Mercy Health
Care first

phn
NORTH WESTERN MELBOURNE
An Australian Government Initiative

# *Common gastroenterology problems in general practice and outpatient settings: optimising evaluation and management*

**Tuesday 17th March 2026**

*The content in this session is valid at date of presentation*

# *Acknowledgement of Country*

North Western Melbourne Primary Health Network and Mercy Health would like to acknowledge the Traditional Custodians of the land on which our work takes place, the Wurundjeri Woi Wurrung People, the Bunurong People, the Boon Wurrung People and the Wathaurong People.

We pay respects to Elders past, present and emerging as well as pay respects to any Aboriginal and Torres Strait Islander people in the session with us today.

# *Housekeeping – Zoom webinar*

## All attendees are muted

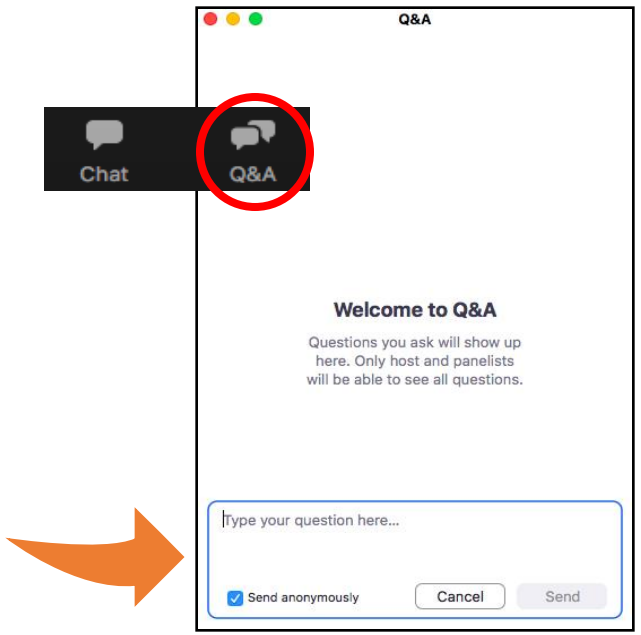
## Please ask questions via the Q&A box only

- Q&A will be at the end of the presentation
- Questions will be asked anonymously to protect your privacy

## This session is being recorded.

You will receive a link to this recording and copy of slides in post session correspondence.

**Type your questions in the Q&A box.**

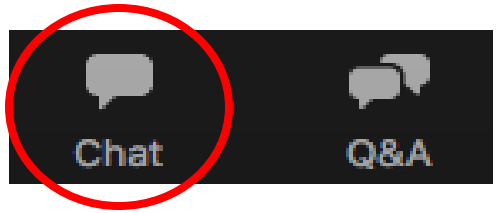


# *Housekeeping – Zoom webinar*

## Is your session name the same as your registration?

**To ensure we can issue your certificates and CPD** please ensure you have joined the session using the same name as your event registration (or phone number, if you have dialled in).

**Not sure if your name matches**, send a Chat message to 'NWMPHN Education' to identify yourself.

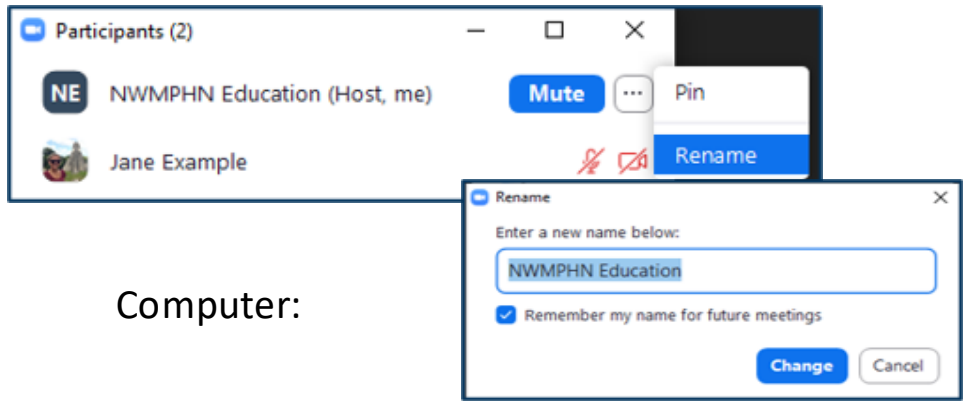


# *Housekeeping – Zoom webinar*

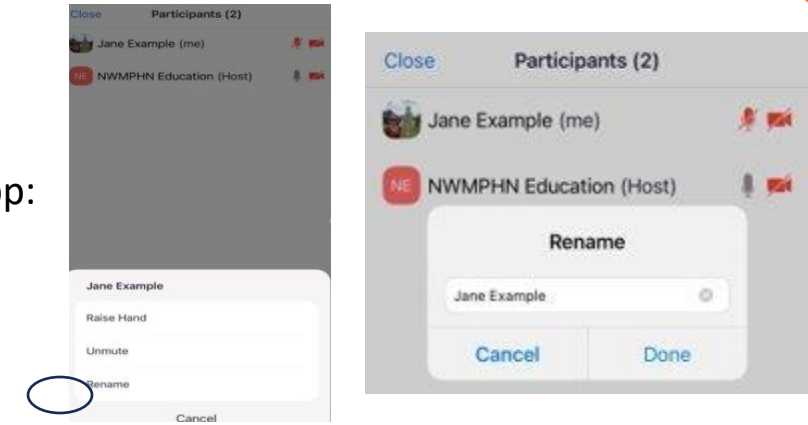
## How to rename yourself

1. Click on **Participants**
2. If using
   - App: click on your name
   - Computer: hover over your name and click the 3 dots
   - Mac: hover over your name and click More
3. Click on **Rename**
4. Enter the name you registered with and click **Done** / **Change** / **Rename**

Computer:




Phone/App:



# Speakers

## ***Dr Fiona Jones,*** *Gastroenterologist, Werribee Mercy Hospital*

Dr Fiona Jones is a gastroenterologist with specialised training in therapeutic luminal endoscopy, including device-assisted enteroscopy and capsule endoscopy. She completed an interventional endoscopy fellowship at St Vincent's Hospital Melbourne. She trained in Ireland, earning her medical degree and MD from University College Dublin, where her research focused on inflammation in IBD. She has presented and published widely in the field.

## ***Dr Niranjan Arachchi,*** *Gastroenterologist, Werribee Mercy Hospital*

Dr Arachchi is a general gastroenterologist with expertise in endoscopy, liver disease and inflammatory bowel disease. He trained at the University of Melbourne, completed his FRACP in 2006 and undertook postgraduate research at St Vincent's Hospital. He worked full-time at Western Health before becoming a VMO at Western Health and Werribee Mercy. He also holds a Master's degree in Clinical Epidemiology from the University of Sydney.

## ***Dr Catherine Croagh,*** *Head of Gastroenterology, Werribee Mercy Hospital*

Dr Catherine Croagh is the Head of Gastroenterology at Werribee Mercy Hospital. She trained at the University of Melbourne and completed specialist gastroenterology training across major Melbourne hospitals. She holds a Master of Public Health and an MD focused on Hepatitis B biomarkers and disease progression. Her interests include metabolic-associated liver disease and improving access to high-quality care for the Werribee community.

HealthPathways Melbourne

# Common gastroenterology problems in general practice and outpatient settings: optimising evaluation and management

*17 March 2026*

HealthPathways Melbourne

# Your Clinical Management and Referral Resource

**Localised Clinical Pathways**
(Evidence-based guidance adapted for Melbourne clinicians)

**Referral Information**
(Clear referral instructions for local health services and hospitals)

**Regular Updates**
(Pathways reviewed and updated regularly by Clinical Editors)

**CPD Hours**
(Track and record CPD activities directly through Pathway page)

**Collaborative Development**
(Created by GPs, specialists, allied health and other health professionals)

**Easy Access**
(Web-based platform, mobile-friendly for point-of-care use)

**Streamlined Workflow**
(Quick navigation with Assessment, Management and Referral sections all in one place)

**Free for Clinicians**
(No cost access for all health professionals in North Western and Eastern Melbourne PHN catchments)

# Search for the Dyspepsia and Heartburn (GORD) pathway on the Homepage

Use the search bar to **quickly** locate clinical pathways or conditions


Use the left-hand menu to access clinical categories — **quick and easy to navigate**

**Essential quick-access** links for latest updates, Pathway updates, clinical resources and MBS items

Click '**Send Feedback**' to add comments and questions about this pathway.

# Relevant and Related Pathways

## Relevant Pathways

Dyspepsia and Heartburn/GORD

Acute Chest Pain

Dysphagia

NSAIDs and Dyspepsia or Heartburn

Helicobacter Pylori (H. pylori)

## Related Pathways

Biliary Colic and Cholecystitis

Bowel Cancer

Coeliac Disease in Adults

Constipation in Adults

Diarrhoea in Adults

Inflammatory Bowel Disease (IBD)

Liver Conditions

CPD Hours for HealthPathways Use

## Referrals

Acute Hepatobiliary and Upper Gastrointestinal Surgery Referral (Same-day)

Non-acute Hepatobiliary and Upper Gastrointestinal Surgery Referral (> 24 hours)

Acute Gastroenterology Referral (Same-day)

Non-acute Gastroenterology Referral (> 24 hours)

Non-acute Liver Referral (> 24 hours)

Transient Elastography (FibroScan) Referrals

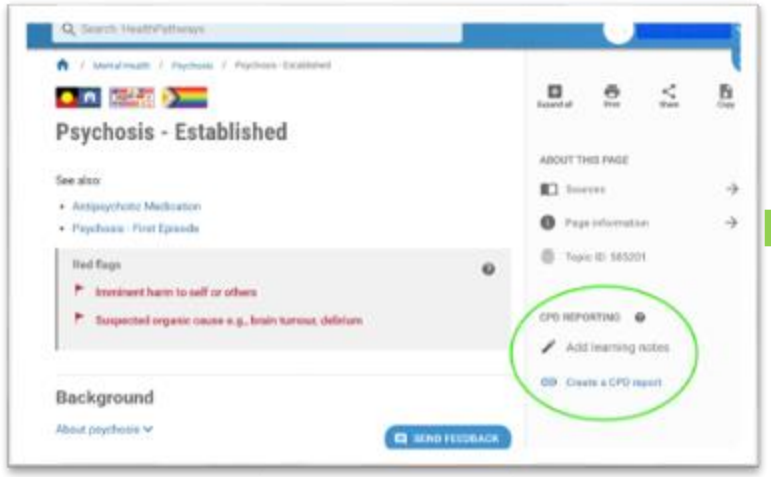
Acute Colorectal Surgery Referral (Same-day)

Non-acute Colorectal Surgery Referral (> 24 hours)

Gastrointestinal Investigations

Statewide Referral Criteria for Specialist Clinics

# Log CPD Effortlessly with HealthPathways CPD Reporting

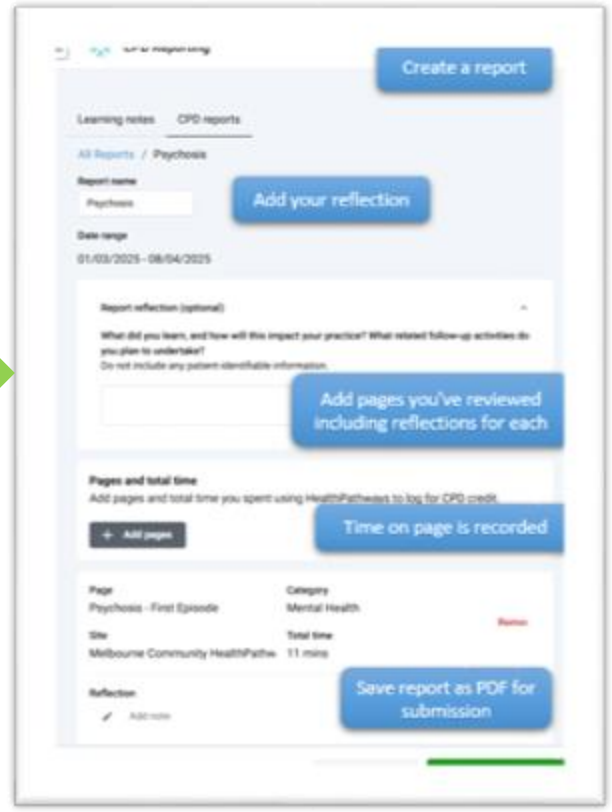



## Step 1: Access a Pathway Page

- Navigate to a clinical pathway (e.g., *Psychosis – Established*).
- Click **"Add learning notes"** or **"Create a CPD report"** to begin tracking your CPD activity.


## Step 2: Add Learning Notes

- Reflect on what you learned and how it will impact your practice.
- Include any planned follow-up activities.
- These notes are saved to your CPD record.




## Step 3: Generate Your CPD Report

- Go to the **CPD Reporting** section.
- Add reflections, review pages, and confirm time spent.
- Export your report as a **PDF for submission**.

For further information on the CPD reporting tool, please see these videos:

- How to create a CPD report
- How to add learning notes

# Stay Informed: Access Case Studies and Monthly Bulletin

**Latest Case Study**


**Monthly Bulletin**


## Real clinical scenarios for everyday GP practice

- Concise, practical case studies designed to reflect real presentation in General Practice.
- Includes management summaries, pathway links and local service consideration for quick navigation.
- Access all case studies here.

## Monthly updates straight to your inbox

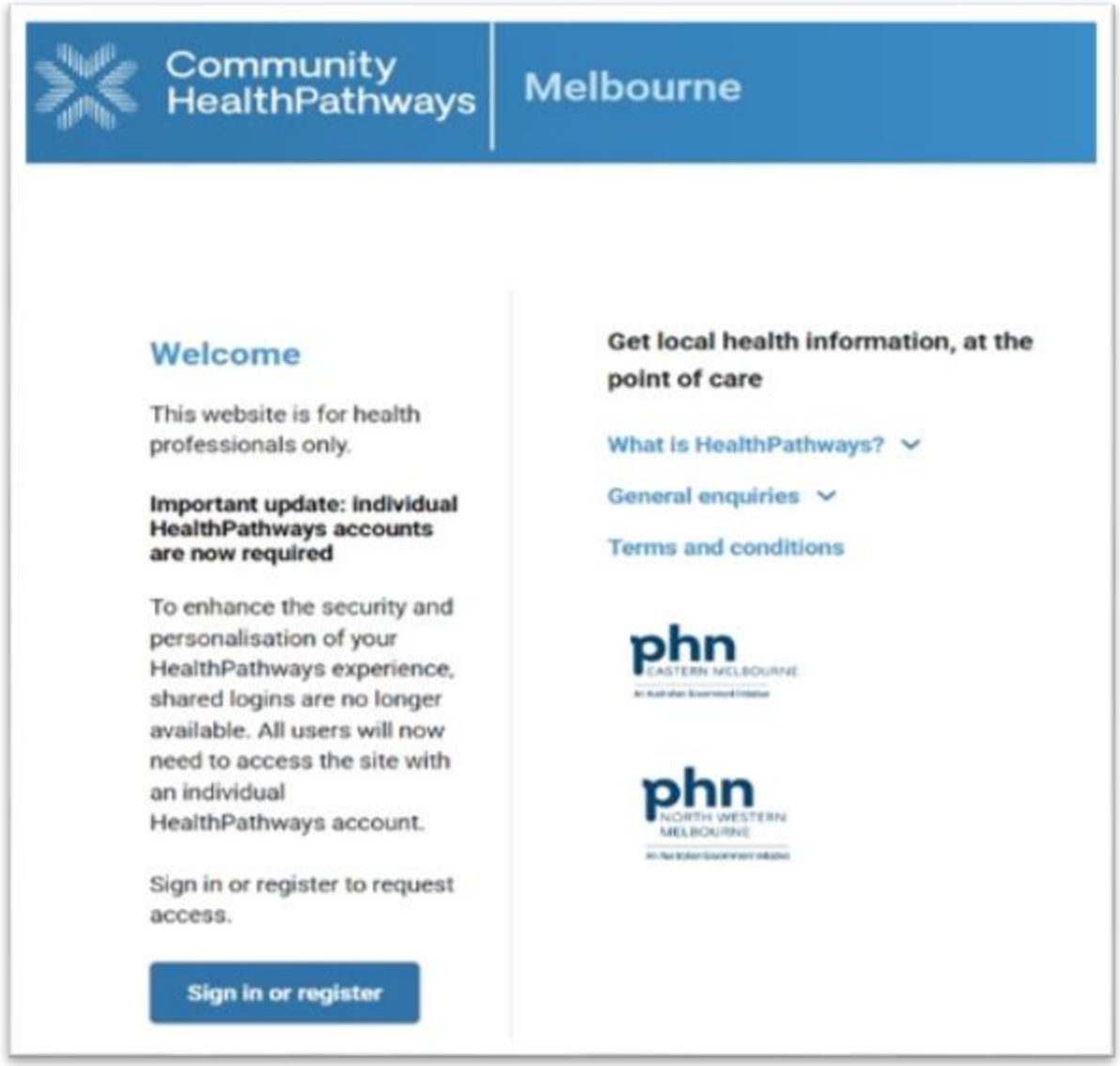
- Be the first to know about pathway updates, service changes , new case studies and employment opportunities

**Subscribe to the HealthPathways Melbourne Monthly bulletin or contact us at info@healthpathwaysmelbourne.org.au**

# Access Now: Sign In or Scan to Register

Please click on the **Sign in or register** button to create your individual account or scan the QR code below.

If you have any questions, please email the team info@healthpathwaysmelbourne.org.au

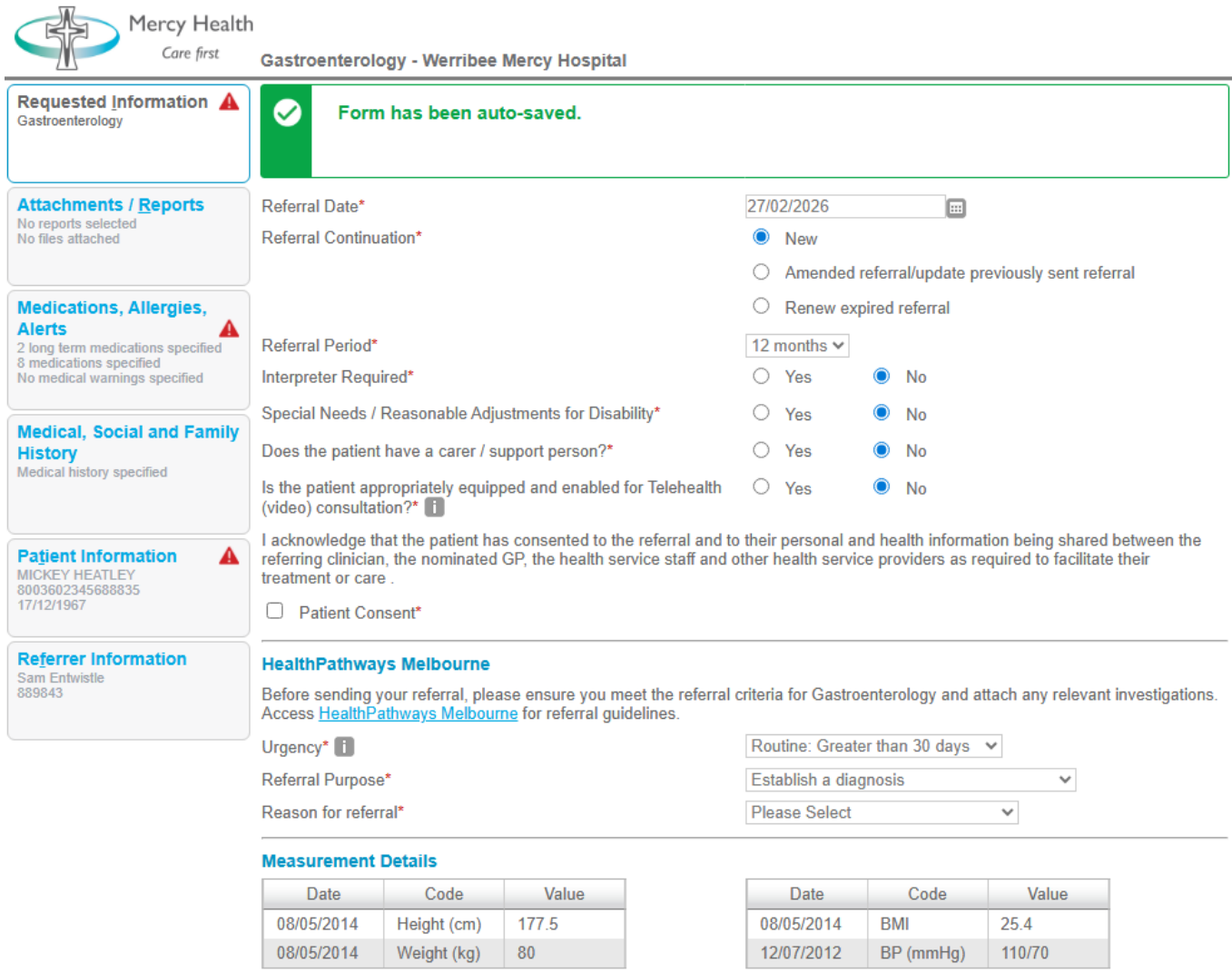


# *Mercy Health update*

**Mercy Health encourages all referrals to our Outpatient Specialist Clinics to be submitted via eReferrals using HealthLink SmartForms.**

- HealthLink is built into most general practice software, making referrals quick and seamless.
- It supports secure information exchange and allows you to easily attach test results and reports.
- Using eReferrals helps ensure referrals reach the right clinic promptly and reduces follow-up delays.

For more information, visit our [HealthLink eReferral information website]



# *Mercy Health Update*

Mercy Health
Care first

## Feedback form for Health Professionals

The Primary Care Liaison Unit would love to hear from you!

Please provide any feedback easy and quick via our online feedback form.

For any education requests please complete our education request form.

For more information visit our Primary Care Liaison website: https://health-services.mercyhealth.com.au/health-professionals/primary-care-liaison-unit

# *Mercy Health update*

**Stay informed about key updates from the hospital.**

Subscribe to our new, mobile-friendly quarterly newsletter to receive essential hospital updates, clinical insights, and upcoming education opportunities tailored for primary care.

[Register for the Primary Care Liaison newsletter today.]


# Investigation and management of GORD and it's complications including Barrett's oesophagus

Dr Fiona Jones

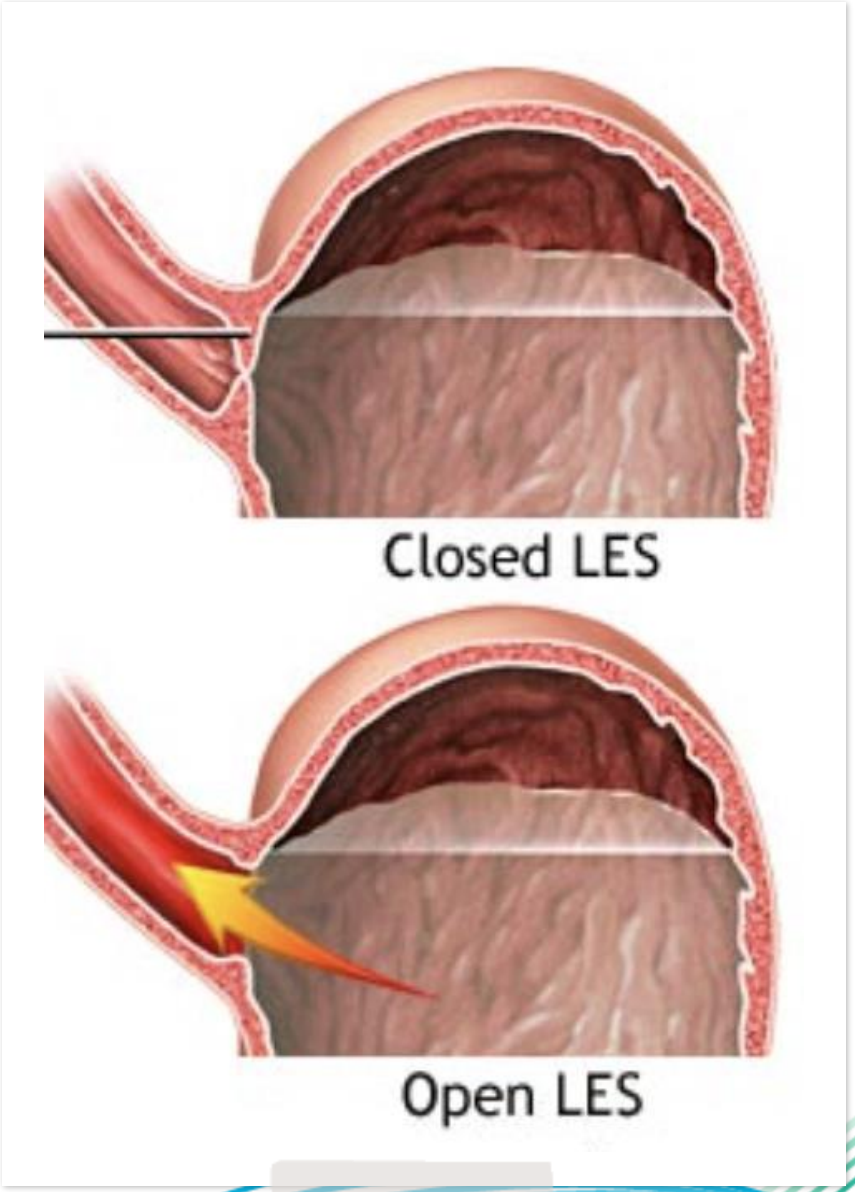
# Overview

Mercy Health
Care first

- What is GORD
  - GERD severity: LA classification (and follow-up)
  - Management algorithm
    - Lifestyle, medical, endoscopic and surgical
  - Complications of long-term GORD
- Barrett's
  - Screening
  - Management
  - Surveillance
- PPI
  - Potency
  - Risk
  - Tapering appropriately

# GORD:

- One of the most common diagnoses in General Practice and Gastroenterology Practice
- Prevalence of GERD ranges from 15-30% percent in Western countries and 5 -10% percent in Asia. The incidence in the Western world is approximately five per 1000 person-years or 0.5 percent per year
- From a conventional pathophysiological perspective, GERD is conceptualized as incompetence of the antireflux barrier at the oesophagogastric junction



# Definition of GORD:A problem: Symptoms of heartburn do not correlate with objective evidence of GORD

Mercy Health
Care first

**Clinical: Montreal Definition**

- GERD was defined as a condition that develops when the reflux of stomach contents causes troublesome symptoms (heartburn +/- regurgitation) and/or complications.
- Recognition of extra- oesophageal manifestations:  laryngitis, cough, asthma, and dental erosions as possible GERD syndromes.

**Objective: Lyon Consensus**

- The modern definition of actionable GERD requires conclusive evidence of reflux-related pathology on endoscopy and/or abnormal reflux monitoring (using Lyon consensus thresholds) in the presence of compatible troublesome symptoms

Mercy Health
Care first

# Approach to Management

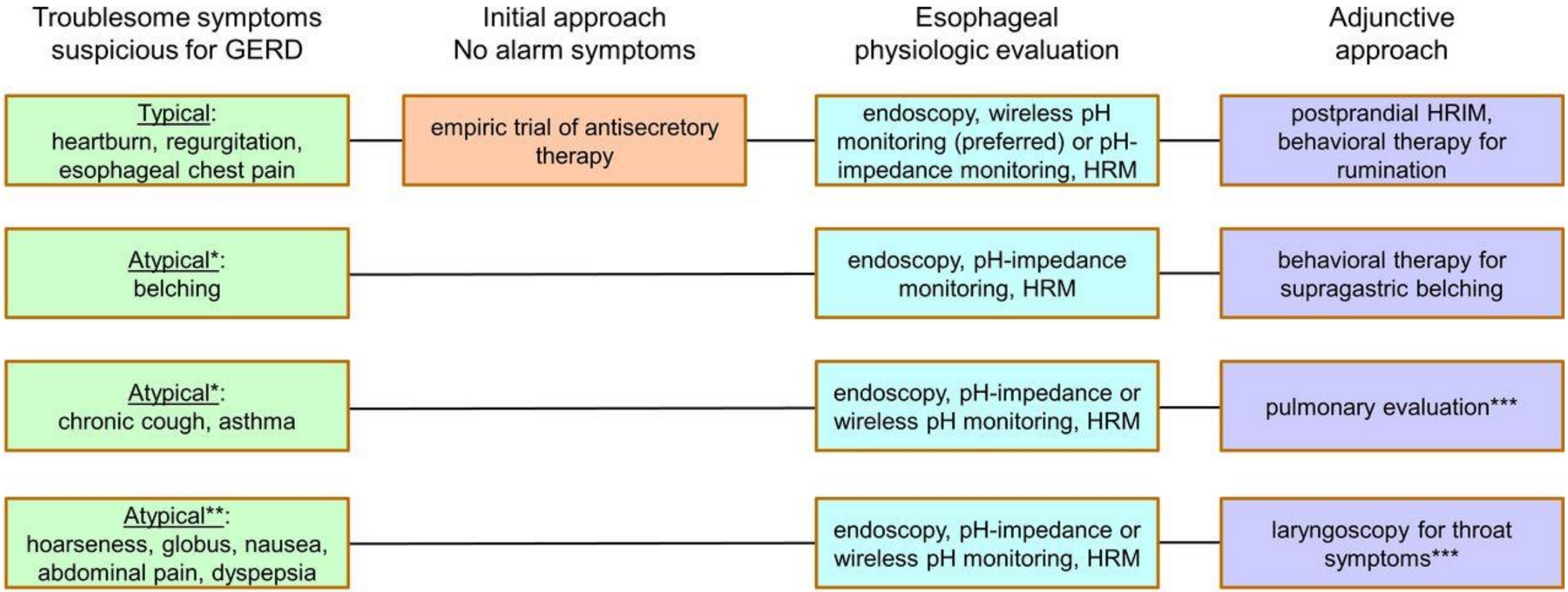
1) Establish likelihood of pathologic GERD and discern which mechanisms may be driving symptoms and develop shared decision making care plan

- Typical oesophageal symptoms- 70% specific
- Obesity/known HH
- Extra-oesophageal- low likelihood
- Stress/anxiety- visceral hypersensitirity

*2) Patient education: standardized educational material on GERD mechanisms, weight management, lifestyle and dietary behaviours, relaxation strategies, and awareness about the brain-gut axis relationship to patients with reflux symptoms*

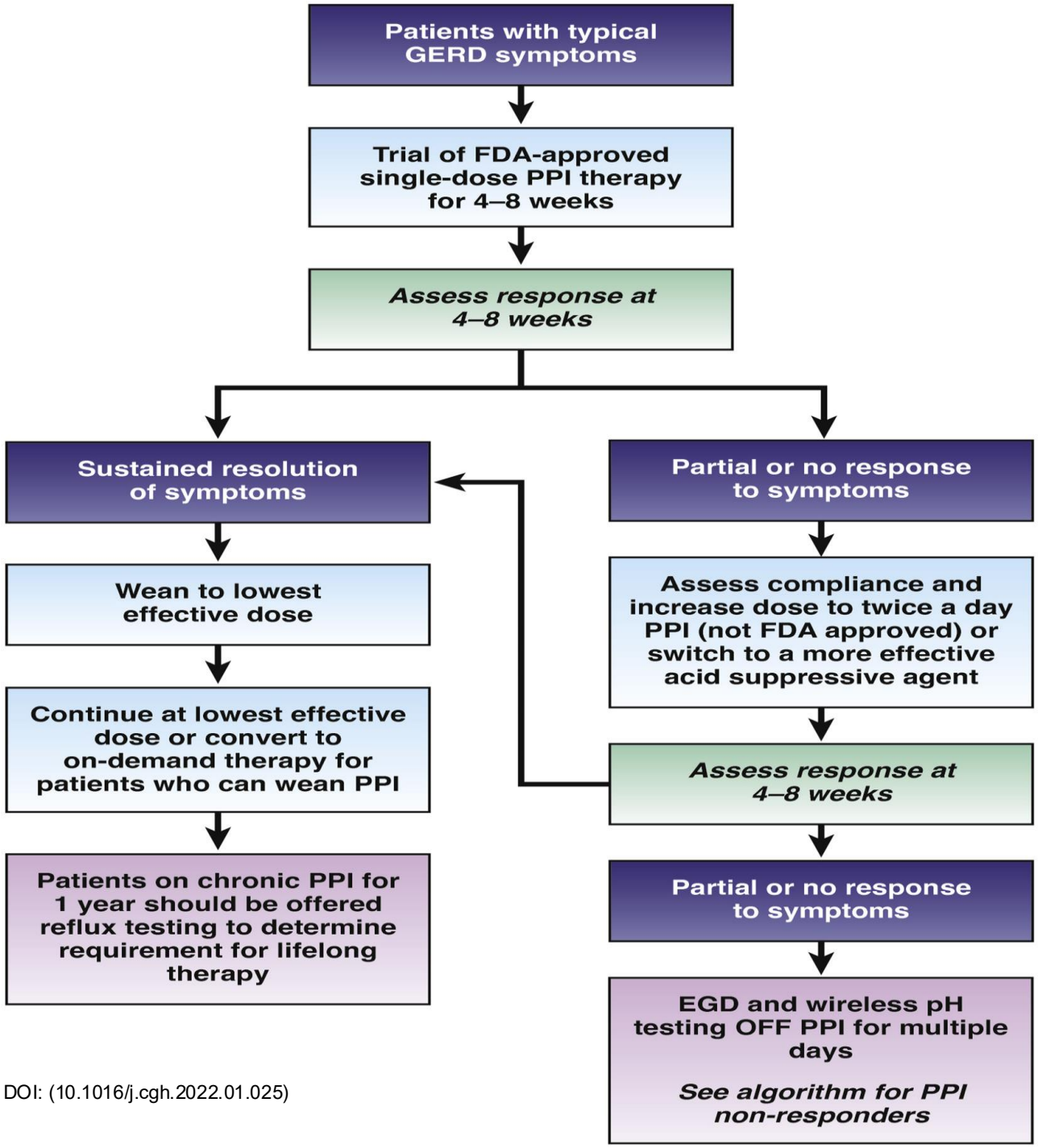
*3) PPI Trial :* Clinicians should provide patients presenting with troublesome heartburn, regurgitation, and/or non-cardiac chest pain without alarm symptoms a 4- to 8-week trial of single-dose PPI therapy

# Troublesome typical and atypical symptoms suspicious for gastro-oesophageal reflux disease (GERD), and usual approach to evaluation of these symptoms.




* likelihood of GERD is lower than with typical symptoms, testing is performed to identify or rule out a reflux basis for symptoms
** likelihood of GERD is very low, upfront testing is typically not recommended except to rule out a reflux basis for symptoms
***adjunctive approaches may precede esophageal evaluation to rule out primary pulmonary and laryngeal disorders

**C Prakash Gyawali et al. Gut 2024;73:361-371**
Copyright © BMJ Publishing Group Ltd & British Society of Gastroenterology. All rights reserved.




*Clinical Gastroenterology and Hepatology* 2022 20984-994.e1DOI: (10.1016/j.cgh.2022.01.025)
Copyright © 2022 AGA Institute Terms and Conditions

# Routine modifications to improve esophageal health if you have symptoms:

Yadlapati R, Gyawali CP, Pandolfino JE. *Best Practice Advice for Personalized Approach to the Evaluation and Management of GERD.* 2022.

Mercy Health
Care first

## Stress reduction

Integrate methods to reduce stress in your life to disrupt the overstimulated gut-brain connection. These include mindfulness, meditation, and massage therapy.

## Belly breathing

Belly breathing or diaphragmatic breathing can help strengthen the diaphragm and reduce esophageal disease and reflux. A video on diaphragmatic breathing can be found here:

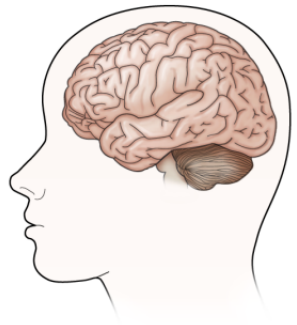
https://www.youtube.com/watch?v=UB3tSaiEbNY

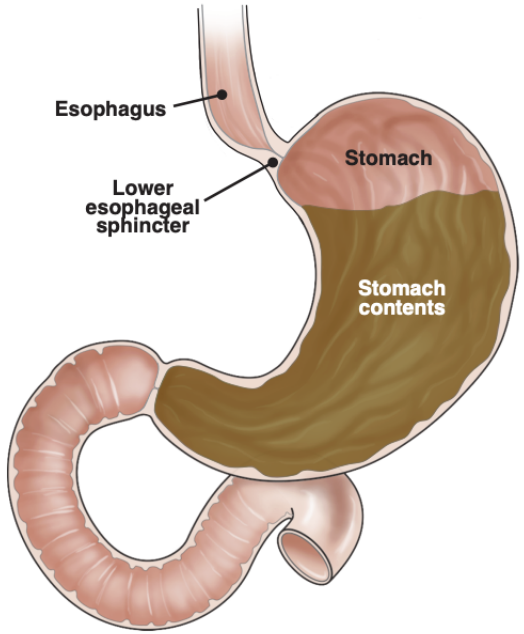
## Avoid late meals

Lying down with a full stomach may increase the risk of acid reflux. By avoiding eating within three hours before bedtime and avoiding late night snacks, nighttime reflux may be reduced.

## If you have nighttime symptoms, raise the head of your bed

Raising the head of your bed by 6 to 8 inches raises the head and shoulders higher than the stomach, allowing gravity to prevent acid from refluxing. Raising the head of the bed can be done with blocks of wood/bricks under the legs of the bed or a foam wedge under the mattress. Several manufacturers have developed commercial products for this purpose. However, it is not helpful to use additional pillows; this can cause an unnatural bend in the body that increases pressure on the stomach, which can worsen acid reflux.






## Avoid trigger foods

If you have noticed that certain food items trigger your symptoms, it will be useful to avoid these items, as some foods may relax the lower esophageal sphincter and promote acid reflux. However, not all patients have the same trigger foods.

## Quit smoking

Saliva helps to neutralize refluxed acid, and smoking reduces the amount of saliva in the mouth and throat. Smoking also lowers the pressure in the lower esophageal sphincter and provokes coughing, causing frequent episodes of acid reflux in the esophagus. Quitting smoking can reduce or eliminate symptoms of mild reflux.

## Chew gum or use oral lozenges

Chewing gum or using lozenges can increase saliva production, which may help to neutralize and clear stomach acid that has entered the esophagus.

## Weight management

Losing weight, particularly weight around the abdomen, is a powerful tool to improve GERD symptoms for patients that are overweight. Maintaining a healthy weight is important to control GERD in patients with a normal weight.

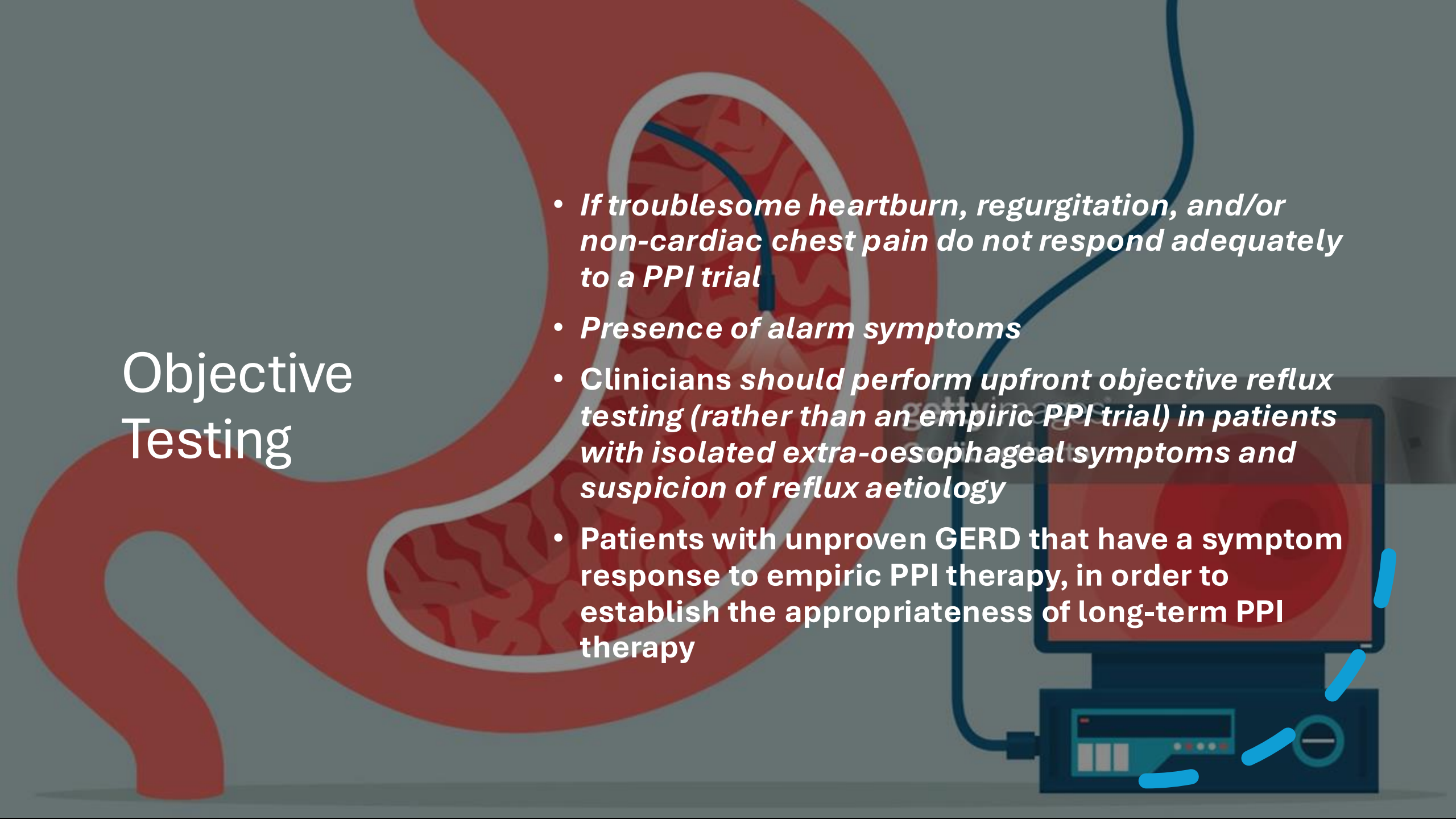
## Avoid tight fitting clothing

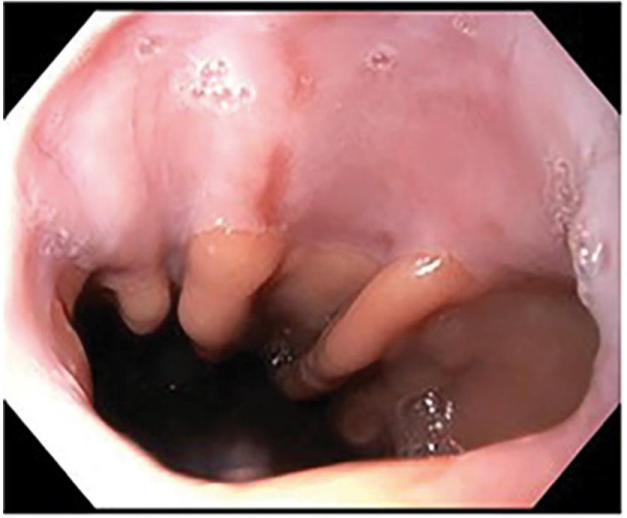
Tight-fitting clothing can increase discomfort, and may also increase pressure in the abdomen, forcing stomach contents into the esophagus.

aga American Gastroenterological Association

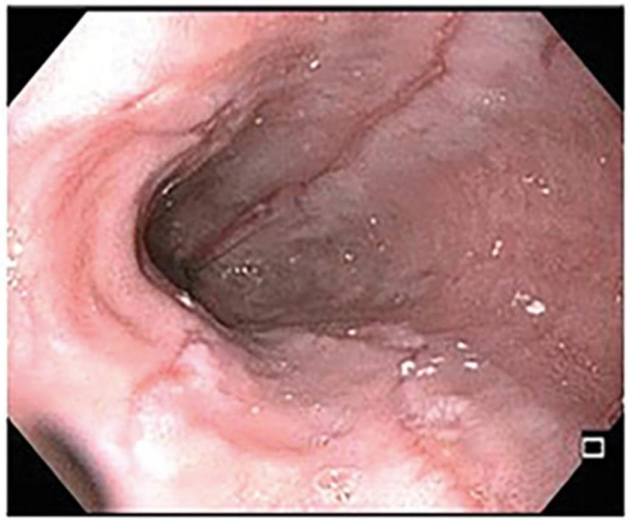
# Objective Testing

- *If troublesome heartburn, regurgitation, and/or non-cardiac chest pain do not respond adequately to a PPI trial*
- *Presence of alarm symptoms*
- Clinicians *should perform upfront objective reflux testing (rather than an empiric PPI trial) in patients with isolated extra-oesophageal symptoms and suspicion of reflux aetiology*
- **Patients with unproven GERD that have a symptom response to empiric PPI therapy, in order to establish the appropriateness of long-term PPI therapy**

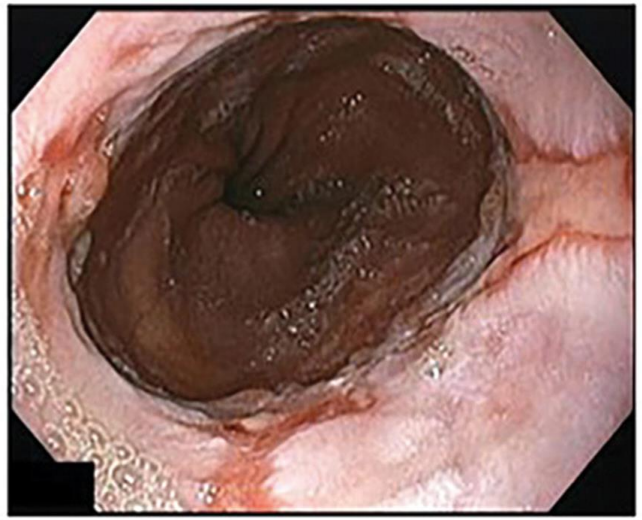




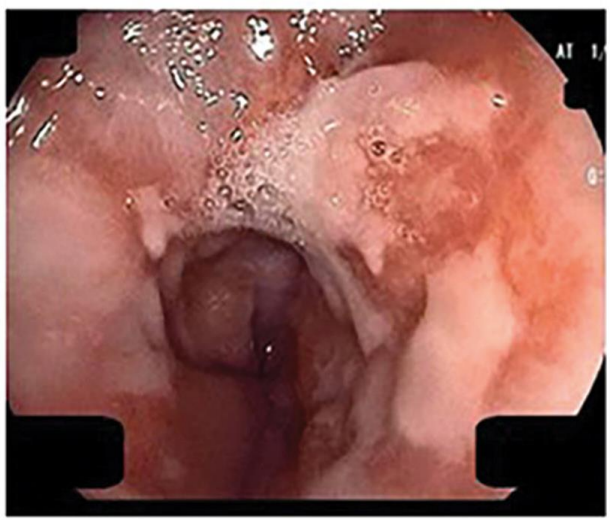
**LA-A**
≥1 mucosal break,



**LA-B**
≥1 mucosal break,



**LA-C**
≥1 mucosal break,

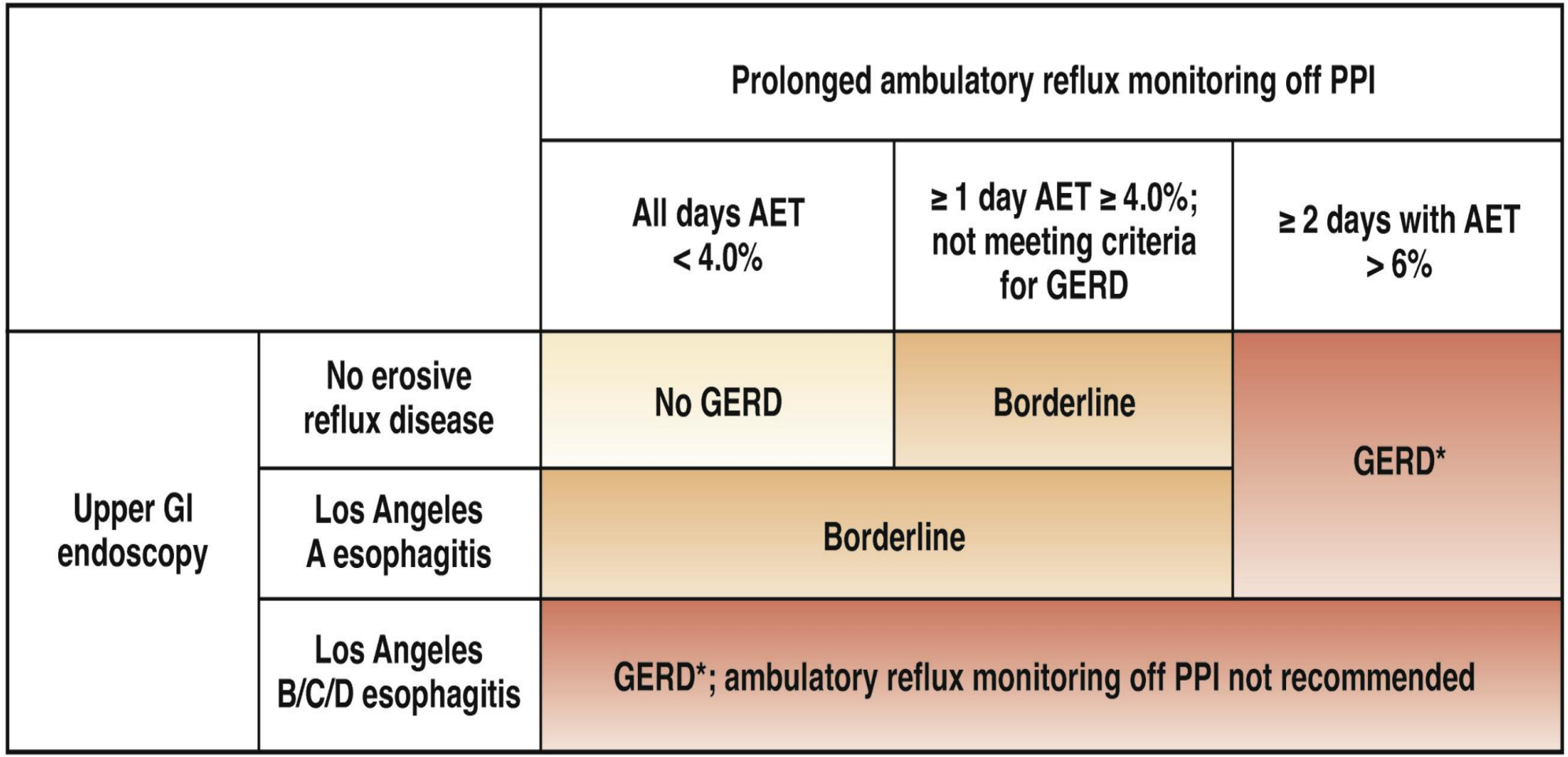


**LA-D**
≥1 mucosal break,

# Endoscopy: LA Classification

- Complete endoscopic evaluation of GERD
  - erosive esophagitis (graded according to the Los Angeles classification when present)
  - diaphragmatic hiatus (Hill grade of flap valve), axial hiatus hernia length,
  - inspection for Barrett's esophagus (with grading according to the Prague classification and biopsy when present) (BPA 7)
  - Confirmatory evidence of erosive reflux on endoscopy is found in a minority of patients

*Clinical Gastroenterology and Hepatology* 2024 222526-2528.e1DOI: (10.1016/j.cgh.2024.05.007)




*In a patient with GERD, the presence of Los Angeles C or D esophagitis, AET > 12.0%, DeMeester score > 50, bipositional reflux, and/or a large hiatal hernia indicates a more severe GERD phenotype

*Clinical Gastroenterology and Hepatology* 2022 20984-994.e1DOI: (10.1016/j.cgh.2022.01.025)
Copyright © 2022 AGA Institute Terms and Conditions

# pH Studies- wireless vs catheter

Mercy Health
Care first

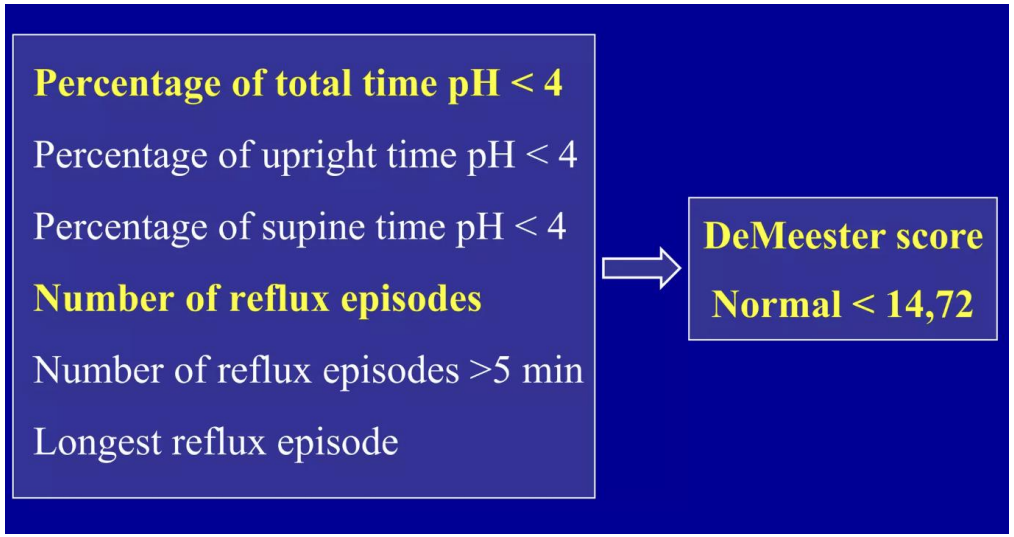
- Prolonged wireless pH-monitoring uses a pH capsule introduced via a trans-oral catheter during sedated gastroscopy that adheres to the distal oesophagus (6-cm proximal to the endoscopically identified squamocolumnar junction) using a vacuum suction mechanism

    - Wireless pH monitoring measures acid exposure in the distal oesophagus for up to 96 hours (based on recorder battery life) and assesses the relationship between patient reported symptoms and acid reflux episodes

- Catheter-based pH monitoring uses a trans-nasal catheter placed without sedation, which measures acid exposure in the distal oesophagus as well as reflux-symptom association for up to 24 hours

# pH study interpretation

- AET: Oesophageal acid exposure time (AET), the percent time spent at pH of 4.0 or less, is a key physiologic marker for phenotyping patients with GERD
  - Generally, an AET <4% is normal, 4–6% is borderline, and >6% is abnormal.
- SAP: Reflux symptom association on ambulatory reflux monitoring (symptom association probability >95% and symptom index >50%) increase confidence that symptoms are truly associated with reflux when AET is increased




DeMeester score components

# Refractory GERD and refractory symptoms

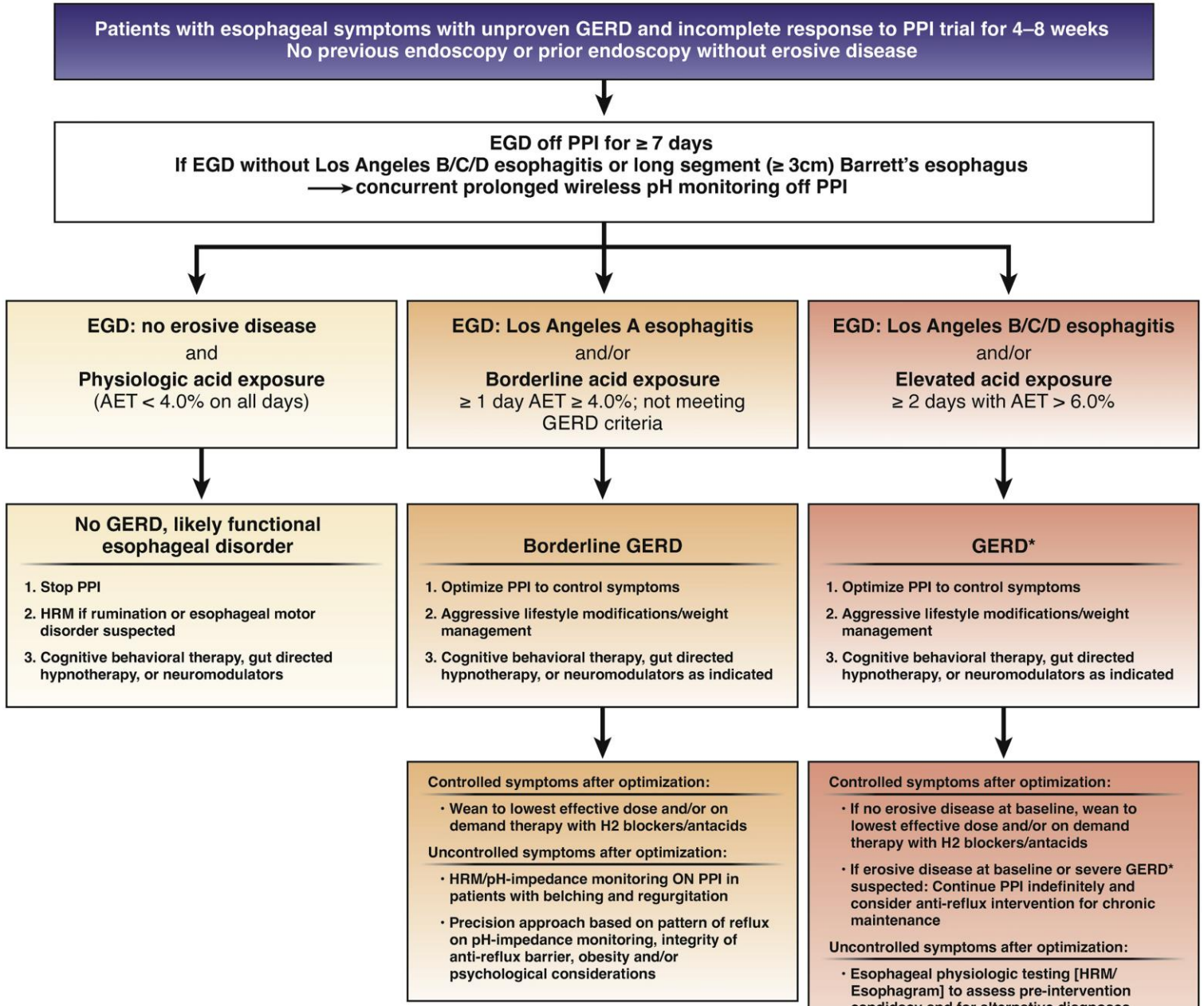
Mercy Health
Care first

- LA grade B, C and D oesophagitis and recurrent peptic stricture on endoscopy while on optimised antisecretory therapy are indicative of refractory GERD
  - Adjunctive pharmacotherapy and lifestyle measurements
  - Surgical management
    - Laproscopic fundoplication +/- HH repair
    - Roux-en-Y gastric bypass in obese patients

- Refractory Symptoms:
  - Consider alternative diagnoses
  - Clinicians should consider ambulatory 24-hour pH-impedance monitoring on PPI as an option to determine the mechanism of persisting oesophageal symptoms despite therapy

# Lifestyle and medication optimisation

- Dietry:
  - (1) avoidance of foods that may precipitate reflux (coffee, alcohol, chocolate, fatty foods),
  - (2) avoidance of acidic foods that may precipitate heartburn (citrus, carbonated drinks, spicy foods)
- Weight loss
- Elevation of head of bed- 20cm/ Avoid eating 2-3 hours before lying flat
- Optimizing PPI- +/- repeating pH studies while on optimized dose
- Adjunctive pharmacotherapy
  - H2RA
  - Alginate antacids for breakthrough symptoms- esp HH or nocturnal sx
  - Baclofen (for belch predominant or regurgitation)
  - Prokinetics only if concomitant gastroparesis
- Consideration of management of functional overlay
  - CBT, diaphragmatic breathing, hypnotherapy +/- pharmacologic neuromodulation
- Exclude missed diagnoses: Achalasia, EOE, alterations in gastric emptying, other functional disorders




GERD, gastroesophageal reflux disease; PPI, proton pump inhibitor; AET, acid exposure time; HRM, high resolution manometry.

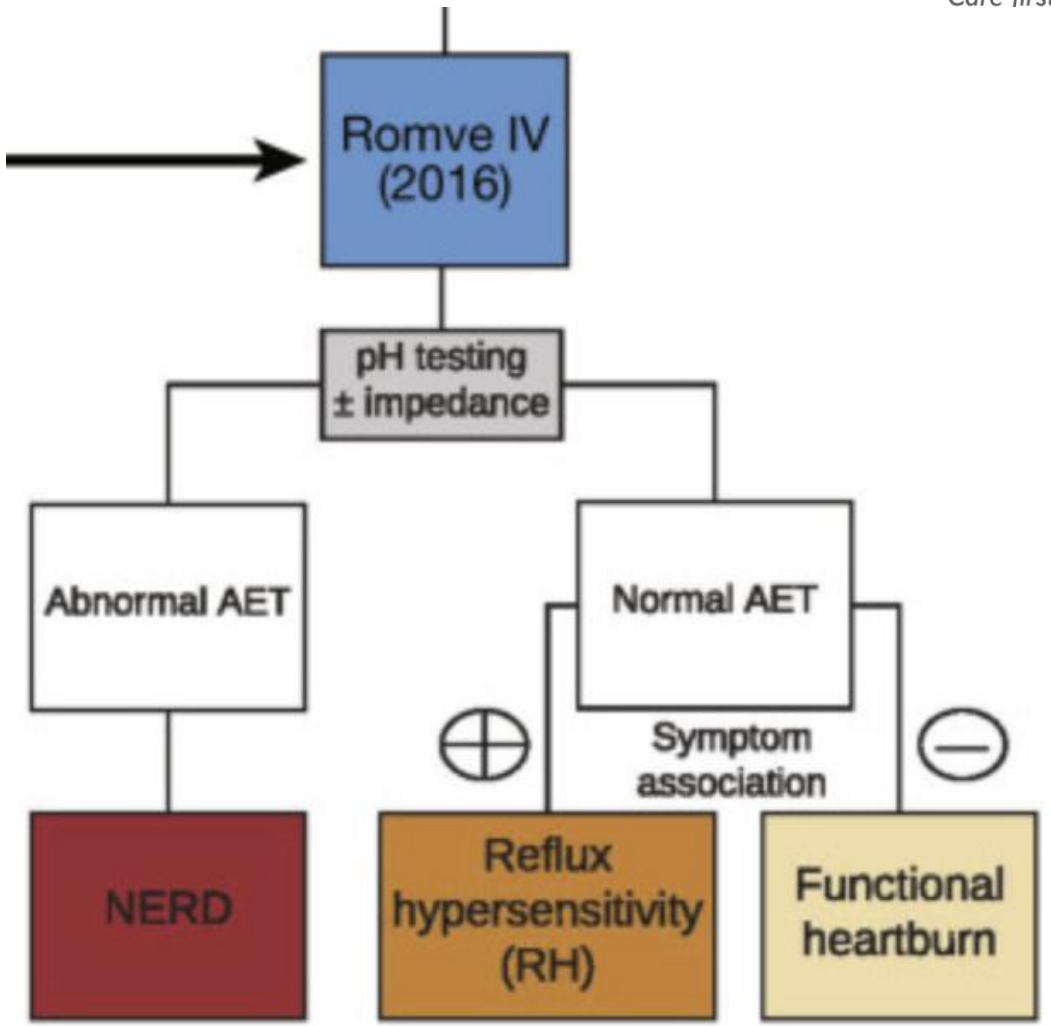
*The presence of Los Angeles C or D esophagitis, bipositional reflux, extreme levels of acid exposure (AET > 12.0% or DeMeester Score > 50) and/or a large hiatal hernia may indicate a more severe phenotype of GERD.

*Clinical Gastroenterology and Hepatology* 2022 20984-994.e1DOI: (10.1016/j.cgh.2022.01.025)
Copyright © 2022 AGA Institute Terms and Conditions

# Heartburn and normal endoscopy

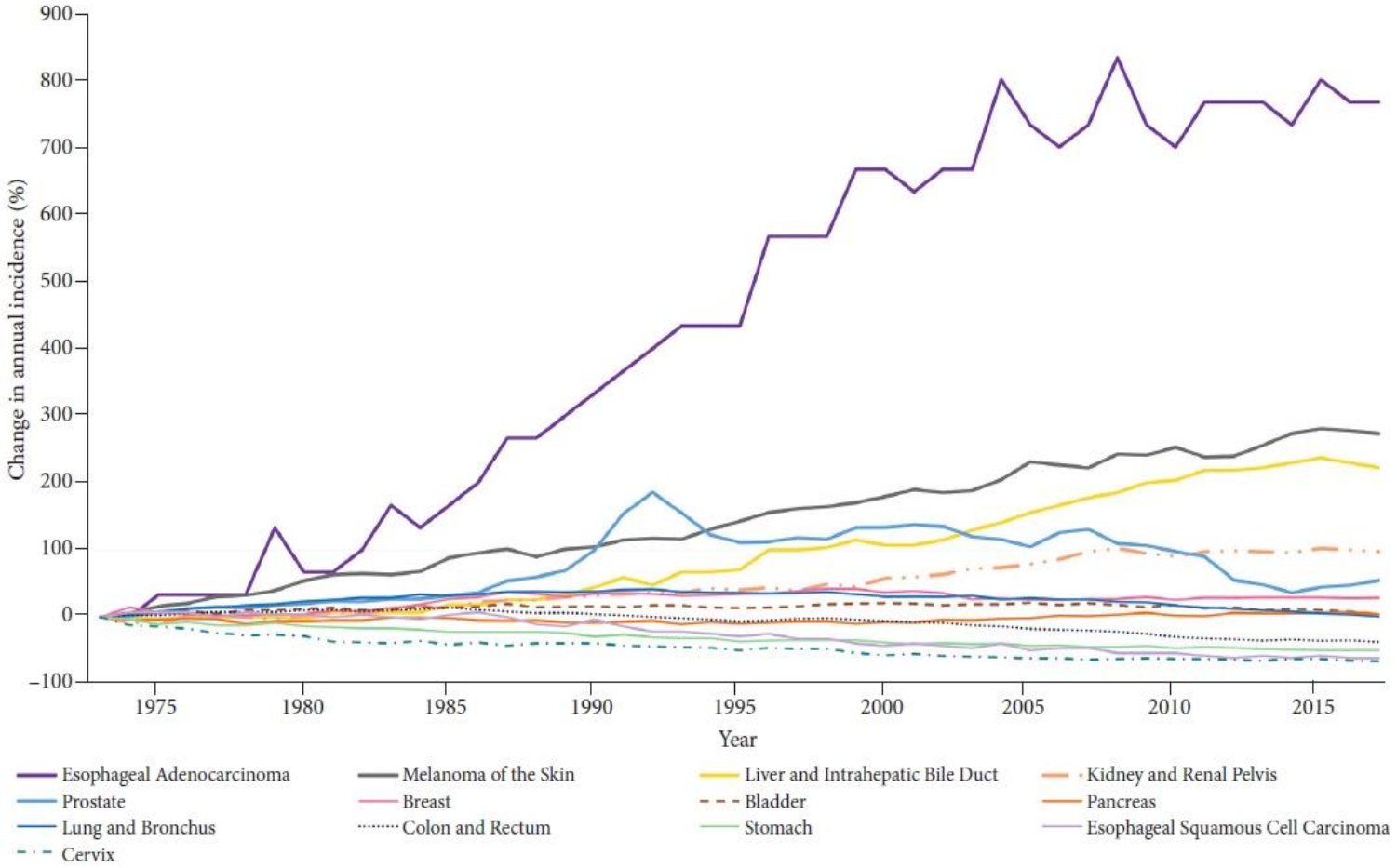
Troublesome Reflux symptoms with:

- Non-erosive reflux disease (NERD)- abnormal oesophageal acid exposure
- Reflux Hypersensitivity (RH)- Evidence of triggering of symptoms by reflux events despite normal acid exposure on pH- or pH-impedance monitoring
- Functional Heartburn (FH)- normal AET, no symptom association



# Barrett’s oesophagus

- Barrett’s esophagus (BE) is the only identifiable precursor lesion for EAC and characterized by replacement of squamous epithelium in the distal esophagus by metaplastic specialized columnar epithelium in response to chronic gastroesophageal reflux– induced injury and inflammation.



# Barrett’s Oesophagus

Mercy Health
Care first

- Progression of Barrret's oesophagus to adenocarcinoma is thought to develop in a stepwise manner; following the sequence of oesophagitis, metaplasia, dysplasia, and finally adenocarcinoma

- **Approximately 4% to 5% of patients with Barrett esophagus will be diagnosed with oesophageal adenocarcinoma in their lifetime**
  - people with Barrett oesophagus have approximately a **0.2% to 0.5% annual rate of developing oesophageal adenocarcinoma (AGA)**

- Data has demonstrated a 30% reduction in mortality for people who received endoscopic surveillance compared to those that did not (NICE)

# ESGE recommendations

**RECOMMENDATION 2**

**a** ESGE recommends against screening for BE in an unselected population.
Strong recommendation, low quality of evidence.

**b** ESGE suggests that case finding for BE could be considered in a select population, consisting of patients ≥50 years of age with a history of chronic GERD symptoms, and at least one of the following risk factors (white ethnicity, male sex, obesity, smoking, having a first-degree relative with BE or EAC).
Weak recommendation, low quality of evidence.

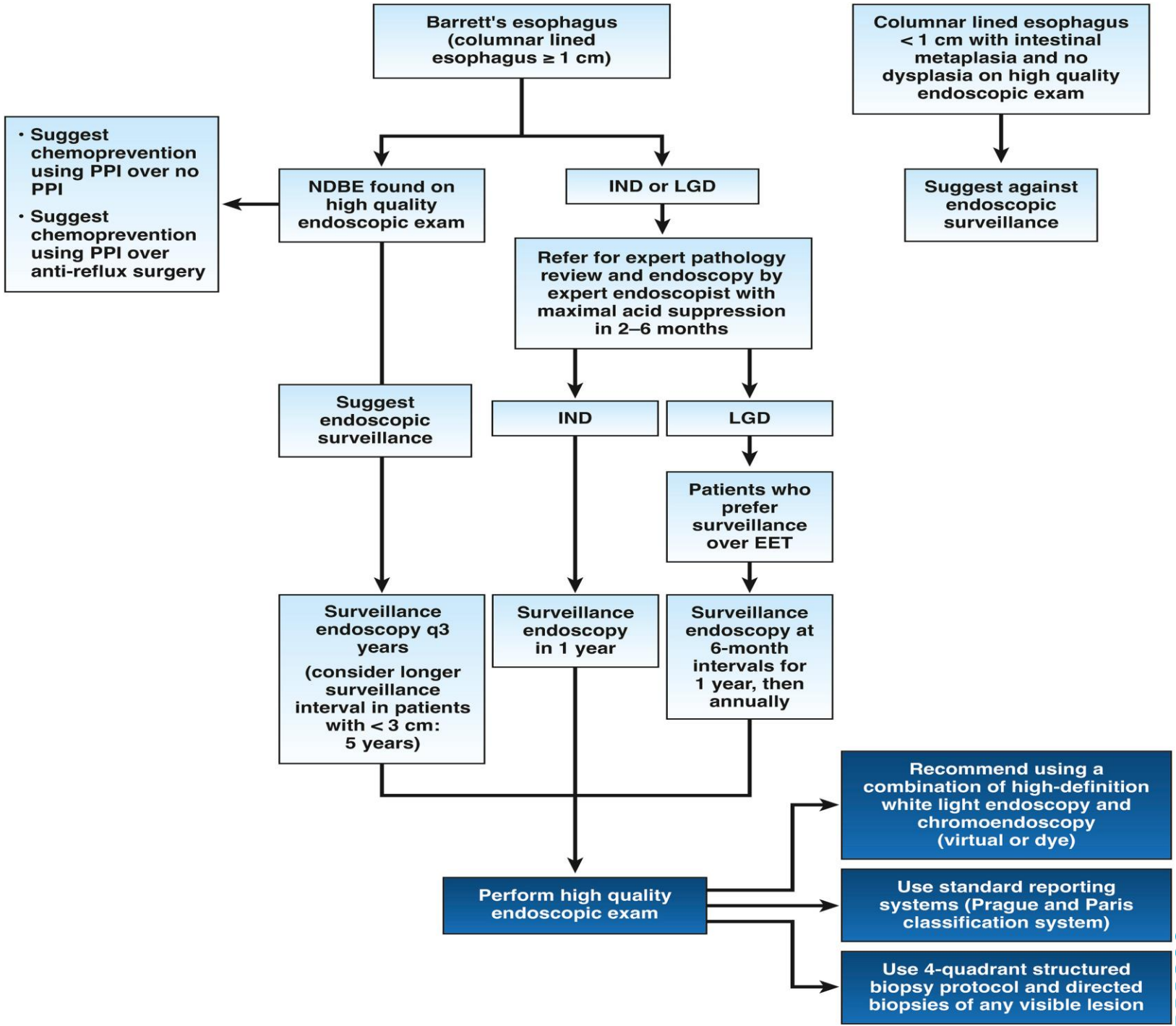
**RECOMMENDATION 1**

**a** ESGE suggests a proton pump inhibitor (standard dose* once daily) for chemoprevention in patients with BE.
Weak recommendation, moderate quality of evidence.

**b** ESGE recommends against the use of aspirin or non-steroidal anti-inflammatory drugs (NSAIDs) for chemoprevention.
Strong recommendation, moderate quality of evidence.

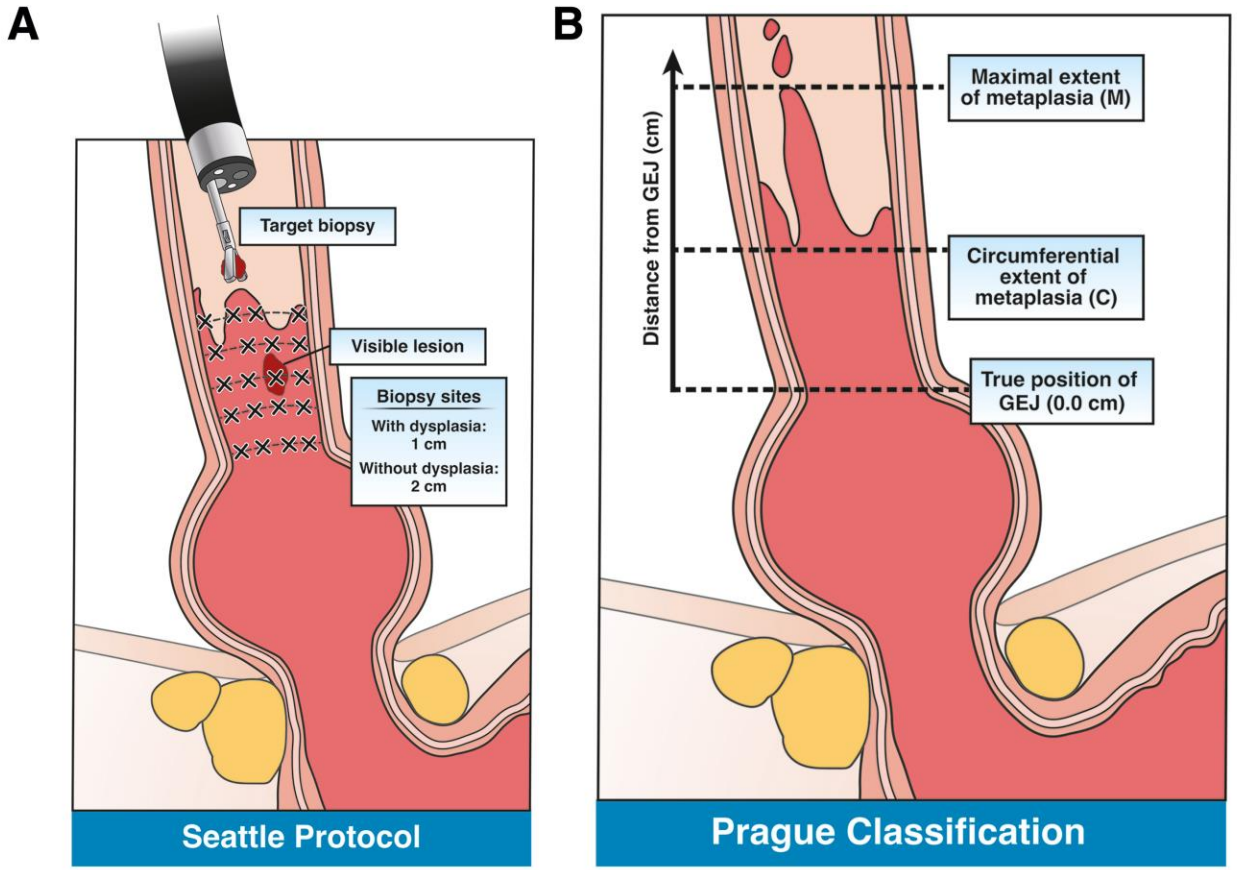
**RECOMMENDATION 10**

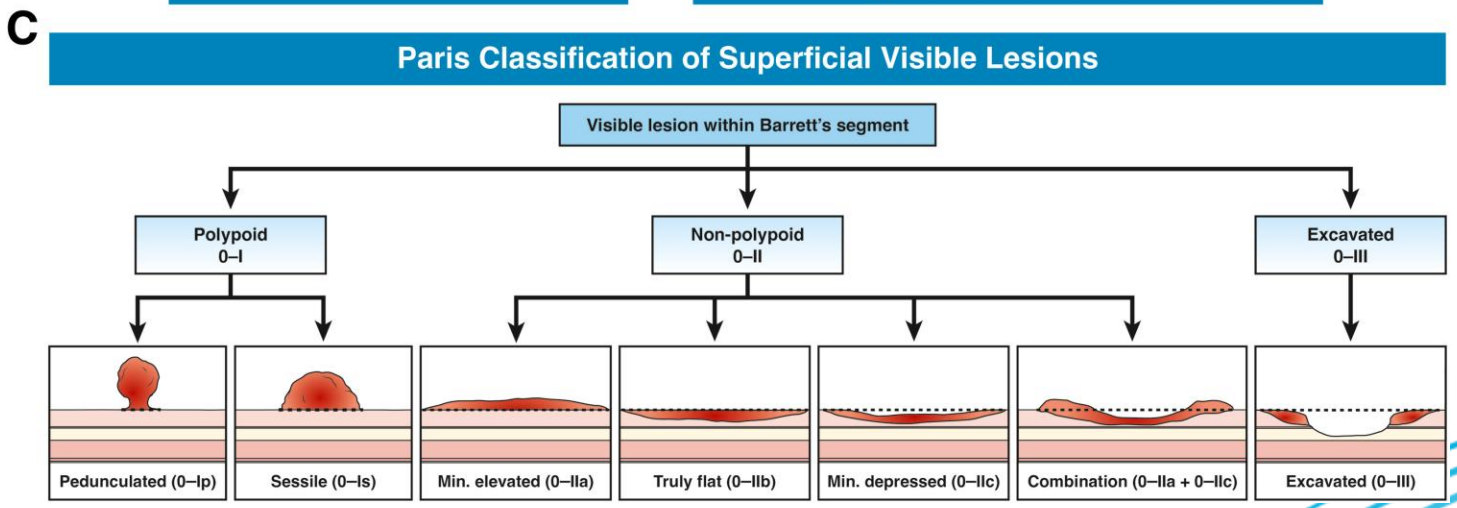
ESGE suggests that, if a patient has reached 75 years of age at the time of the last surveillance endoscopy and/or the patient's life expectancy is less than 5 years, the discontinuation of further surveillance endoscopies can be considered.
Weak recommendation, very low quality of evidence.




*Gastroenterology* 2025 1691184-1231DOI: (10.1053/j.gastro.2025.09.012)

Copyright © 2025 AGA Institute Terms and Conditions




*Gastroenterology* 2025 1691184-1231DOI: (10.1053/j.gastro.2025.09.012)
Copyright © 2025 AGA Institute Terms and Conditions

# If your patient has long segment, complicated or dysplastic Barrett's – they should be managed in a BE expert centre

Mercy Health
Care first

**RECOMMENDATION 9**

ESGE suggests varying surveillance intervals for different BE lengths. For BE with a maximum extent of ≥ 1 cm and < 3 cm, BE surveillance should be repeated every 5 years. For BE with a maximum extent of ≥ 3 cm and < 10 cm, the interval for endoscopic surveillance should be 3 years. Patients with BE with a maximum extent of ≥ 10 cm should be referred to a BE expert center for surveillance endoscopies.

For patients with an irregular Z-line/columnar-lined esophagus of < 1 cm, no routine biopsies or endoscopic surveillance are advised.

Weak recommendation, low quality of evidence.

- Access to HD endoscopes
- Expertise in optical diagnosis
- Access to expert GI pathology and independent review
- Discussion at MDM to confirm dysplasia and degree of dysplasia
- Access to endoscopic management
- Access to expert upper GI surgery

# Endoscopic Therapy

- HGD and IMC- ER preferred over survey (up to T1b SM1 in high-risk patients, well diff, no poor prognostic factors)

- T1b SM 2/3- ER not curative

- Resect all visible abnormalities
  - EMR vs ESD
  - CT and PET CT often prior to ER if suspicion of SM invasion (not in guidelines)
  - EUS no longer used

- No VSM with HGD/IMC- RFA

- For all lesions- EET (endoscopic eradication therapy- 3/12 scopes until Barrett's is gone), then 6/12 for 12/12, then annual

# PPI's: Which to use

- More than 15 million PPI prescriptions were dispensed through the Australian Pharmaceutical Benefits Scheme in 2020–21
- The Gastroenterological Society of Australia and The Royal Australian College of General Practitioners recommend regular review of long-term PPI use with the goal of dose reduction and cessation

**Box 1. Recommendations for long-term (>8 weeks) and short-term (≤8 weeks) proton pump inhibitor treatment[11]**

Long-term use (>8 weeks)

- Barrett's oesophagus
- Zollinger–Ellison syndrome
- Clinically significant severe erosive oesophagitis (LA grade C/D)
- Peptic strictures
- Oesophageal scleroderma
- Eosinophilic oesophagitis
- Gastroprotection from NSAIDs or aspirin in patients at high risk of GI bleed*
- Prevention of pulmonary fibrosis progression

Short-term indications (≤8 weeks)

- *Helicobacter pylori* eradication
- Treatment of aspirin or NSAID-related gastric and duodenal ulcers in patients at low risk
- GORD
- Dyspepsia

**AGA guidelines consider patients to be high risk if age >60 years, severe medical comorbidity, using multiple NSAIDs or aspirin, taking and antithrombotic or oral corticosteroid.*

*AGA, American Gastroenterological Association; GI, gastrointestinal; GORD, gastro-oesophageal reflux disease; LA, Los Angeles classification of gastro-oesophageal reflux disease; NSAID, nonsteroidal anti-inflammatory drug*

# Choice of PPI

- Ease of access
- Cost
- Potency

**Table 1.** Potency of PPIs Based on OE

| Drug at lowest available dosage | OE |
|---|---|
| Pantoprazole 20 mg | 4.5 mg |
| Lansoprazole 15 mg | 13.5 mg |
| Omeprazole 20 mg | 20 mg |
| Esomeprazole 20 mg | 32 mg |
| Rabeprazole 20 mg | 36 mg |

**Box 2. Proton pump inhibitor (PPI) availability**

Initial therapy, high-dose PPIs:

- Esomeprazole 20 mg, 40 mg
- Lansoprazole 30 mg
- Omeprazole 20 mg
- Pantoprazole 40 mg
- Rabeprazole 20 mg

Maintenance therapy, low-dose PPIs:

- Lansoprazole 15 mg
- Omeprazole 10 mg
- Pantoprazole 20 mg
- Rabeprazole 10 mg

# Media Reports

- Short term: headache, nausea, vomiting, diarrhoea, abdominal pain, constipation and flatulence
- Increased risk acute interstitial nephritis
- Possible increase in GI infection (C diff)
- Hypomagnemesia

- Consider indication for ongoing PPI use in patients with newly diagnosed vitamin B12 deficiency, enteric infections or fractures

# Tapering PPIs

Abrupt PPI discontinuation may result in short-term rebound acid hypersecretion that can mimic symptom return

Consider gradual dose tapering prior to discontinuation followed by switching to PRN treatment and other OTC remedies or H2RA

Mercy Health
Care first

# Questions?

# Chronic Liver Disease

Updates in the Assessment and Management
of Cirrhosis and Portal Hypertension

Dr NJ Arachchi
Gastroenterologist

Werribee Mercy Hospital and Western Health

GP Education 2026

# Learning Outcomes

1

## Diagnosis and Risk stratification

Who to screen and identifying the aetiology
Non-Invasive Fibrosis Assessment  - Identify cirrhosis risk using FIB-4 and elastography

2

## Managing the Risk and minimising the progression

Understand indications and protocols for HCC screening

3

## Nutritional and Pharmacological Management

Apply evidence-based strategies for cirrhosis and portal hypertension

4

## Referral and Palliative Pathways

Recognise when to refer and manage end-stage liver disease symptoms

# Meet the 56-year-old male with vague tiredness and abnormal LFTs

**Presentation:** RUQ pain, vague tiredness. No peripheral signs of cirrhosis.

**History:** Fasting hyperglycaemia (no frank diabetes). Moderate alcohol intake (2-3 standard drinks on weekends).

**Vitals:** Weight 92kg, BMI 29.

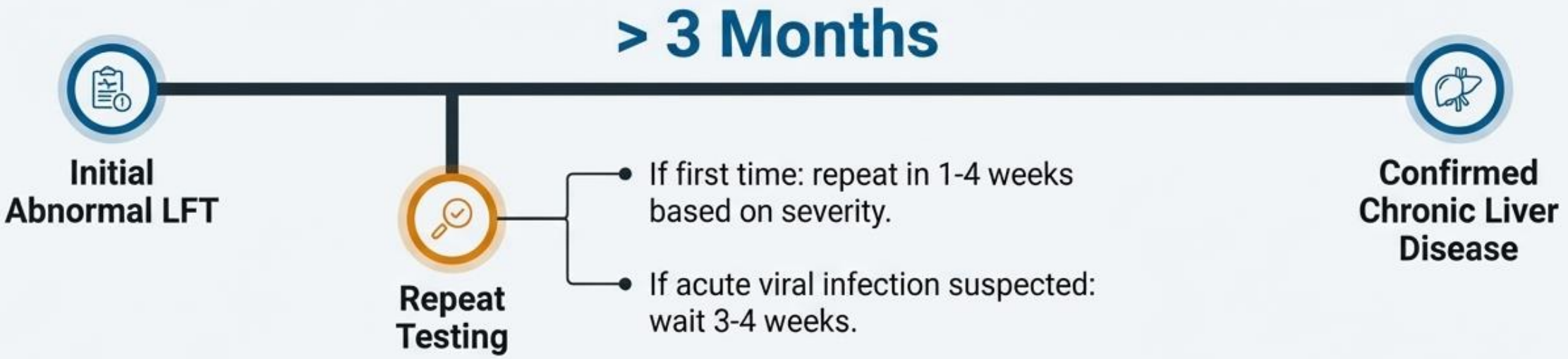
**Pathology Report**

**Lab Results:**
Bilirubin: 7
**ALT: 88**
**AST: 95**
ALP: 140
**GGT: 200**
Albumin: 41
Platelets: Normal

**Context:** Previous records show similarly abnormal LFTs over the last 12 months.

NotebookLM

# Defining the baseline for chronic liver disease




- The Diagnostic Threshold: Chronic liver disease (CLD) is defined by persistently abnormal liver function tests (LFTs) for more than 3 months.
- The purpose of this timeline is to distinguish true chronic conditions from transient acute liver injuries.

NotebookLM

# What is next?

Diagnosis – Chronic liver disease

# Burden of Chronic Liver Disease in Australia

PREVALENCE

## 6.1M+

Australians living with liver disease (2012), projected to exceed 8 million by 2030

ECONOMIC IMPACT

## $50.7B

Total socio-economic impact when accounting for burden of disease (2012)

MORTALITY

## 7,200+

Deaths from liver disease in Australia per year (2012)

# Who to Screen: At-Risk Groups

## Lifestyle and Infections

Excessive alcohol consumption
Hepatitis B/C risk factors (IVDU, tattoos in non-commercial settings, born in endemic countries, pre-screening transfusion)

## Metabolic Dysfunction

Obesity, diabetes, dyslipidaemia, hypertension, sleep apnoea

## Medications

Hepatotoxic drugs: amiodarone, steroids, methotrexate

## Autoimmune and Inflammatory

Inflammatory bowel disease
Autoimmune conditions
Coeliac disease
Endocrinopathies

## Hereditary Conditions

Family history of Wilson disease, haemochromatosis, alpha-1 antitrypsin deficiency

## Symptoms and Signs

Fatigue, nausea, RUQ pain, pruritus, jaundice
Hepatomegaly, splenomegaly, ascites, spider naevi, palmar erythema

# Alcohol: Current NHMRC Guidelines (2020)

## Recommended Limits

No more than 10 standard drinks per week
No more than 4 standard drinks on any one day
1 Australian standard drink = 10 g pure ethanol

## WHO Position (2023)

No level of alcohol consumption is safe for health at the population level

## Key Change from 2009

Reduced from 14 to 10 drinks/week due to stronger evidence on alcohol and cancer risk (breast, oropharyngeal, oesophageal, colorectal)

## Liver-Specific Risk

Risk is exponential: RR ~3 at 20 units/week, RR ~30 at 80 units/week
Obesity (BMI >35) doubles the liver disease risk at any alcohol intake

# Management Steps

| | | |
|---|---|---|
| Aetiology | Staging of the fibrosis | When to refer |
| Manging patient who do not need referral to tertiary services | Managing patient who are already under the care of teriary services | Palliative care in End stage liver disease patients |

# Investigations for aetiology: The Liver Screen

## Viral Hepatitis

HBsAg, anti-HBs, anti-HBc
Hep C antibody (HCV RNA if positive)
Hep A IgG (immunity/vaccination)
Hep D if HBsAg positive
(CMV EBV HSV – do not cause chronic hepatitis

## Autoimmune Markers

ANA, AMA, anti-smooth muscle antibodies
Serum immunoglobulins (IgG, IgA, IgM)
Anti-LKM1 only in younger patients

## Metabolic Markers

HbA1c, fasting glucose, lipids
Ferritin + transferrin saturation
Caeruloplasmin (Wilson, age <40)
Alpha-1 antitrypsin level

## Other Investigations

Coeliac serology (tTG-IgA)

Liver ultrasound: steatosis, structural lesions,

HIV if risk factors

# Natural History: Progression of Liver Disease

1

## No Fibrosis (F0)

Normal liver architecture. Assess and manage risk factors.

2

## Mild-Moderate Fibrosis (F1-F2)

Slow progression. MASLD: ~1 stage per 14 years (simple steatosis), ~7 years (MASH).

3

## Bridging Fibrosis (F3)

~20% progress to cirrhosis within 2 years (the 20% Rule).

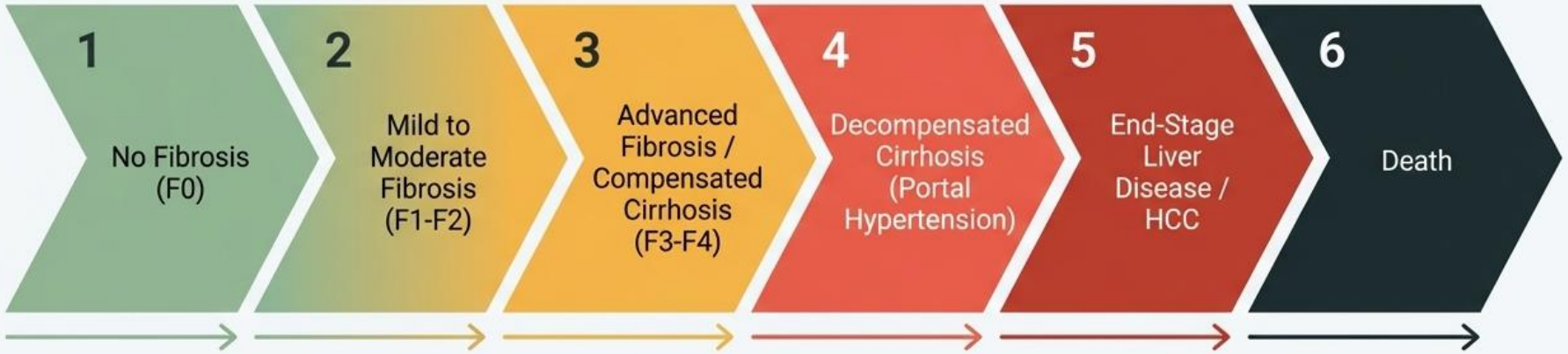
4

## Cirrhosis (F4) and Beyond

~20% with compensated cirrhosis decompensate within 2 years. HCC risk 1-2.6%/year. Median survival 2 years from first decompensation without transplant.

# The natural progression from steatosis to liver failure

The clinical trajectory follows a predictable decline if interventions are not made:




NotebookLM

# MASLD Natural History: Key Numbers

Fibrosis stage is the strongest predictor of disease-specific mortality, not the presence of steatohepatitis. CVD remains the leading cause of death across all MASLD stages.

### Simple Steatosis

1 stage per 14 years. ~31% develop MASH over 4.7 years. Overall ~0.5% progress to cirrhosis.

### MASH

1 stage per 7 years. 20-30% develop cirrhosis over 10-20 years.

### MASH + DM + Alcohol

Accelerated: 1 stage per 4 years. DM doubles HCC risk. Alcohol synergises with insulin resistance.

# The "20% Rule" of MASH Progression

**RAPID PROGRESSORS**

## ~21%

of F0-F1 patients develop F3-F4 over approximately 5.9 years

**F3 TO CIRRHOSIS**

## ~20%

of patients with bridging fibrosis progress to cirrhosis within just 2 years

**CIRRHOSIS TO DECOMPENSATION**

## ~20%

of patients with compensated cirrhosis decompensate within 2 years

# The 20% rule dictates rapid progression to cirrhosis


For patients who have reached the bridging fibrosis stage (F3), the clock accelerates significantly.

This narrow 2-year window represents a critical opportunity for aggressive **primary care before irreversible structural damage occurs**.

NotebookLM

# The second 20% rule dictates rapid decompensation


**Once compensated cirrhosis is established, clinical instability is imminent for many.**

**Overall risk:** Approximately 45% of patients with MASH cirrhosis will develop decompensated cirrhosis within 10 years (roughly 10% risk per year).

NotebookLM

# Returning to our patient to map the progression timeline

**The 56yo Male:**

- Has risk factors (alcohol, metabolic features) and abnormal LFTs indicating steatohepatitis.
- Baseline risk: ~15% risk of progressing to cirrhosis in 5 years.
- 10% risk of decompensation in the next decade.

**The GP Dilemma:** How do we determine where he currently sits on the fibrosis timeline without resorting to an invasive liver biopsy?

**Step 1 - First Pass Investigations:**

- General/Specific Bloods: Viral serology, iron studies, metabolic markers, celiac serology.
- Ultrasound: Rule out biliary obstruction/tumours; look for steatosis, irregular margins, splenomegaly, or ascites.

NotebookLM

# METAVIR Fibrosis Staging

F 0

## No Fibrosis

Normal liver architecture, organised hepatocyte plates, no excess collagen

F 1

## Portal Fibrosis

Early scarring confined to portal areas, no septa extending into the lobule

F 2

## Periportal Fibrosis

Fibrous septa radiate into parenchyma, lobular architecture remains intact

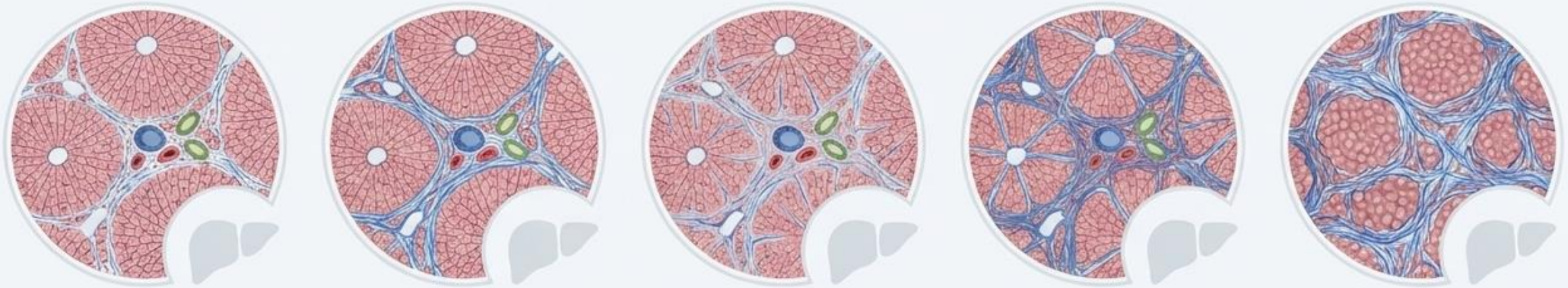
F 3

## Bridging Fibrosis

Septa bridge portal tracts to each other or central veins, architecture distorted

# Visualising histological damage using the METAVIR score

Understanding the target for our non-invasive tests:



**F0: No Fibrosis**

(Normal liver architecture)

**F1: Portal Fibrosis**

(Fibrosis confined to portal tracts)

**F2: Periportal Fibrosis**

(Extension of fibrosis beyond portal tracts)

**F3: Bridging Fibrosis**

(Fibrous septa connecting portal tracts; the threshold for the 20% rapid progression rule)

**F4: Cirrhosis**

(Widespread nodular formation and structural collapse)

NotebookLM

# Non invasive tests (NITs) for assessing Fibrosis

FIB-4

Fibroscan

Ultasround based Shearwave elastography

May not need these if u/s liver clearly showed cirrhosis –such as nodular liver ascites and splenomegaly

# FIB-4 is the ultimate primary care triage tool


**What it is:** A validated, non-invasive index used to estimate the risk of advanced hepatic fibrosis and cirrhosis.

**Why it matters:** It replaces the need for immediate biopsy and acts as a highly accurate negative predictor to safely rule out advanced disease in the community. Requires only standard, inexpensive pathology results (Age, AST, ALT, Platelets).

NotebookLM

# FIB-4 Index: The First-Line Screening Tool

FIB-4 = (Age x AST) / (Platelets x square root of ALT). Uses 4 routine variables.

Recommended first-line NIT by BSG, EASL, AASLD, NICE, and GESA.

### Low Risk: FIB-4 <1.3

NPV ~90% for advanced fibrosis. Manage in primary care. Repeat 2-3 years (1-2 years if T2DM or metabolic risk factors).

### Indeterminate: 1.3-2.67

Proceed to second-line test: FibroScan, shear wave elastography

### High Risk: 2.67 or above

PPV ~80% for advanced fibrosis. Refer to hepatology/gastroenterology.

# FibroScan vs Shear Wave Elastography

## FibroScan (VCTE)

Dedicated standalone device
Most extensively validated worldwide
Includes CAP for steatosis assessment
Higher failure rate in obesity (M probe ~20% if BMI >30; XL probe mitigates)
Low operator dependence

## Ultrasound-Based SWE (2D-SWE/pSWE)

Integrated into conventional US machines
Real-time B-mode imaging guides ROI placement
Lower failure rate in obesity
Higher operator dependence
Cut-offs are system-specific

## Accuracy (Cassinotto 2016)

AUROC: 0.82 (F2+), 0.86 (F3+), 0.90 (F4)

## Accuracy (Cassinotto 2016)

2D-SWE AUROC: 0.88 (F2+), 0.93 (F3+), 0.93 (F4)
pSWE AUROC: 0.77 (F2+), 0.84 (F3+), 0.84 (F4)

# Elastography: Pitfalls and Confounders

These confounders affect BOTH FibroScan and US-based SWE. Always check before interpreting liver stiffness measurements.

### Food Intake

Increases LSM by 20-25%. Mandatory fasting of 2-3 hours or more before any measurement.

### Hepatic Inflammation / ALT Flare

ALT >5x ULN significantly overestimates fibrosis. Repeat when inflammation settles.

### Hepatic Congestion

Right heart failure, Budd-Chiari raise LSM independent of fibrosis. Check JVP, cardiac history.

# The Two-Step Fibrosis Assessment Pathway

1

## Step 1: Calculate FIB-4 (Primary Care)

FIB-4 <1.3 (or <2.0 if age 65+): Low risk. Manage metabolic risk factors. Repeat in 2-3 years.
FIB-4 1.3 or above: Proceed to Step 2.

2

## Step 2: Elastography

LSM <8 kPa: Low risk of advanced fibrosis. Manage in primary care.
LSM 8-12 kPa: Indeterminate. Consider specialist referral.
LSM 12-15 kPa or above: Probable advanced fibrosis. Refer to specialist.

3

## Outcome

This sequential approach reduces the indeterminate zone to approximately 5% of patients and reduces unnecessary referrals by up to 80%.

# When to Refer to Specialist

## High Fibrosis Risk

FIB-4 above 2.67
Elevated elastography (LSM 8 kPa+)
Indeterminate FIB-4 without access to second-line tests

## Evidence of Cirrhosis

Nodular liver, splenomegaly, ascites on imaging
Falling platelet count with splenomegaly
LSM above 12-15 kPa

## Decompensation Events

Ascites (clinical or imaging)
Clinical jaundice
Variceal bleeding
Hepatic encephalopathy

## Specific Aetiologies

Chronic Hep B or C (if not comfortable managing)
Wilson disease, haemochromatosis
Autoimmune hepatitis
Persistently unexplained abnormal LFTs

# Managing Non-Cirrhotic CLD in the Community

# When to Repeat FIB-4 and FibroScan

Monitoring Intervals for Non-Cirrhotic CLD
and Compensated Cirrhosis Without CSPH

# Non-Cirrhotic Chronic Liver Disease

## FIB-4 Repeat Intervals

FIB-4 <1.3 (no metabolic risk): every 3 years

FIB-4 <1.3 + DM or ≥2 metabolic risk factors: every 1-2 years

FIB-4 1.3-2.67: do not repeat; proceed to FibroScan/SWE

FIB-4 ≥2.67: refer to specialist

## Key Point

FIB-4 is a screening/triage tool, not a standalone diagnostic test. Sequential two-step approach (FIB-4 then elastography) reduces unnecessary referrals by up to 80%.

## FibroScan Repeat Intervals

LSM <8 kPa (advanced fibrosis excluded): repeat in 1-2 years

Confirmed F1-F2 on elastography: repeat annually

LSM 8-12 kPa (indeterminate): refer or repeat in 6-12 months

LSM ≥12 kPa: specialist referral

Hazardous alcohol use, no cirrhosis: every 2 years

~12% of patients show worsening LSM after 3 years, supporting periodic reassessment even in low-risk patients.

# Compensated Cirrhosis Without CSPH: Monitoring

CSPH excluded when LSM <15 kPa AND platelets >150 × $10^9$/L (NPV >90%). Endoscopy can be safely avoided in these patients. Annual monitoring is essential to detect progression.

### Annual LSM + Platelets

Repeat FibroScan and platelet count every 12 months. If both remain stable, patient stays in "rule out CSPH" category. No endoscopy needed.

### LSM Rises ≥20% or Reaches ≥20 kPa

"Clinically significant LSM increase" per Baveno VII. Triggers urgent re-evaluation, consider endoscopy for varices, and reassess management.

### Platelets Drop <150 × $10^9$/L

Patient no longer meets Baveno VII rule-out criteria. Re-stratify risk and consider screening endoscopy for varices.

# Managing Non-Cirrhotic CLD in the Community

## When to Repeat FIB-4

## When to Repeat Elastography

## Vaccinations – to protect the liver

Hep A if IgG is negative
Hep B - If Hb sAg , sAb and core Ab negative

## Lab Monitoring

LFTs, FBE (platelets), UEC: Every 6-12 months
FIB-4 calculated: Per risk-stratified interval
HbA1c/glucose: 3-6 monthly if diabetic
Lipid profile: Annually

## Weight Loss Targets (MASLD)

5% or more: Reduces steatosis
7% or more: Can resolve MASH
10% or more: May regress fibrosis
Mediterranean diet preferred, 150 min/week exercise

## Alcohol

Follow NHMRC guidelines- ? Perhaps less than that

# Managing Cirrhosis without clinically significant portal hypertension in the Community

## When to Repeat Elastography

FIB-4 is less usefull

## Lab Monitoring

LFTs, FBE (platelets), UEC: Every 6- months
Ultrasound scan and AFP every 6 moths for HCC surveillance
HbA1c/glucose: 3-6 monthly if diabetic
Lipid profile: Annually

## Vaccinations – to protect the liver

Hep A if IgG is negative
Hep B - If Hb sAg , sAb and core Ab negative
Annual vaccinations

## Aggressively Manage the underlying aetiology

5% or more: Reduces steatosis
7% or more: Can resolve MASH
10% or more: May regress fibrosis
Dietician review

## Alcohol

# Managing patients with clinically significant portal hypertension

In collaboration with specialist service

# Portal Hypertension: Non-Invasive Assessment (Baveno VII)

CSPH is defined as HVPG of 10 mmHg or above. Baveno VII (2022) established validated non-invasive criteria using liver stiffness measurement and platelet count to rule in or rule out CSPH.

### Rule OUT CSPH

LSM <15 kPa AND platelets >150. Sensitivity and NPV above 90%. No endoscopy needed.

### Rule IN CSPH

LSM 25 kPa or above. Specificity and PPV above 90%. Start treatment (NSBB).

### Grey Zone

LSM 15-25 kPa or platelets 150 or below. Consider Gastroscopy; Consider spleen stiffness or early, regular repeat elastography.

# Beta-Blockers for CSPH: The Paradigm Shift

PREDESCI trial (Villanueva et al, Lancet 2019): NSBBs reduced ALL forms of decompensation (not just variceal bleeding) in compensated cirrhosis with CSPH. HR 0.51, 3-year decompensation 16% vs 27% placebo.

### New Paradigm

Treat CSPH, not just varices. Start NSBBs at the point of CSPH, even without varices. If NSBB started, endoscopy can be avoided (AASLD 2023).

### Carvedilol Preferred

Dual mechanism: beta-blockade (reduces cardiac output and splanchnic flow) PLUS alpha-1 blockade (reduces intrahepatic resistance). Greater HVPG reduction than propranolol.

### Dosing

Start 3.125-6.25 mg daily. Uptitrate to 12.5 mg daily (maximum). Maintain HR >55 bpm, SBP >90 mmHg.

# Varices Screening Strategy

01

## No CSPH (Baveno VII Rule-Out)

LSM <15 kPa + Plts >150: No endoscopy needed. Repeat LSM and platelets annually.

02

## CSPH Present, NSBB Started

Endoscopy can be safely avoided per AASLD 2023. Monitor with annual elastography.

03

## Varices Found on OGD

No/small varices: Repeat OGD in 2-3 years. Medium/large varices: Primary prophylaxis with carvedilol or EVL.

# General Management of Compensated Cirrhosis

## HCC Surveillance

6-monthly USS with or without AFP for ALL patients with cirrhosis. In chronic Hep B: surveillance even without cirrhosis (age-based). FIB-4 above 3.25: annual HCC incidence exceeds 1%.

## Bone Health

DEXA scan at diagnosis of cirrhosis. Calcium 1000-1200 mg/day + Vitamin D supplementation. Bisphosphonates if T-score <-2.5. Repeat DEXA every 1-3 years based on result.

## Nutrition

35 kcal/kg/day or more. Protein 1.2-1.5 g/kg/day (NEVER restrict). Late evening snack (50 g carbohydrate). Screen for sarcopenia. Mediterranean diet.

## Vaccinations

Hepatitis A and B (if non-immune). Annual influenza. Pneumococcal (PCV13/PCV20 + PPSV23). COVID-19 per ATAGI. Shingrix if age 50 or above. Vaccinate early: response attenuated in advanced disease.

# Monitoring Schedule: Compensated Cirrhosis Without CSPH

## Investigations

LFTs, FBE (platelets), UEC, INR: Every 6 months
FibroScan/Elastography: Annually
HCC surveillance (USS with or without AFP): Every 6 months
DEXA scan: At diagnosis, then 1-3 yearly

## Baveno VII Monitoring

If LSM <15 kPa + Plts >150: Repeat annually
If LSM rises 20% or more AND reaches 20 kPa or above: Re-evaluate urgently, consider endoscopy
If LSM decreases 20% or more: Favourable; continue annual monitoring

## General Measures

Vitamin D level: At diagnosis, then annually
OGD: Every 2-3 years if no varices (when Baveno criteria not met)
Nutritional screening: 6-12 monthly
HbA1c/metabolic profile: 6-12 monthly

## Medications to Note

Statins: SAFE in compensated cirrhosis; recommended for CV risk
Avoid NSAIDs, ACE inhibitors/ARBs in decompensation
Paracetamol: Safe at 2 g/day or less
Avoid hepatotoxic medications and herbal supplements

# What Is Decompensation?

Decompensation marks a dramatic change in prognosis. Median survival from first decompensation to death or transplant is just 2 years. These patients need urgent specialist referral.

### 1. Ascites

Most common first event (69.5% of first decompensations). Clinical or on imaging.

### 2. Clinical Jaundice

Indicates significant hepatocellular dysfunction.

### 3. Variceal Bleeding

First variceal bleed carries 15-20% 6-week mortality.

### 4. Encephalopathy

# Child-Pugh Score: Staging & Prognosis

## Scoring Components (5 Variables)

Each scored 1-3 points (total 5-15):

Total Bilirubin (µmol/L)
1pt: <34 | 2pt: 34-50 | 3pt: >50

Albumin (g/L)
1pt: >35 | 2pt: 28-35 | 3pt: <28

INR (or PT prolongation)
1pt: <1.7 | 2pt: 1.7-2.3 | 3pt: >2.3

Ascites
1pt: None | 2pt: Mild/controlled | 3pt: Moderate-severe

Encephalopathy
1pt: None | 2pt: Grade I-II | 3pt: Grade III-IV

## Classification & Survival

Class A (5-6 points)
- Good hepatic reserve
- 1-year survival: ~100%
- 2-year survival: ~85%
- Compensated cirrhosis

Class B (7-9 points)
- Significant functional compromise
- 1-year survival: ~80%
- 2-year survival: ~60%
- Consider transplant referral

Class C (10-15 points)
- Decompensated cirrhosis
- 1-year survival: ~45%
- 2-year survival: ~35%
- Urgent transplant assessment

## Key Limitations

- Subjective components (ascites, HE grading)
- Does not account for renal function
- Ceiling effect in severe disease
- Not used for transplant allocation (MELD preferred)

# MELD-Na Score: Prognosis & Transplant Referral

## What Is MELD-Na?

Model for End-Stage Liver Disease with Sodium

Objective, laboratory-based score predicting 3-month mortality in cirrhosis. Used globally for transplant organ allocation priority.

## Formula Components (4 Variables)

Serum Bilirubin (µmol/L or mg/dL)
Serum Creatinine (µmol/L or mg/dL)
INR
Serum Sodium (mmol/L)

MELD-Na = MELD + 1.32 × (137 - Na)
- (0.033 × MELD × [137 - Na])

Score range: 6-40
Higher score = more severe disease

## Advantages Over Child-Pugh

- Entirely objective (no subjective grading)
- Incorporates renal function (creatinine)
- Hyponatraemia adds prognostic value
- Better predicts short-term mortality
- Continuous scale (finer discrimination)

## 3-Month Mortality by MELD-Na

MELD-Na <10: ~2% mortality
MELD-Na 10-19: ~6% mortality
MELD-Na 20-29: ~20% mortality
MELD-Na 30-39: ~52% mortality
MELD-Na ≥40: ~71% mortality

## 🚨 Transplant Referral Thresholds

MELD-Na ≥15:
Refer for transplant assessment
(EMA: MELD ≥15 synonymous with qualification for transplant listing)

MELD-Na ≥20-25:
Expedited transplant work-up

Child-Pugh ≥B7 with any decompensation:
Refer regardless of MELD-Na

## When to Refer for transplant (Summary)

- MELD-Na ≥15 (or rising trajectory)
- First episode of decompensation (ascites, variceal bleed, HE, jaundice)
- HCC within Milan criteria
- Hepatopulmonary syndrome
- Refractory ascites
- Recurrent HE despite lactulose + rifaximin

Median survival without transplant from first decompensation: ~2 years

# Palliative Care in End-Stage Liver Disease: Symptom Burden

ESLD symptom burden rivals or exceeds metastatic cancer. Palliative care should run PARALLEL to disease-directed therapy, not be reserved for end of life (AASLD 2022, Rogal et al).

### Pain

Prevalence 30-79%. First-line: paracetamol (2 g/day or less). NSAIDs CONTRAINDICATED. Preferred non opioid: Tapentadol

### Pruritus

Stepwise: cholestyramine, then rifampicin, then naltrexone, then sertraline. Emollients as adjunct. (may need involvement of palliative care or liver specialists

### Muscle Cramps

Prevalence 56-68%. Consider Magnesium (??), baclofen, zinc supplementation.

# Medications to Avoid in ESLD

## NSAIDs (All)

Ccontraindicated. Precipitate renal failure, GI bleeding, worsen ascites and sodium retention.

## Codeine and Morphine

Codeine: unpredictable CYP2D6 metabolism. Morphine: active metabolite (M6G) accumulates in renal impairment. Use fentanyl instead.

## Benzodiazepines

Precipitate or worsen hepatic encephalopathy. Prolonged half-life. Only for alcohol withdrawal or end-of-life care.  Prefer

## Careful with nephrotoxic medications

ARBs or ACEI + spiranolactone – monitor carefully

# Nutrition and Diet in Decomepnsated liver disease of ESLD

## What to Do

Calories: 35 kcal/kg/day or more
Protein: 1.2-1.5 g/kg/day (NEVER restrict, even with HE)
Late evening snack: 50 g complex carbohydrate and protein
4-6 small meals/day
BCAA supplements if intake inadequate
Zinc, thiamine, fat-soluble vitamins (A, D, E, K)
Sodium <2 g/day if ascites present

## What to Avoid

Protein restriction (worsens sarcopenia, does NOT improve HE)
Excessive sodium (>2 g/day)
Alcohol (any amount)
Prolonged fasting (>6 hours)
Raw/undercooked shellfish (Vibrio vulnificus risk)
Herbal supplements (many hepatotoxic)

Highly recommend – Dietician review

# End-of-Life Care: Key Principles

1

## Parallel Planning

Palliative care runs alongside disease-directed therapy. Discuss goals of care early. Identify substitute decision-maker. MELD-Na and CTP guide prognostic conversations.

2

## Symptom Assessment

Screen for depression and anxiety. Assess carer burden. Regular MDT review with hepatology, palliative care, social services.

3

## Transplant and Hospice

Assess transplant candidacy early if appropriate. Consider hospice when transplant is not an option and repeated decompensation occurs despite maximal medical therapy.

# Back to Our Case: Putting It All Together

1

## Diagnosis

56M with chronic liver disease (LFTs abnormal >12 months). Likely MASH given metabolic risk factors (BMI 29, fasting hyperglycaemia). Moderate alcohol use.

2

## Investigations Ordered

Core liver screen (viral, autoimmune, metabolic). Liver ultrasound. Calculate FIB-4 from available bloods. (FIB 4 – 3.8), referral for Fibroscan

3

## Risk Stratification

MASH with possible DM and moderate alcohol: ~15% risk of cirrhosis in 5 years (if not already present). ~10% risk of decompensation and HCC in 10-20 years.

4

## Management Plan

Lifestyle: weight loss target 7-10%, Mediterranean diet, exercise 150 min/week, minimise alcohol. Metabolic optimisation. And referral to sepcailist service for fibrosis assessment

# Key Take-Home Messages for GPs

01

## Screen and Stage Early

Use FIB-4 as first-line. Sequential pathway with elastography reduces unnecessary referrals by 80%. Focus on at-risk populations: diabetes, obesity, alcohol.

02

## Fibrosis Stage Drives Prognosis

Not steatohepatitis. The 20% Rule: F3 to cirrhosis in 2 years, compensated cirrhosis to decompensation in 2 years for rapid progressors.

03

## Treat CSPH, Not Just Varices

Carvedilol 3.125-12.5 mg/day at the point of CSPH (LSM 25 kPa or above). PREDESCI: halved decompensation. HCC surveillance 6-monthly for all cirrhosis.

# Thank You

**Questions and Discussion**

Dr NJ Arachchi
Gastroenterologist

Key References:

Baveno VII (de Franchis et al, J Hepatol 2022)
BSG Guidelines (Newsome et al, Gut 2018)
NHMRC Alcohol Guidelines 2020
AASLD Practice Guidance 2023
GESA Pathways

# Helicobacter pylori Management and Gastric Intestinal Metaplasia



Werribee Mercy:  Health education session
Department of Gastroenterology
Dr Catherine Croagh
Head of Gastroenterology.

# Learning Objectives

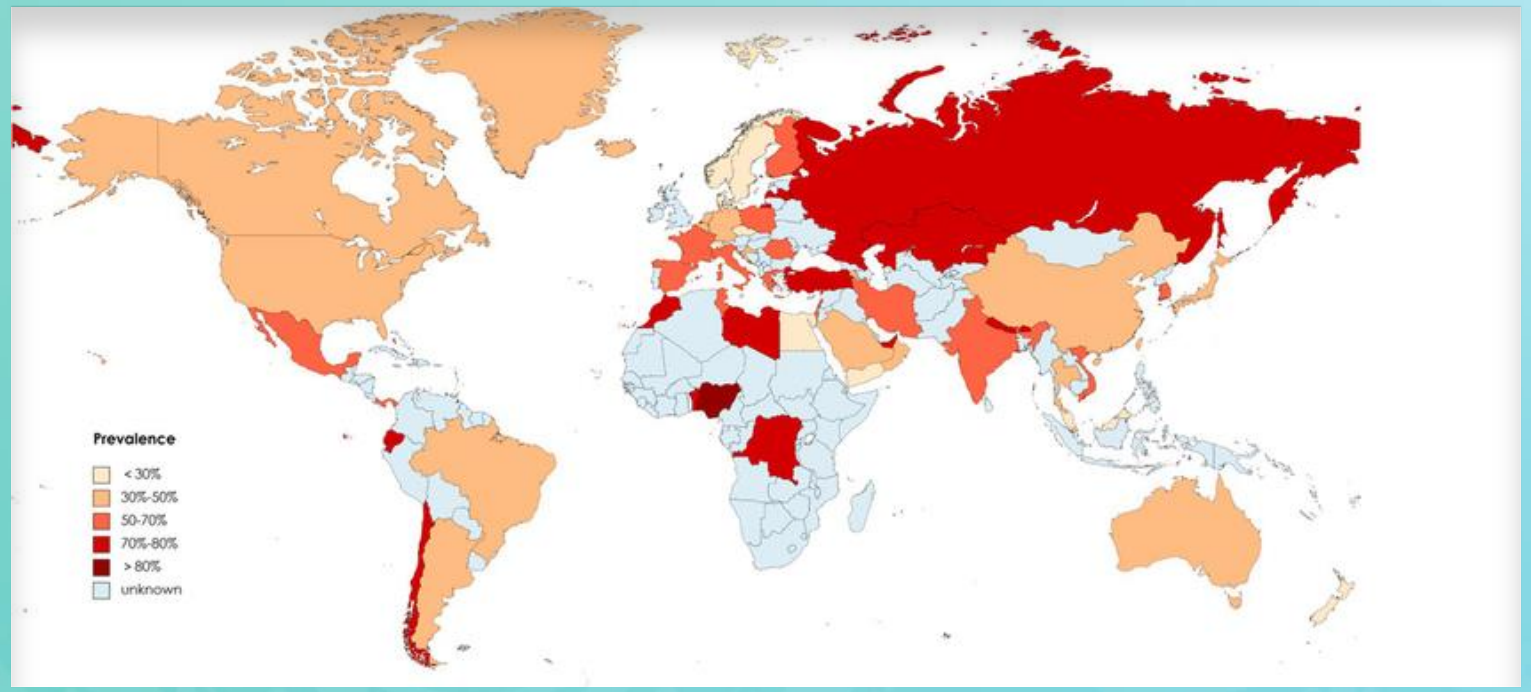
- Identify patients who should be tested for H pylori
- Select appropriate eradication regimens
- Formulate evidence-based management plans for patients with persistent H Pylori infection and treatment failure.
- Recognise gastric intestinal metaplasia, describe the risk factors requiring consideration of surveillance of gastric intestinal metaplasia.

Mercy Health
Care first

# Why H pylori Matters

- Most prevalent chronic bacterial infection worldwide.
- Gram negative bacterium - coevolved with humans for nearly 60,000 years.
- 1982: Drs. Barry Marshall and Robin Warren elucidated *H. pylori*'s role in gastritis and peptic ulcers and shared the 2005 Nobel Prize for their landmark discovery
- Major cause of peptic ulcer disease
- WHO class I carcinogen (leading cause of infection atttributable cancers 37%)
- Eradication reduces gastric cancer risk

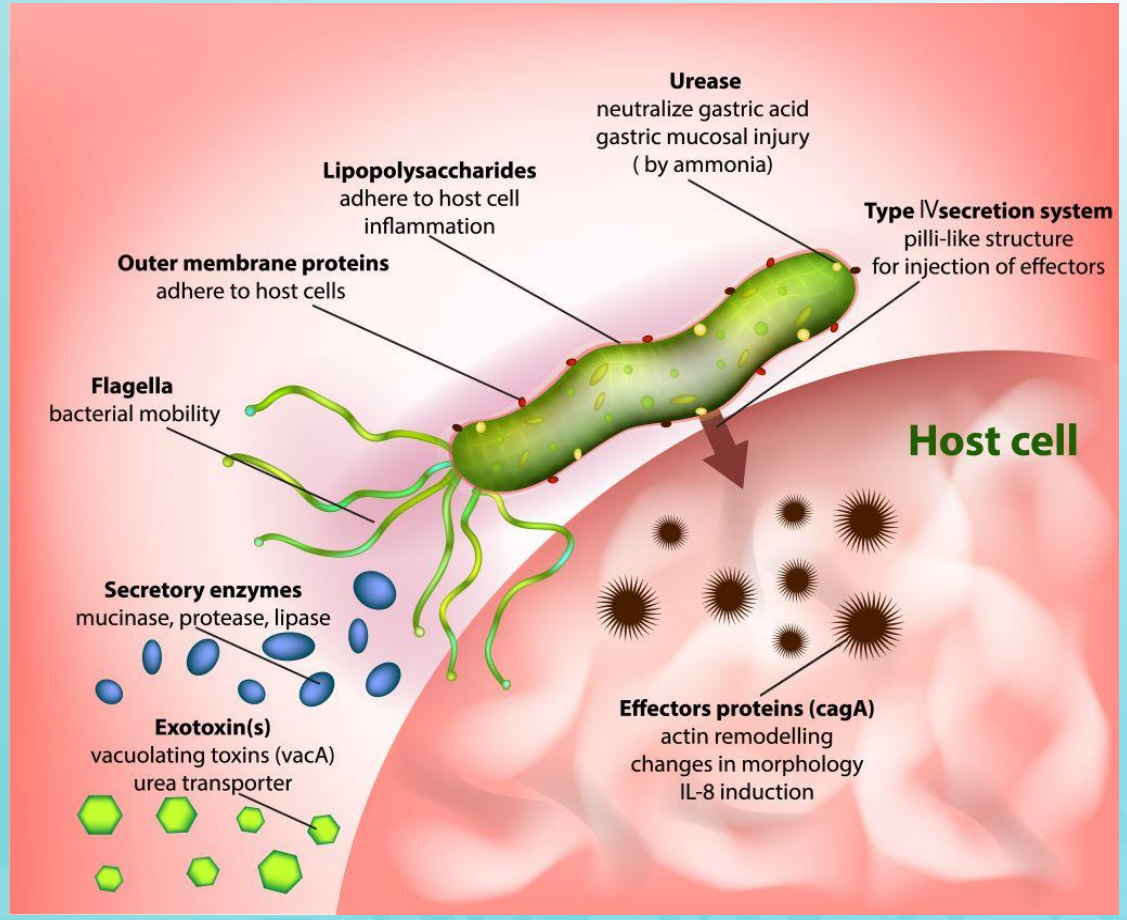
# Epidemiology




- Global prevalence ~40–50%
- Australia ~15–30%
- Major determinant of infection is Socioeconomic status in childhood
- Transmission: person to person (gastro oral, oral oral). Transmission through water, food, pets speculative.

# Pathophysiology

- Colonises gastric mucous layer
- Urease neutralises acid (splits Urea into CO2 and Ammonia)
- Virulence factors: CagA (carcinogen) and VacA (apoptosis and cell death).
- Chronic inflammation leads to mucosal damage




https://acesurgery.sg/condition/h-pylori-infection/

# Clinical Conditions Associated

Mercy Health
Care first

- Peptic ulcer disease (15-20% lifetime risk if H pylori infected)
- Chronic gastritis.
- Gastric Cancer (globally 6th most common cancer and 3rd most common cause of cancer death – WHO). H pylori confers a 2% lifetime risk.
- MALT (mucosa associated lymphoid tissue) lymphoma
- Iron deficiency anaemia
- Dyspepsia

# When Should GPs Test? - Only if treatment is intended.

Mercy Health
Care first

- Dyspepsia.
- Peptic ulcer disease
- Family history gastric cancer
- Known history of
  - **Gastric IM/Gastric cancer/MALT lymphoma**
  - **Autoimmune gastritis (due to being a cofactor in development of adenocarcinoma/NETs)**

# Limited evidence to support testing

- Chronic use of aspirin or NSAIDs.
- ITP (eradication may improve this).
- Unexplained iron deficiency (H pylori interferes with absorption of iron. Data more conclusive in children than adults)
- Individuals from high gastric Ca prevalence areas (Japan, Korea or Chinese origin)
- Adult household members of infected individual.

Fig. 1.2, Age-standardized incidence and mortality rates (per 100 000 person-years) for the 25 countries with the highest incidence rates of gastri

ncbi.nlm.nih.gov/books/NBK615780/figure/fig1_2/?report=objectonly

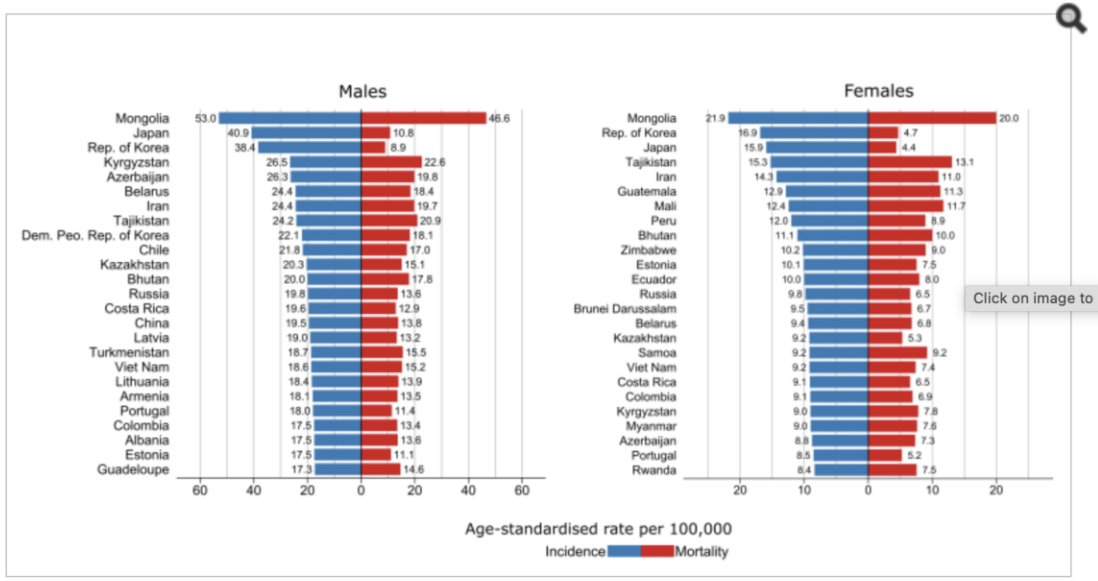



**Fig. 1.2 Age-standardized incidence and mortality rates (per 100 000 person-years) for the 25 countries with the highest incidence rates of gastric cancer, from GLOBOCAN 2022. Source: Ferlay et al. (2024) [1].**

From: Chapter 1, The global epidemiology of gastric cancer and *Helicobacter pylori*: current and future perspectives for prevention

Population-Based *Helicobacter pylori* Screen-and-Treat Strategies for Gastric Cancer Prevention: Guidance on Implementation.
Park JY, editor.
Lyon (FR): International Agency for Research on Cancer; 2025.

# Upper gastrointestinal endoscopy categorisation guidelines for adults 2018

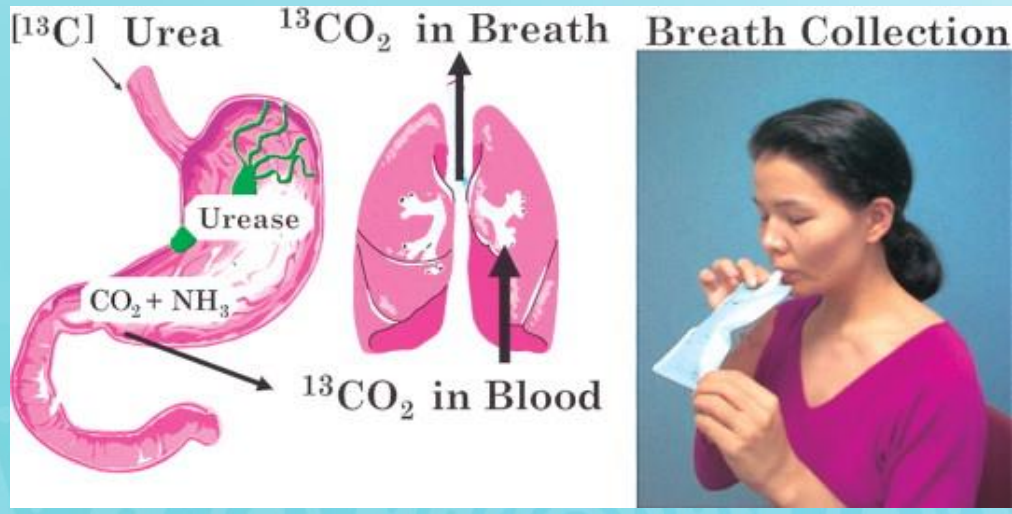
# When to refer for Endoscopy

Mercy Health
Care first

| | Category 1 (<30 days) | Category 2 (<60 days) | Category 3 (<180 days) | Comments/Not Indicated |
|---|---|---|---|---|
| **Indication A: Symptoms and investigations** | | | | |
| ➢ **Dysphagia** alone is an automatic Category 1<br>➢ **Additional symptom**: dyspepsia, GORD, upper abdominal pain, persistent nausea/vomiting, early satiety or unexplained loss of appetite<br>➢ **Abnormal blood test**: low Hb, low ferritin, microcytosis, hypochromia, raised platelets | | | | |
| 1. | **Dysphagia**<br>• any age | | | |
| 2. | **Haematemesis/Melaena**<br>• any age (see Comments) | | | Delayed presentation of symptoms; assume haemodynamically stable and no ongoing acute bleed requiring immediate admission. |
| 3. | **Anaemia and/or Iron deficiency**, *and:*<br>• age ≥ 55 years | **Anaemia and/or iron deficiency**, *and:*<br>• age < 55 years | | Refer to investigation of iron-deficiency anaemia in Explanatory notes. |
| 4. | **Abnormal imaging**, likely oesophageal or gastric cancer<br>• any age | | | Upper gastrointestinal endoscopy is not indicated for metastatic adenocarcinoma of unknown origin when results will not alter management. |
| 5. | **Weight loss, unexplained**, *and:*<br>• age ≥ 55 years, *plus*<br>– any **additional symptom**<br>*or*<br>– **abnormal blood test** or imaging | **Weight loss, unexplained**, *and:*<br>• age < 55 years, *plus*<br>– any **additional symptom**<br>*or*<br>– **abnormal blood test** or imaging | | |
| 6. | **Dyspepsia**, *and:*<br>• age ≥ 55 years, *plus*<br>– any **additional symptom**<br>*or*<br>– **abnormal blood test** or imaging<br>*or*<br>– atrophic gastritis<br>*or*<br>– FHx of upper GI cancer in 1st degree relative | **Dyspepsia**, *and:*<br>• age < 55 years, *plus*<br>– any **additional symptom**<br>*or*<br>– **abnormal blood test** or imaging<br>*or*<br>– atrophic gastritis<br>*or*<br>– FHx of upper GI cancer in 1st degree relative<br><br>**Dyspepsia**, *and:*<br>• any age, *plus*<br>– non-responsive to PPI and/or *H. pylori* therapy or *H. pylori*-negative | | Refer to test and treat policy for *H. pylori* in Explanatory notes.<br><br>Upper gastrointestinal endoscopy is not indicated if symptoms resolved after test and treatment for *H. pylori*. |
| 7. | **Dyspepsia**, *and:*<br>• any age, *plus*<br>– known intestinal metaplasia/gastric dysplasia | | | |
| 8. | **GORD, recent onset**, *and:*<br>• age ≥ 55 years, *plus*<br>– any **additional symptom**<br>*or*<br>– **abnormal blood test** or imaging | **GORD, recent onset**, *and:*<br>• age < 55 years, *plus*<br>– any **additional symptom**<br>*or*<br>– **abnormal blood test** or imaging | | Upper gastrointestinal endoscopy is not indicated on assessment of extra-oesophageal GORD symptoms including choking, coughing, hoarseness, asthma, laryngitis, chronic sore throat, or dental erosions.<br><br>Upper gastrointestinal endoscopy is not indicated for asymptomatic or uncomplicated sliding hiatal hernia. |
| 9. | **GORD, non-responsive**, *and:*<br>• age ≥ 55 years, *plus*<br>– known Barrett's | **GORD, non-responsive**, *and:*<br>• age < 55 years, *plus*<br>– known Barrett's oesophagus | **GORD, non-responsive**, *and:*<br>• age < 55 years | Assume GORD non-responsive after 6-8 weeks of double dosage PPI treatment. |
| | | | | |
| | **Category 1 (<30 days)** | **Category 2 (<60 days)** | **Category 3 (<180 days)** | **Comments/Not Indicated** |
| **Indication A: Symptoms and investigations** | | | | |
| | oesophagus | **GORD, non-responsive**, *and:*<br>• age ≥ 55 years | | |
| 10. | **Upper abdominal pain**, *and:*<br>• age ≥ 55 years, *plus*<br>– any **additional symptom**<br>*or*<br>– **abnormal blood test** or imaging | **Upper abdominal pain**, *and:*<br>• age < 55 years, *plus*<br>– any **additional symptom**<br>*or*<br>– **abnormal blood test** or imaging | | Seek specialist review if upper abdominal pain indication not fulfilling criteria. |
| 11. | **Nausea/vomiting, persistent**, *and:*<br>• age ≥ 55 years, *plus*<br>– any **additional symptom**<br>*or*<br>– abnormal **blood test** or imaging | **Nausea/vomiting, persistent**, *and:*<br>• age <55 years, *plus*<br>– any **additional symptom**<br>*or*<br>– **abnormal blood test** or imaging | | |
| 12. | **Inflammatory bowel disease in adults**<br>• no Category 1 indication | **Inflammatory bowel disease in adults**<br>• at the time of diagnosis if upper GI symptoms present | | |
| 13. | **Pernicious anaemia (endoscopically diagnosed)** *and:*<br>• any age, *plus*<br>– any **additional symptom** | **Pernicious anaemia (serologically diagnosed)**<br>• asymptomatic at time of diagnosis | | |
| 14. | **Coeliac disease**<br>• no Category 1 indication | **Coeliac disease**<br>• suspected coeliac disease with positive serology<br>*or*<br>• known coeliac disease with no exposure to gluten, *plus:*<br>– persistent high serological titres after 12 months<br>*or*<br>– persistent diarrhoea, abdominal pain, weight loss, fatigue, or anaemia | | Refer to Explanatory notes for:<br>- serology test<br>- information on gluten |
| 15. | **Cirrhosis**<br>• no Category 1 indication | **Cirrhosis**<br>• at time of diagnosis to assess for oesophageal varices | | |

# Testing Options

- Urea breath test (Sens and Spec 95%).
- Stool antigen test (monoclonal Abs to specific H pylori Ag. Sens 93%, Spec 96%)
- Serology not recommended for active infection. (Cannot distinguish between current and past infection).
- Histology:
  - **H&E : Sens 69-93% and Spec 87-90%**
  - **rapid urease testing**
  - **immunohistochemistry –Sens/Spec to >95%)**



# Factors affecting test accuracy

Mercy Health
Care first

- Anything that diminishes bacterial counts decreases test sensitivity (active upper GI bleeding, antibiotics, bismuth-containing products, PPIs).
- Resistance can be tested for by bacterial culture (but Sens 80%) or molecular testing (to identify specific DNA mutations known to confer resistance).

# Preparing for Testing

Mercy Health
Care first

- Stop PPI for at least one preferably 2 weeks (switch to H2RAs or antacids which don’t impact UBT or Stool Ag)
- Stop antibiotics for 4 weeks
- Stop bismuth for 4 weeks

# Treatment Principles

- Combination antibiotic therapy
- Acid suppression required.
- Major determinant of eradication success with PPI-AC is pretreatment Clarithromycin resistance.
- 14-day therapy may improve eradication rates (by about 10%) but consider compliance and adverse effects.

WGO GUIDELINE

***Helicobacter pylori* World Gastroenterology Organization Global Guideline**

*Katelaris, Peter MD*; Hunt, Richard et al Journal of Clinical Gastroenterology 57(2):p 111-126, February 2023.*

# Antibiotic resistance

Mercy Health
Care first

- Australia has had low clarithromycin (6% to 8%) and high metronidazole (45% to 50%) resistance rates reported.
- Levofloxacin data are sparse but primary resistance seems very low, with the possible exception of rates in migrants from high resistance regions (SE Asia, Western Pacific).
- H. pylori resistance to amoxicillin is very low
- H. pylori resistance to tetracycline is almost nonexistent.

WGO GUIDELINE

***Helicobacter pylori* World Gastroenterology Organization Global Guideline**

*Katelaris, Peter MD*; Hunt, Richard et al Journal of Clinical Gastroenterology 57(2):p 111-126, February 2023.*

# First Line Regimen (Australia)

Mercy Health
Care first

- Esomeprazole 20 mg twice daily
- Amoxicillin 1 g twice daily
- Clarithromycin 500 mg twice daily
- Typically 7 days in pack (Nexium HP7), may extend to 14 days (if pt has previous macrolide exposure).
- In Australia Reported 7-day eradication rates are 80% to 87%. Fourteen-day therapy has not been studied formally.

# Alternative Triple Therapy (Penicillin allergic)

- PPI twice daily
- Clarithromycin 500 mg twice daily
- Metronidazole 400 mg twice daily
- Duration 7-14 days

Mercy Health
Care first

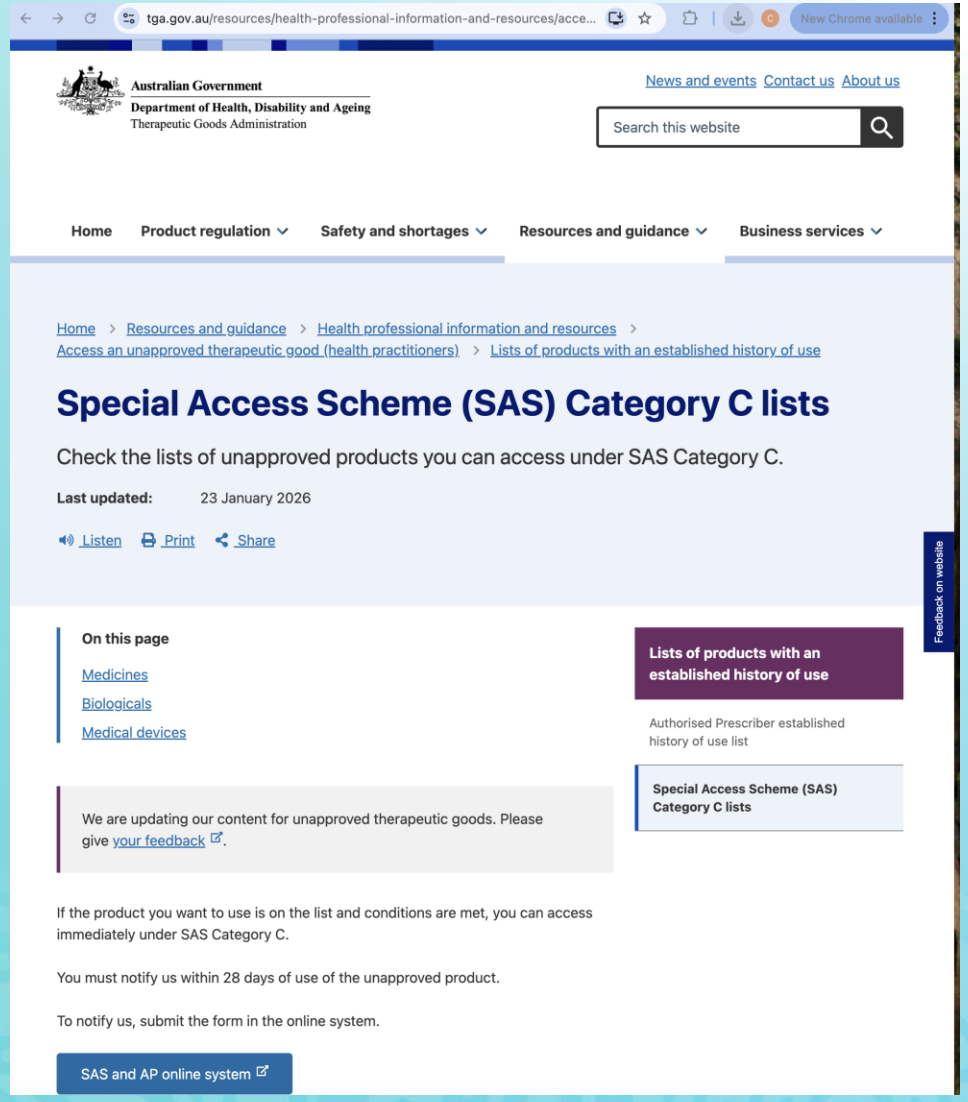
# Second line/Salvage therapies

Mercy Health
Care first

- Do not repeat  clarithromycin-containing regimen if first-line therapy failed.
- Use of salvage therapies should be confined to general practitioners with a special interest or referred for specialist care.
- The indications for therapy should be reaffirmed before proceeding.
- Salvage therapy is only occasionally guided by culture and susceptibility testing obtained from an endoscopic biopsy.

# Second line/Salvage therapies

- Levofloxacin, tetracycline, and bismuth are notregistered locally, so are not often used in first-line therapy.
- These drugs must be obtained through a special access scheme or by compounding pharmacies when required for salvage treatments.




Mercy Health
Care first

# Second line/Salvage therapies

Mercy Health
Care first

## Bismuth Based Quad therapy

**7-14 days**

- PPI twice daily
- Colloidal bismuth subcitrate 120 mg orally, 4 times daily
- Metronidazole 400 mg tds
- Tetracycline 500 mg QID
- Duration 10–14 days

## Levofloxacin based Triple therapy

**10-14 days**

- Proton pump inhibitor orally, twice daily for 10 days
- Amoxicillin 1 g orally, twice daily for 10 days
- Levofloxacin 250 mg orally, twice daily (or 500 mg orally, once daily) for 10 days OR
- Moxifloxacin 400 mg orally, once daily for 10 days.

# Second line/Salvage therapies

Mercy Health
Care first

- Eradication rates of 80-90%.
- When one of these treatments fail, the other therapy is the usual third choice.
- Other salvage therapies:
  - **Rifabutin based triple therapy (risk of significant neutropenia 1%). Eradication rates 76%**
  - **High dose dual PPI with Amoxicillin (some success).**
  - **Vonoprazan based dual or triple therapies. (Vonoprazan : potassium-competitive acid blocker) suppresses acid production of gastric parietal cell through H+, K+-ATPase enzyme system in a potassium competitive manner)**

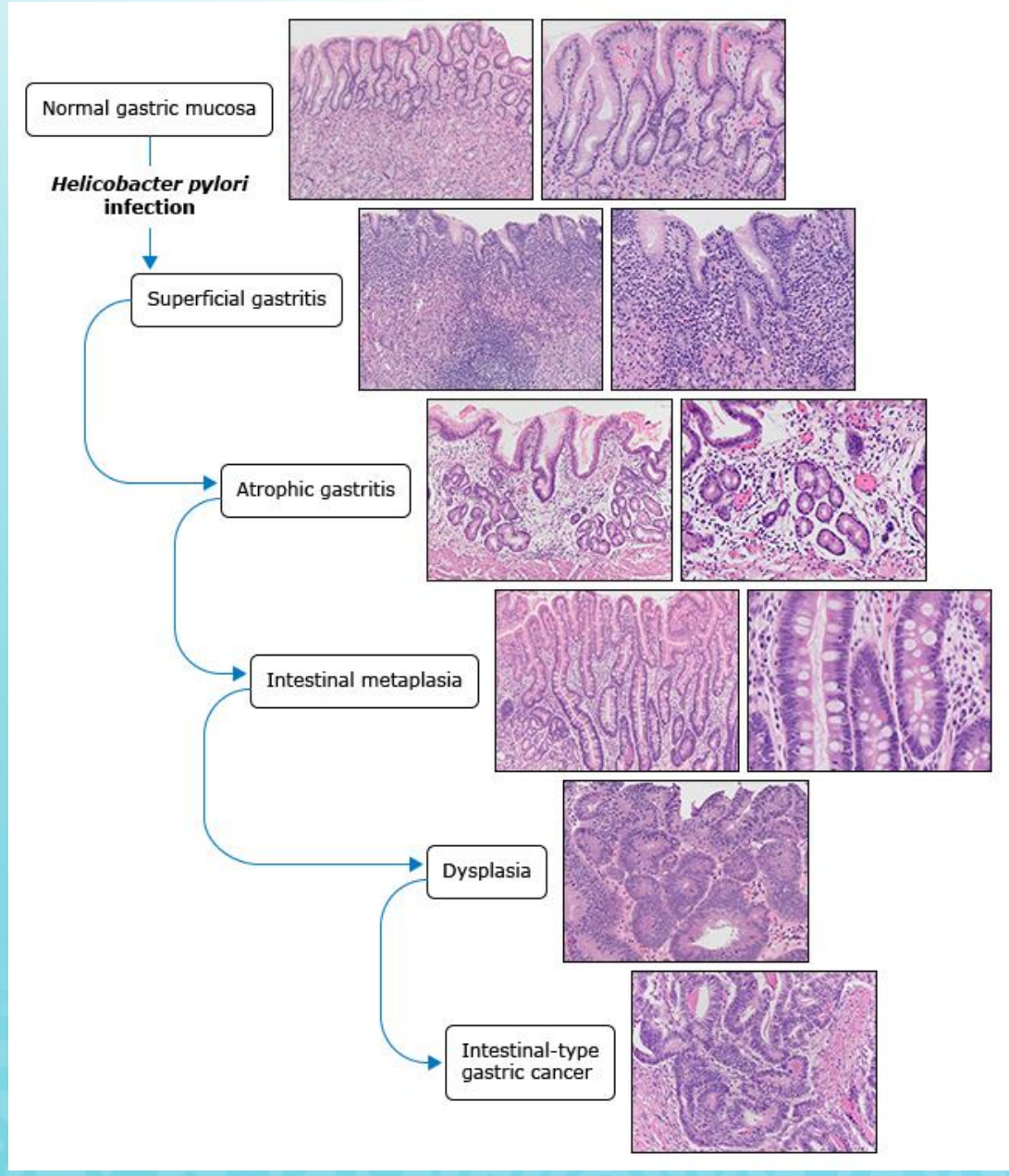
# Prescribing Tips for GPs

Mercy Health
Care first

- Stress adherence to full course.
- Avoid alcohol with metronidazole, warn about metallic taste.
- Advise regarding side effects esp Bismuth blackening of tongue, Tetracyline – avoid in pregnancy.
- Check for eradiation (UBT or Stool Ag) at least 4 weeks after therapy.

# Gastric Intestinal Metaplasia

- Replacement of gastric mucosal epithelium with intestinal type epithelium
- Pre-malignant condition in the gastric cancer pathway.
- Prevalence of Gastric IM increases with age and tobacco use and is higher in those from Gastric Ca high prevalence countries.
- Risk of cancer increased but modest over lifetime. Rates of progression per 1000 person-years for atrophic gastritis (AG), GIM, and dysplasia were 2.1, 3.1, and 9.5, respectively



# Risk Factors for Gastric Cancer

Mercy Health
Care first

- H pylori -persistent H pylori infection (1.5-2% lifetime risk of Gastric cancer in infected individuals)
- Family history gastric cancer
- Smoking.
- Diet: High salt intake, consumption of pickled foods and diets low in antioxidants
- Race, ethnicity and country of origin: (in Australia Japan, Korea and Eastern China highest risk populations).
- Gastric intestinal metaplasia features.

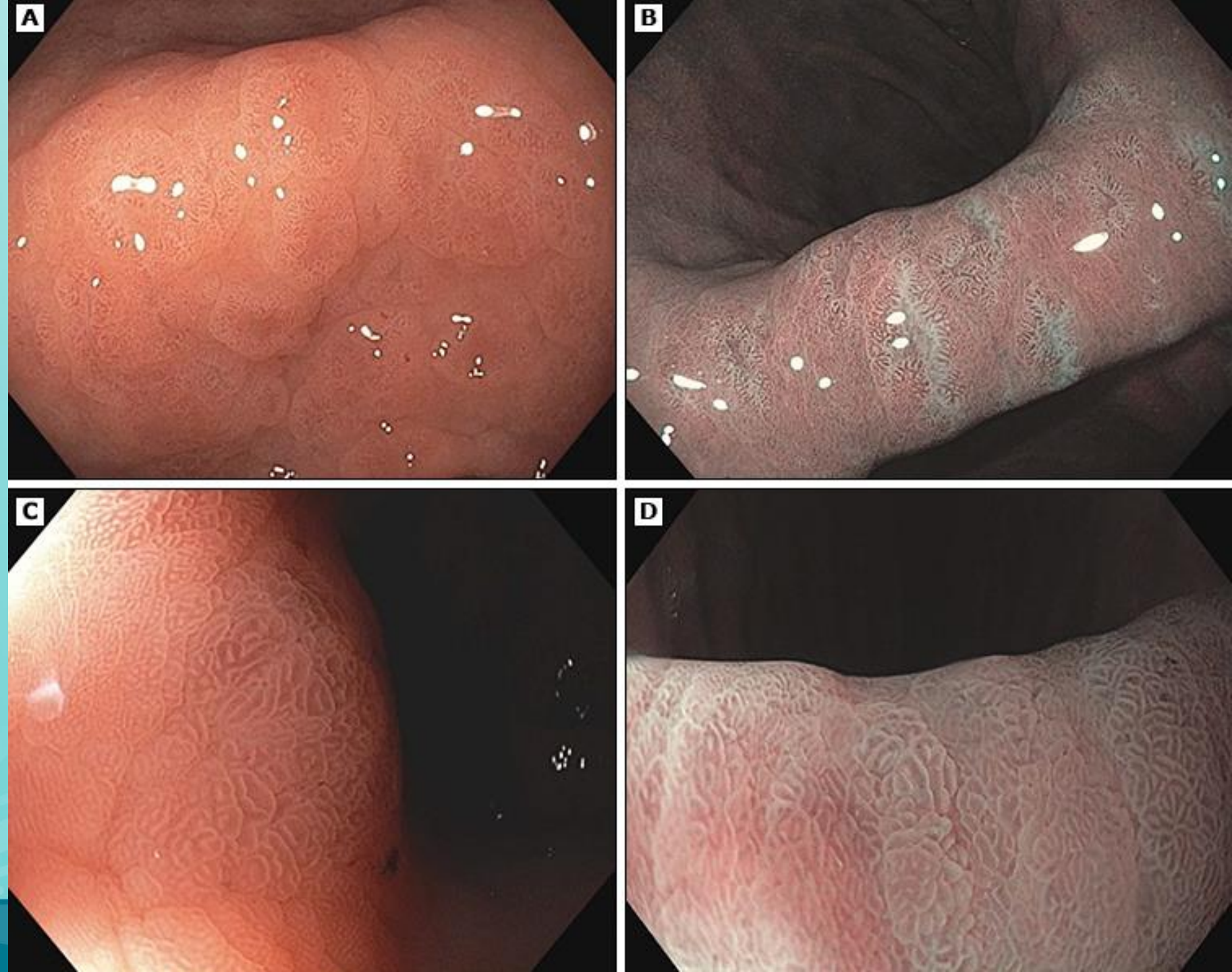
# Endoscopy in Gastric IM

Diagnosis: thorough high definition white light endoscopy and image enhanced endoscopy.

Gastric Mapping (using Sydney Protocol) and targeted biopsy sites. Photodocumentation.

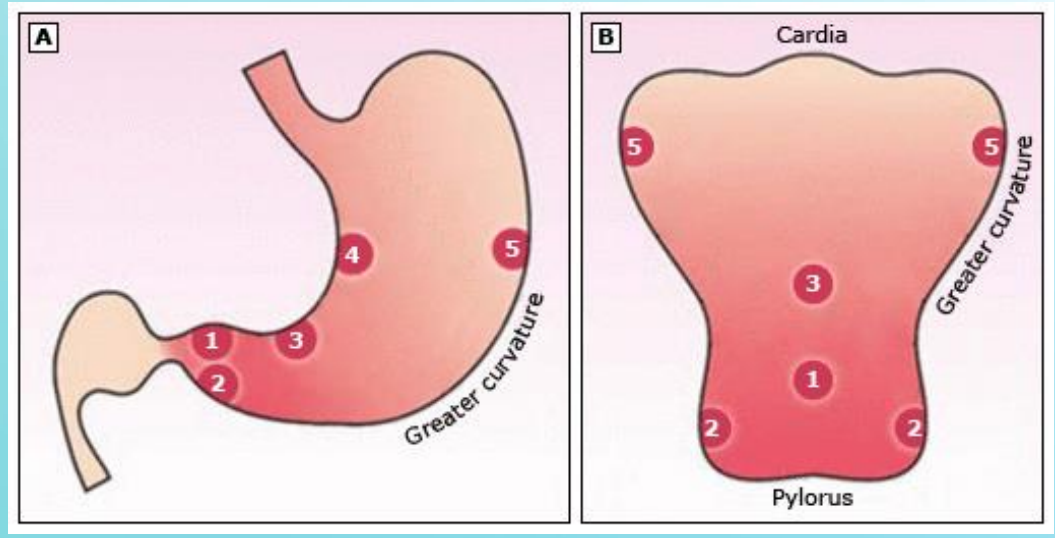
Patchy white mucosa with a tubulovillous pattern.

Light blue crests are thin white (on BMI) or blue lines at the borders of Tubulovillous glands.

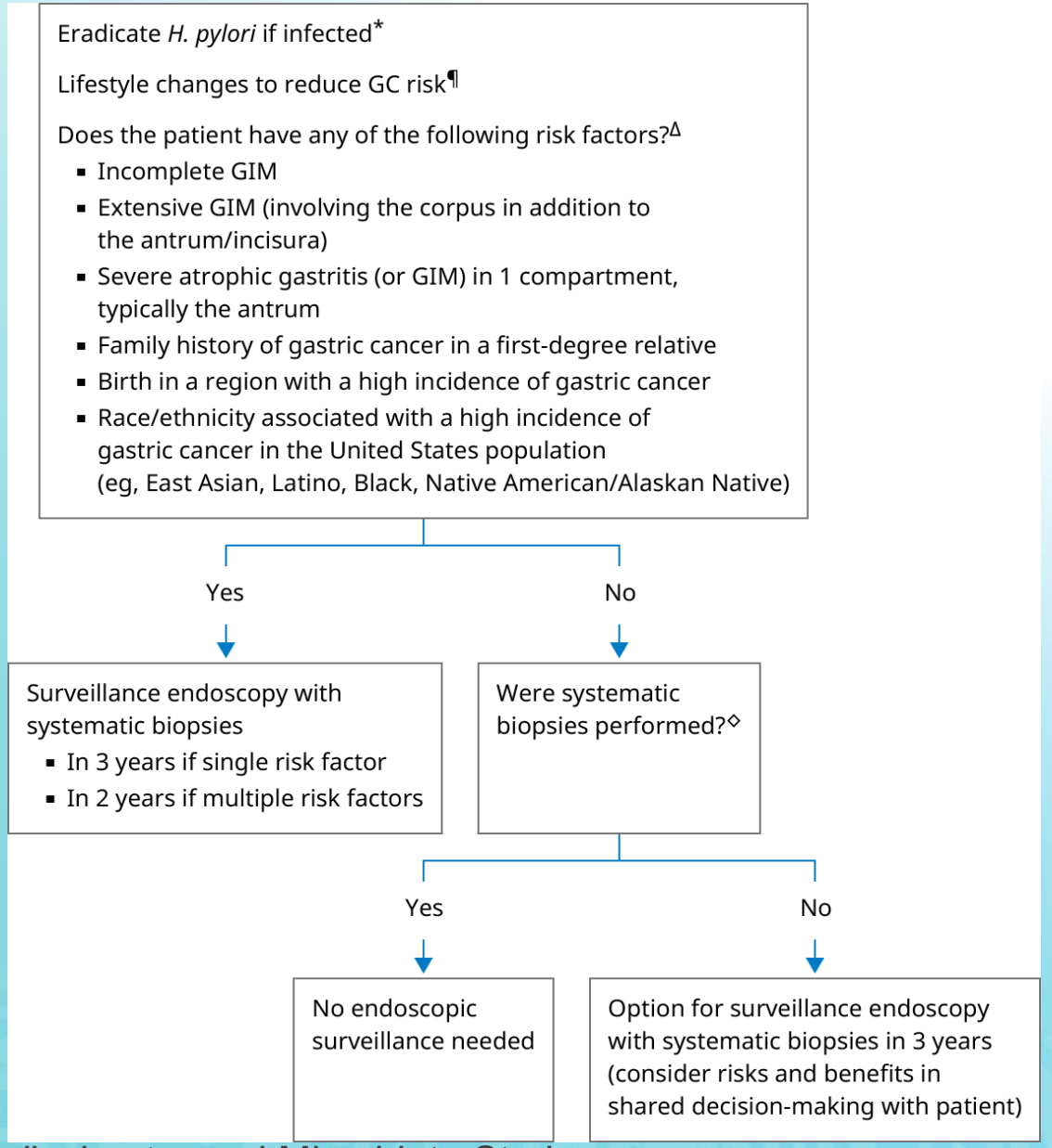


# Increased risk of Gastric Cancer in Gastric Intestinal metaplasia

- Extent of GIM : Extensive (involving the corpus in addition to the antrum/incisura) assoc with higher progression than limited
- Histologic Subtype: Incomplete GIM (colon-type) higher than complete
- OLGA/OLGIM histologic stage: (operative link in Gastritis/Gastric IM Assessment). Validated histology staging based on Sydney protocol.
- Severe atrophic gastritis (at least 2/3 of the glands are involved)



# Management of Gastric IM




Guidelines available :
American College of Gastroenterology 2025.
American Gastroenterological association (AGA) 2020 +2025 update.
European Society of Gastrointestinal Endoscopy (ESGE), European Helicobacter and Microbiota Study Group (EHMSG), European Society of Pathology (ESP): Guideline for the management of epithelial precancerous conditions and early neoplasia of the stomach (MAPS III), update (2025).

Mercy Health
Care first

# Thank you

Questions?

# *Measuring outcomes for this session*

To obtain Measuring Outcomes hours for this session use the RACGP's Measuring Outcomes Tool.

Follow these five steps:

1. Log-in to myCPD
2. At very top of myCPD, click on 'Log'
3. From drop-down menu, click on 'Measuring Outcomes Tool'
4. Complete the form
5. Once you have completed the form, go to top of form and click 'Submit'

# *Thank you for attending. What's next?*

*After this session you will receive:*

1. *Slides, resources and the recording of this session within the week*
2. *RACGP CPD hours will be uploaded within 14 days.*
3. *Attendance certificate will be received within 4-6 weeks.*

- ***Register for more education sessions here:*** *nwmphn.org.au/resources-events/events*
- ***Past education sessions can be found here:*** *nwmphn.org.au/resources-events/resources*

**Feedback - QR code**

We welcome your feedback. Let us know if you got what you needed from this session.