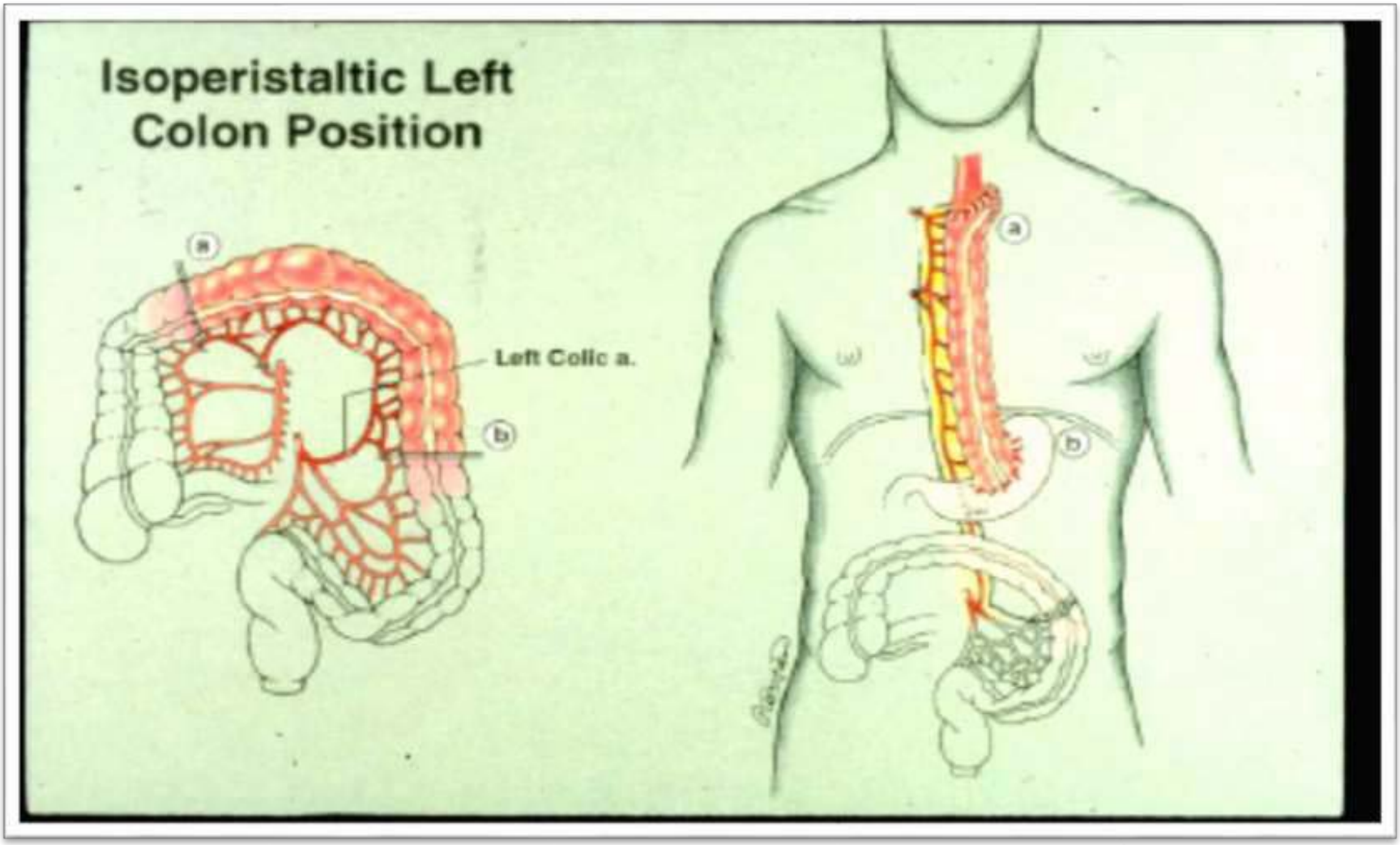
RIGHT COLON


Isoperistaltic Right
Colon Position
Middle Colic a.
b
Right Colic a.
Ileocolic a.
a

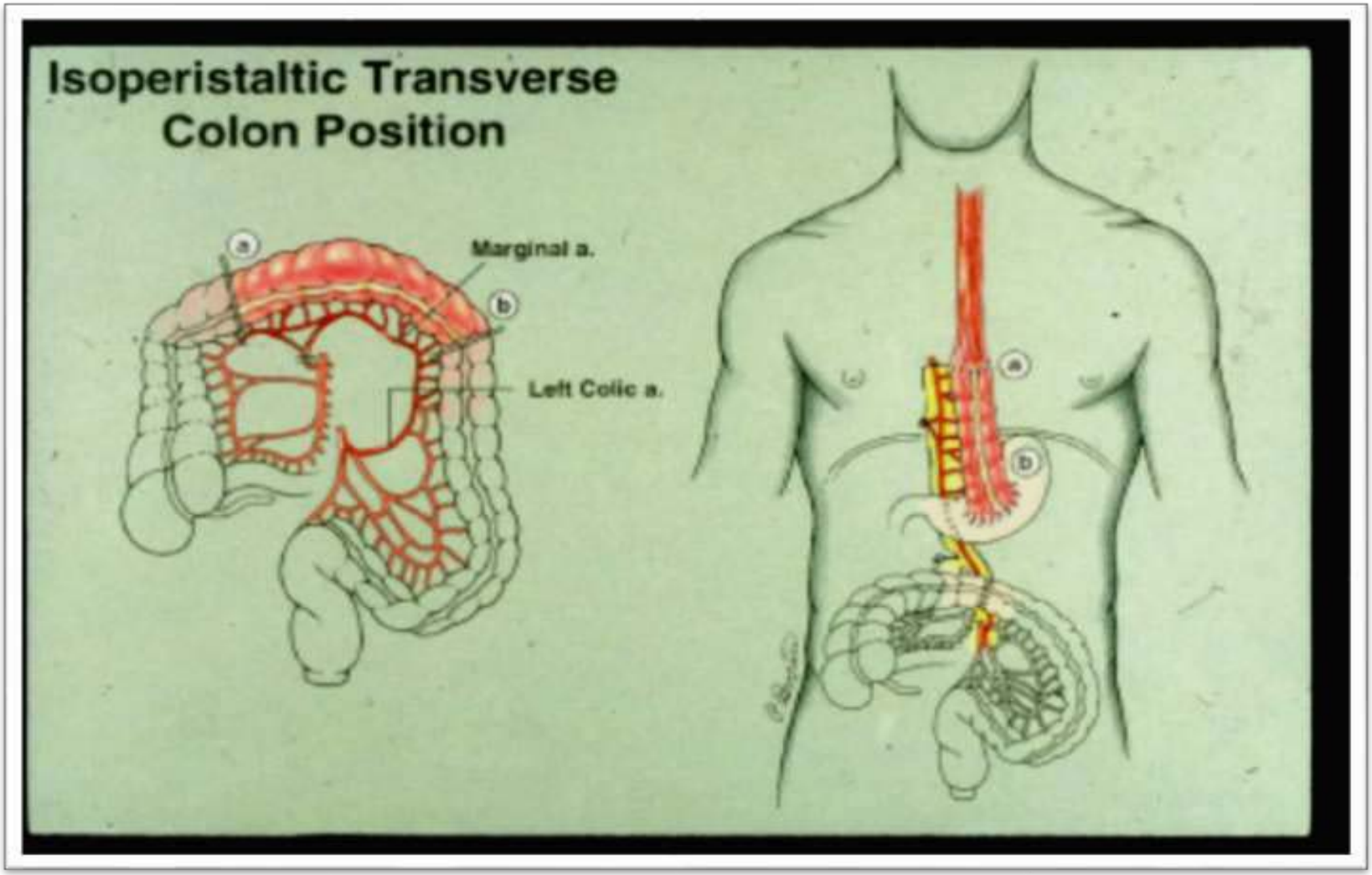
RIGHT COLON


Esophagus
cecum
Marginal artery
Duodenum
Middle colic artery
Left transverse colon
Jejunocolic anastomosis
Ileocolic anastomosis
Right branch
Left branch
First jejunal loop
Mesocolon

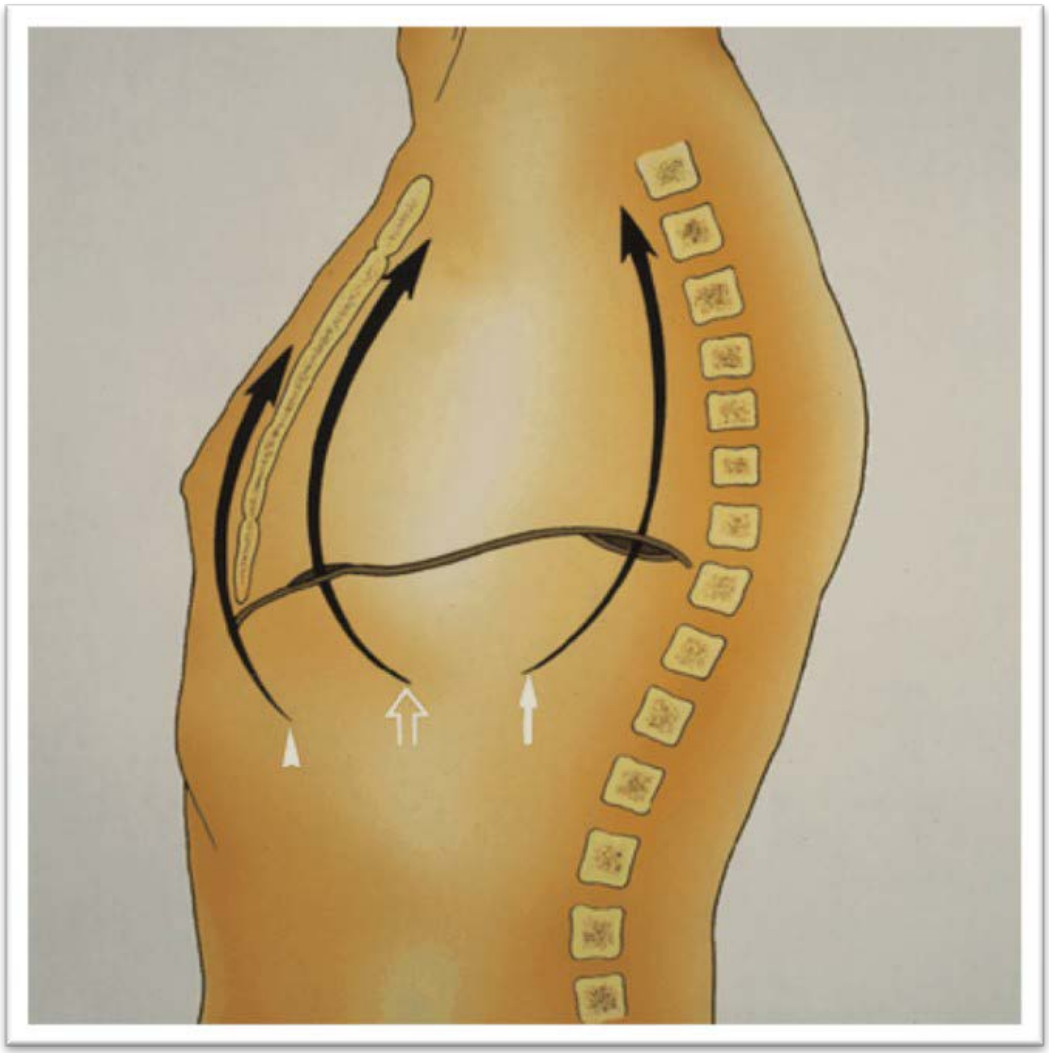
LEFT COLON



TRANSVERSE COLON

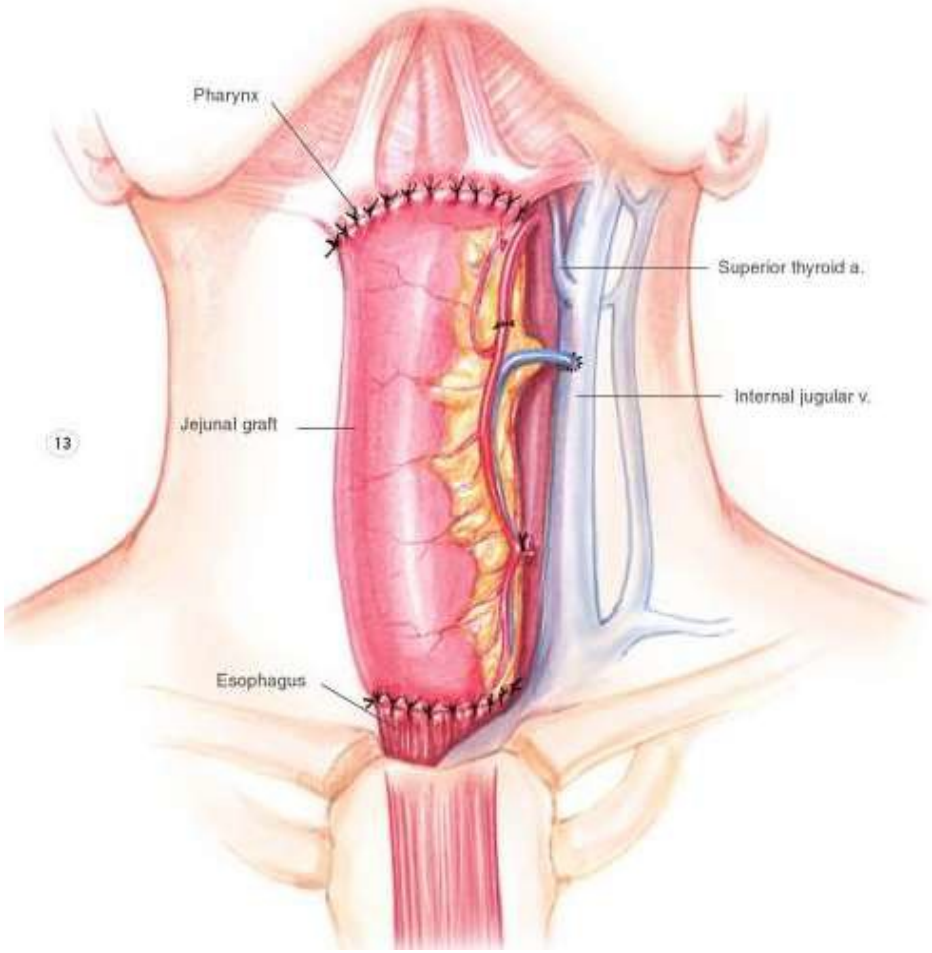


ROUTES OF RECONSTRUCTION



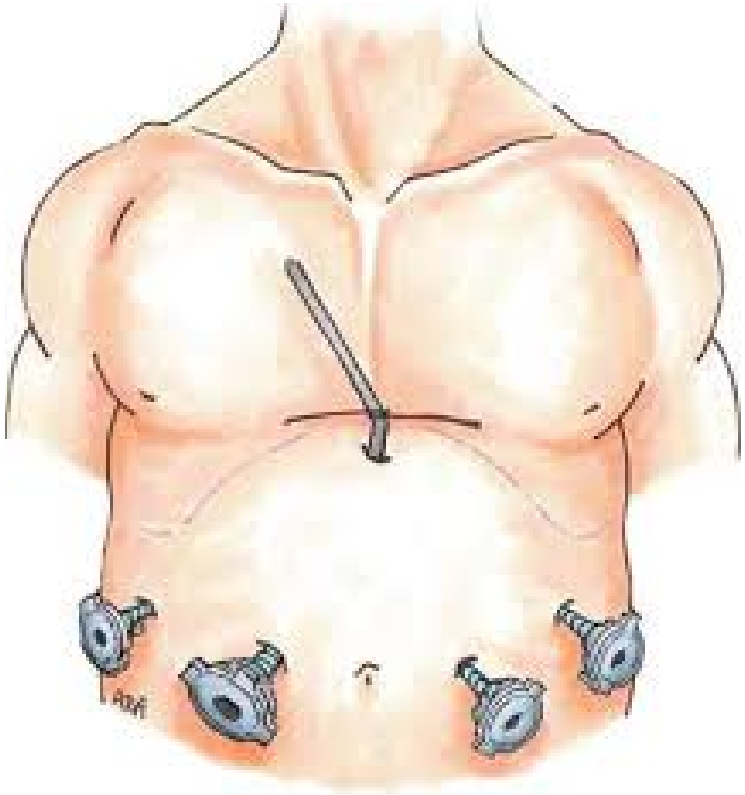
FREE JEJUNAL GRAFT

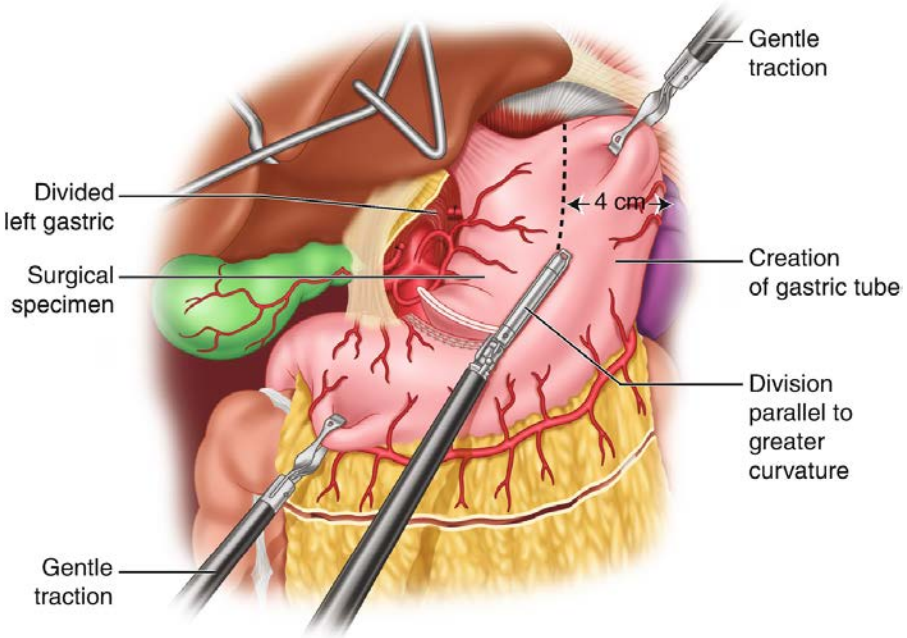
FREE JEJUNAL GRAFT



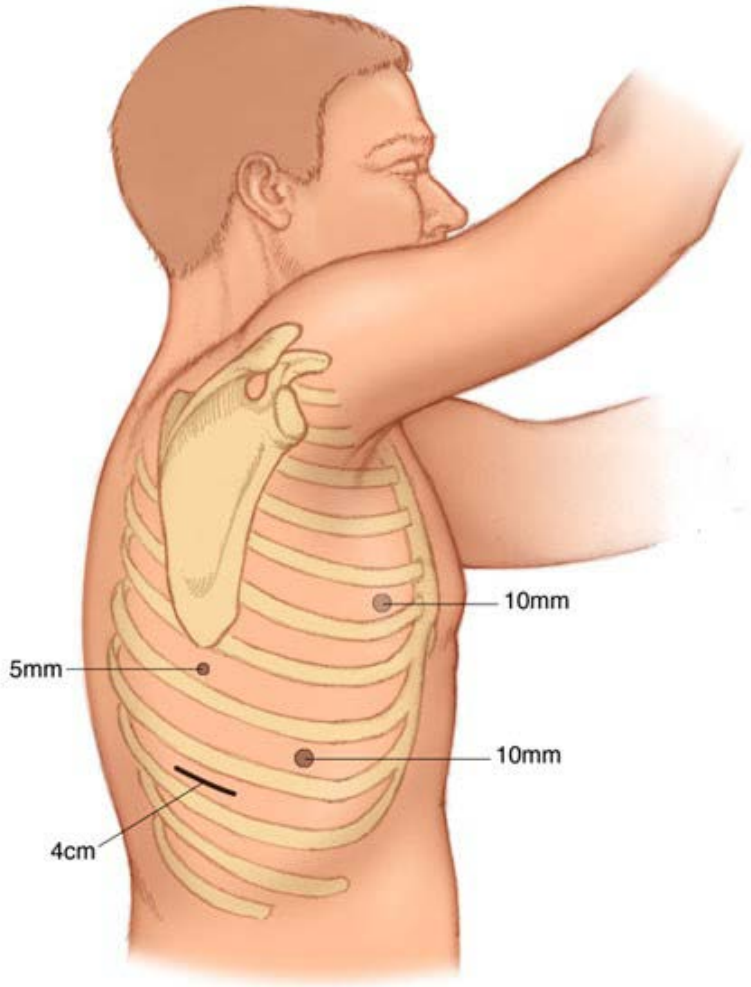
ΕΛΑΧΙΣΤΑ ΕΠΕΜΒΑΤΙΚΗ ΟΙΣΟΦΑΓΕΚΤΟΜΗ (MIE, HYBRID, RAMIE)

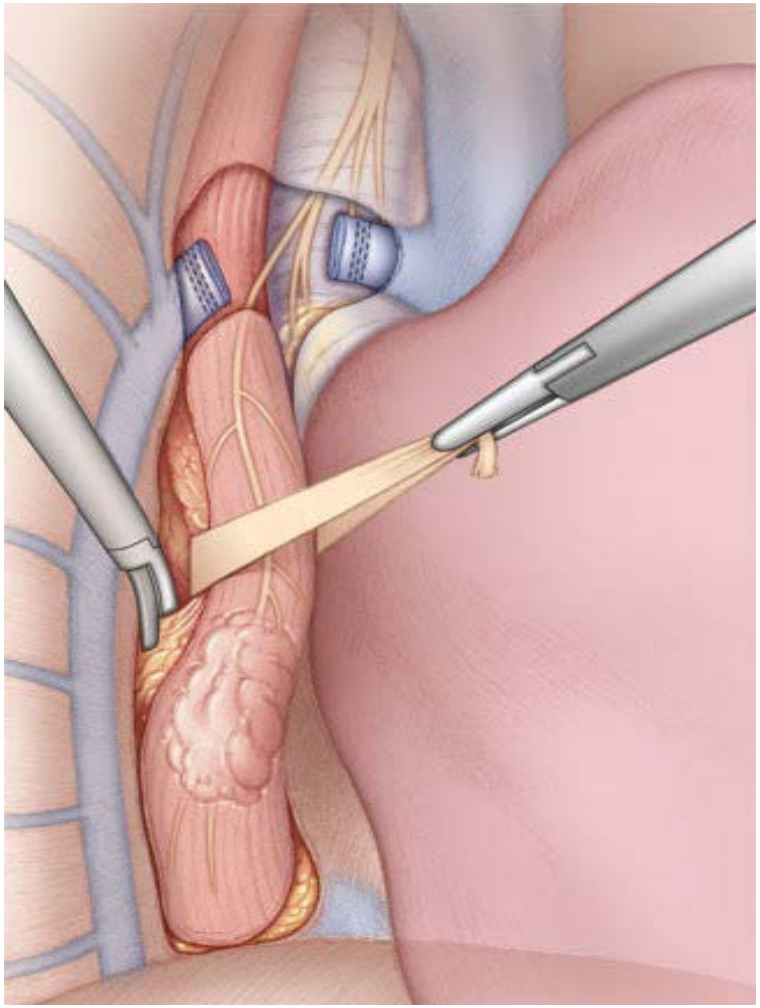
MINIMALLY INVASIVE ESOPHAGECTOMY (MIE)



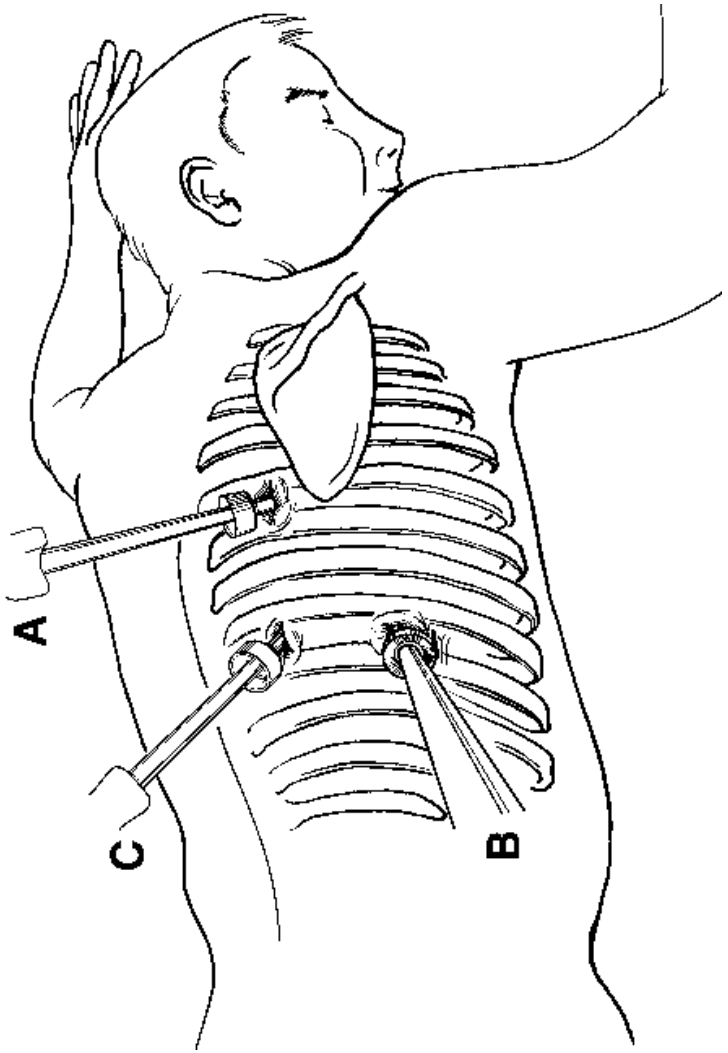


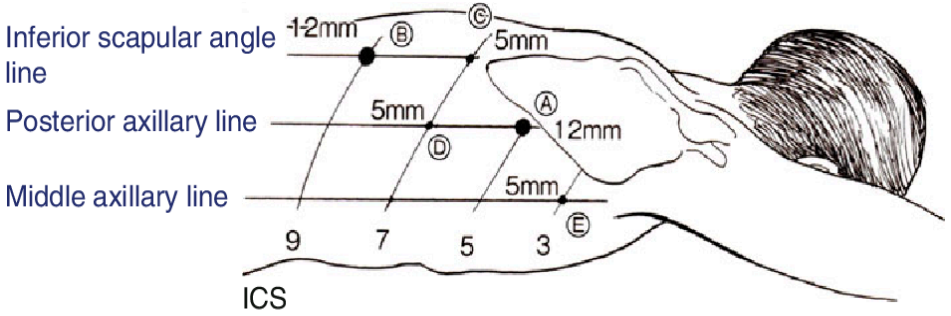
MINIMALLY INVASIVE ESOPHAGECTOMY (MIE)

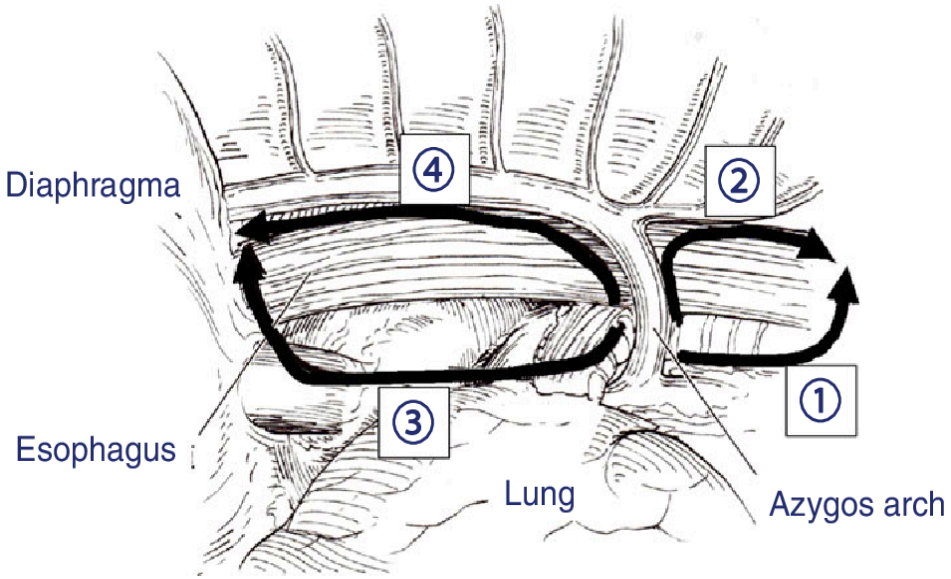




MINIMALLY INVASIVE ESOPHAGECTOMY (MIE)







HYBRID ESOPHAGECTOMY

HYBRID APPRAOCHES 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

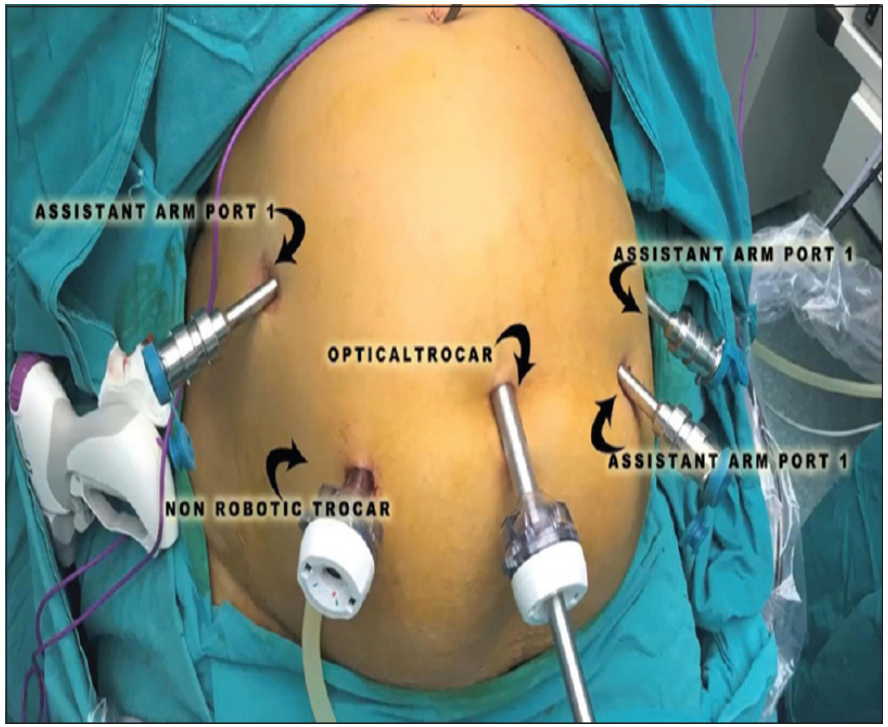
Thoracoscopic McKeown

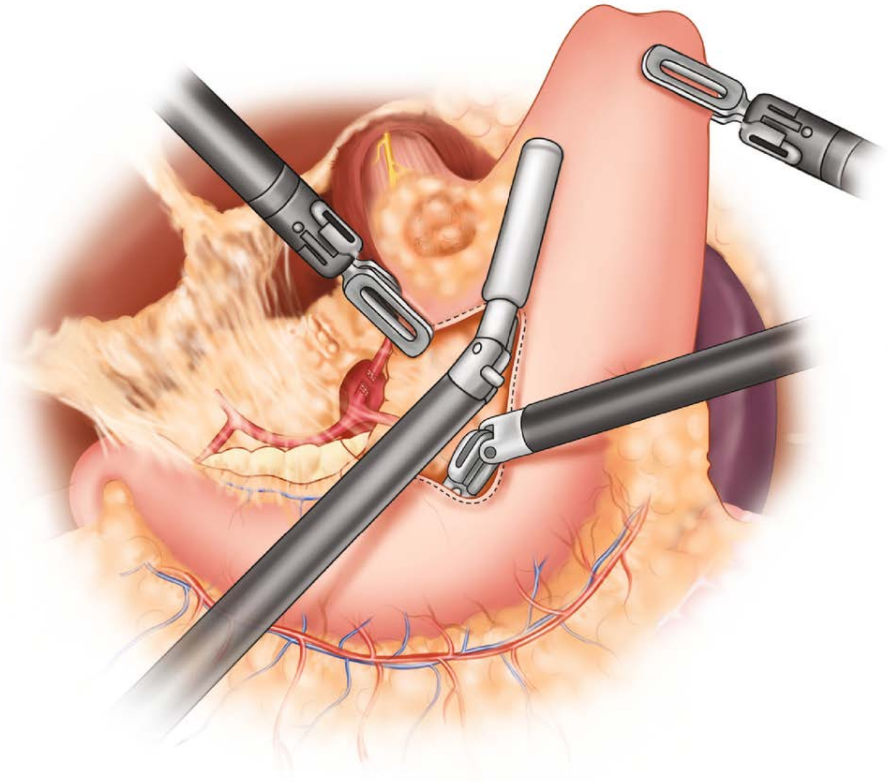
Right VATS

+

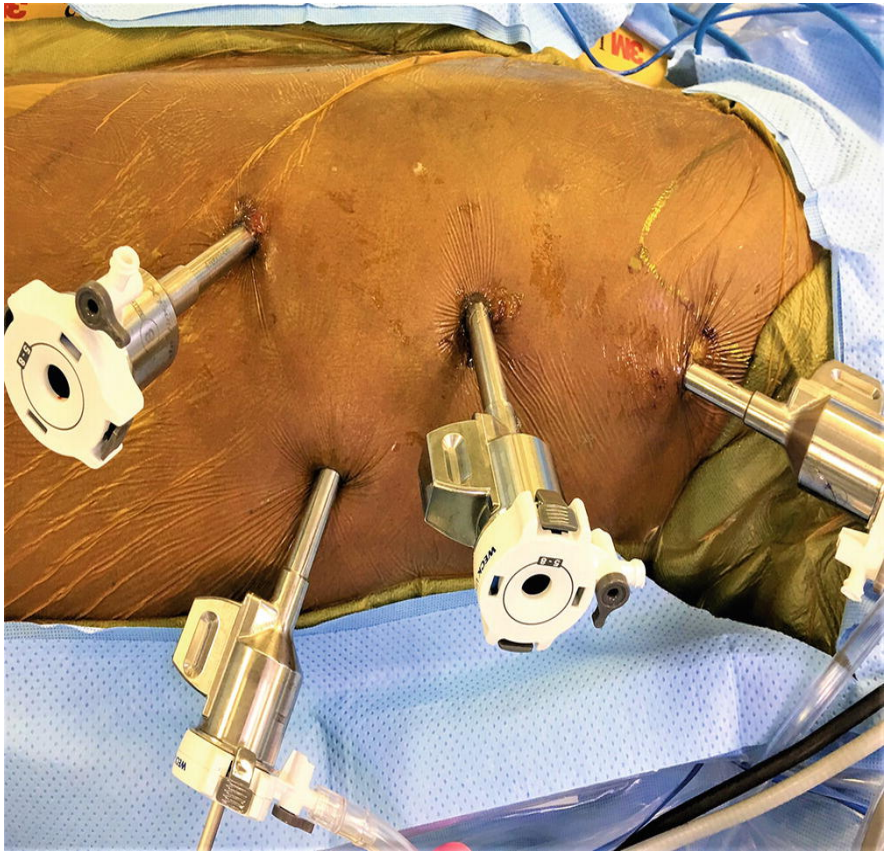
Laparotomy
and
Left cervicotomy

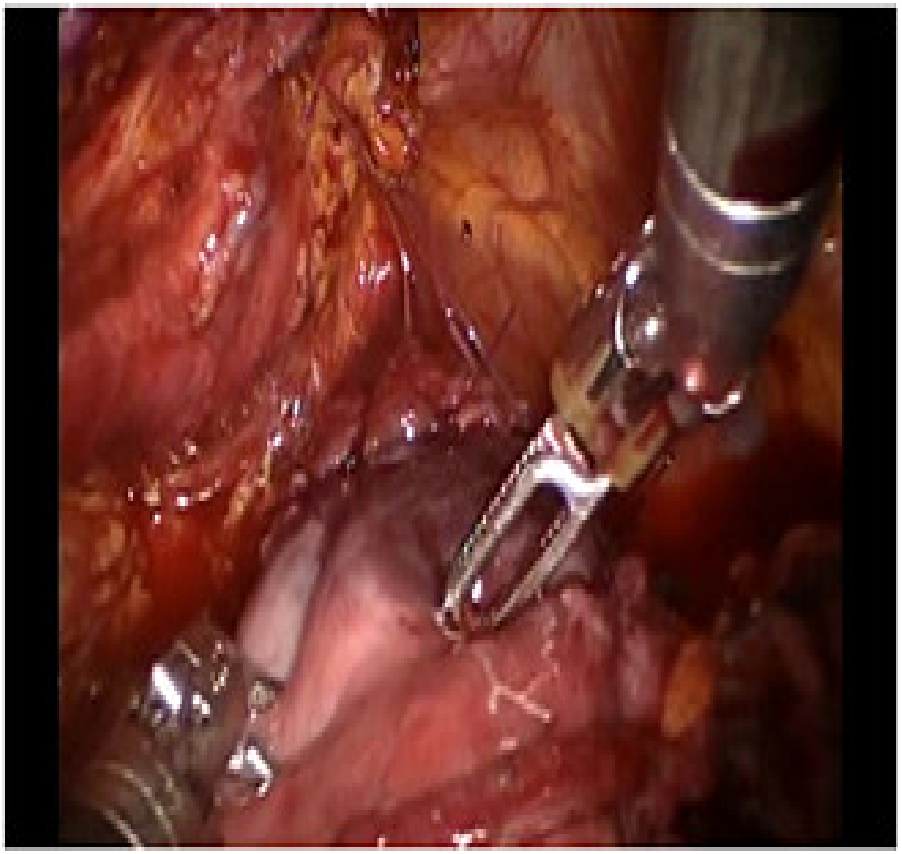
ROBOTIC ASSISTED MINIMALLY INVASIVE ESOPHAGECTOMY (RAMIE)





ROBOTIC ASSISTED MINIMALLY INVASIVE ESOPHAGECTOMY (RAMIE)





ΕΥΧΑΡΙΣΤΩ ΠΟΛΥ