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Happy to be here again for another exciting episode. Today's information comes namely from Tintinallis and the Merck Manual with a few other sources sprinkled in that we'll mention when we get there.

Objective One

Given a patient with undefined chest pain, take an adequate history to make a specific diagnosis (e.g., determine risk factors, whether the pain is pleuritic or sharp, pressure, etc.).

The differential for chest pain is broad. It can be helpful to think about where the pain may be coming from and how that might be experienced by the patient. Pain typically comes in three flavours: Pleuritic, Somatic and Visceral pain. This approach can help localize the source of pain and refine your differential.

What is pleuritic chest pain?

Pleuritic chest pain is a type of sharp, stabbing pain that gets worse when the patient breathes and even worse when they take a deep breath. As the name suggests it is due to pain or irritation at the pleura.

Some considerations for your differential if your history uncovers a pleuritic chest pain:

Pleuritic Chest Pain
- Pulmonary embolism - Pneumonia - Spontaneous pneumothorax - Pericarditis - Pleurisy

The chest wall from the skin down to the parietal pleura, is innervated by spinal nerves, giving rise to somatic pain. Somatic pain can be thought of as the skin, muscle and bones - the things you can touch and break.
Pain from the overlying MSK structures will be specifically localizable by the patient and often feels more 'sharp'. As you can imagine, this could have similar characteristics to pleuritic pain with changes in breathing, and can make it difficult to differentiate these two.

Tintinalli's offers a differential for somatic chest wall pain including:

Somatic, Chest Wall Pain
- Costosternal syndrome - Costochondritis or "tietze's syndrome" - Precordial catch syndrome - Xiphodynia - Radicular syndromes - Intercostal nerve syndromes - fibromyalgia

Deeper than this is the visceral pleura innervated by visceral nerves. These often cluster into one area of the spinal cord and share cerebral mapping with somatic nerves.
So if it's more of a vague, less sharp or poorly localized pain, think about visceral pain.
This is why cardiac pain, for example, can often be felt as radiating to the arms, or neck.

The visceral nerves cause a sensation that is much more difficult for patients to point to and to describe. As a result they'll often gesture to an overall area, and use words like 'discomfort', 'heaviness', 'pressure', 'tightness' or 'aching.

Some items for your differential here include:

Visceral Chest Pain DDx
- Typical angina - Unstable angina - Acute myocardial infarct - Aortic dissection - Oesophageal rupture - Oesophageal reflux, or spasm - Mitral valve prolapse

The table for this differential from Tintinalli's will be in the shownotes.

Visceral Chest Pain DDx	Pleuritic Chest Pain	Somatic, Chest Wall Pain
- Typical angina - Unstable angina - Acute myocardial infarct - Aortic dissection - Oesophageal rupture - Oesophageal reflux, or spasm - Mitral valve prolapse	- Pulmonary embolism - Pneumonia - Spontaneous pneumothorax - Pericarditis - Pleurisy	- Costosternal syndrome - Costochondritis or "tietze's syndrome" - Precordial catch syndrome - Xiphodynia - Radicular syndromes - Intercostal nerve syndromes - fibromyalgia

Objective Two

Given a clinical scenario suggestive of life-threatening conditions, begin timely treatment.

There are seven deadly sins, I mean chest pains, that we need to rule out. These include acute coronary syndrome, pulmonary embolism, tamponade, aortic dissection, pneumothorax, pneumonia and a ruptured esophagus aka Boorhave's syndrome).
ACS will be covered in objective three, as well as Topic 56. Pulmonary embolism will be covered in objective 5. Pneumonia is covered in depth by Topic 77.

Let's start with cardiac tamponade.

Tamponade Tintinalli's Ch 55, pp 387

Risk factors that should make you suspicious:

- Metastatic malignancy (40% of non-traumatic cases)
- Acute idiopathic pericarditis (15% of non-traumatic cases)
- Uremia (10%)
- Bacterial or tubercular pericarditis
- Chronic idiopathic pericarditis
- Haemorrhagic – on anticoagulants
- Others:
    - Systemic lupus erythematosus

    - Post-radiation
    - Myxedema
- Trauma, particularly to the cardiac box

Clinical Scenario to consider Cardiac Tamponade

- Dyspnoea at rest and with exertion
- Symptoms due to the cause (refer to above risk)
- Tachycardia
- Low systolic blood pressure
- Narrow pulse pressure
- Pulsus paradoxus – this is an abnormally large fall, greater than 10mmHg systolic pressure with inspiration

Physical Exam Findings

- Distended neck veins
- Apical impulse is indistinct or 'tapping' in quality
- May be RUQ tenderness due to hepatic venous congestion

Assess for using:

- CXR may show enlarged cardiac silhouette, which will look like cardiomegaly due to surrounding fluid that is indistinguishable from the heart itself
    - Pulmonary vasculature will usually look normal
    - Epicardial fat pad sign may occasionally be seen within the cardiac silhouette (see image in show notes)
- ECG, may show:
    - Low voltage (<0.7mV) QRS complexes and
    - ST segment elevation, due to inflammation of the epicardium
    - PR segment depression, as in pericarditis
    - In a large tamponade, may see Electrical alternans in the P and R waves (see this awesome finding in shownotes as well)
- Of course the best way to definitively identify a pericardial effusion that has tamponaded is with ultrasound.

Management

- volume expansion can help with cardiac output temporarily
- if haemodynaically unstable, emergency pericardiocentesis is necessary, however this is ideally done by your cardiology colleagues in the cath lab using echocardiographic guidance to avoid complications
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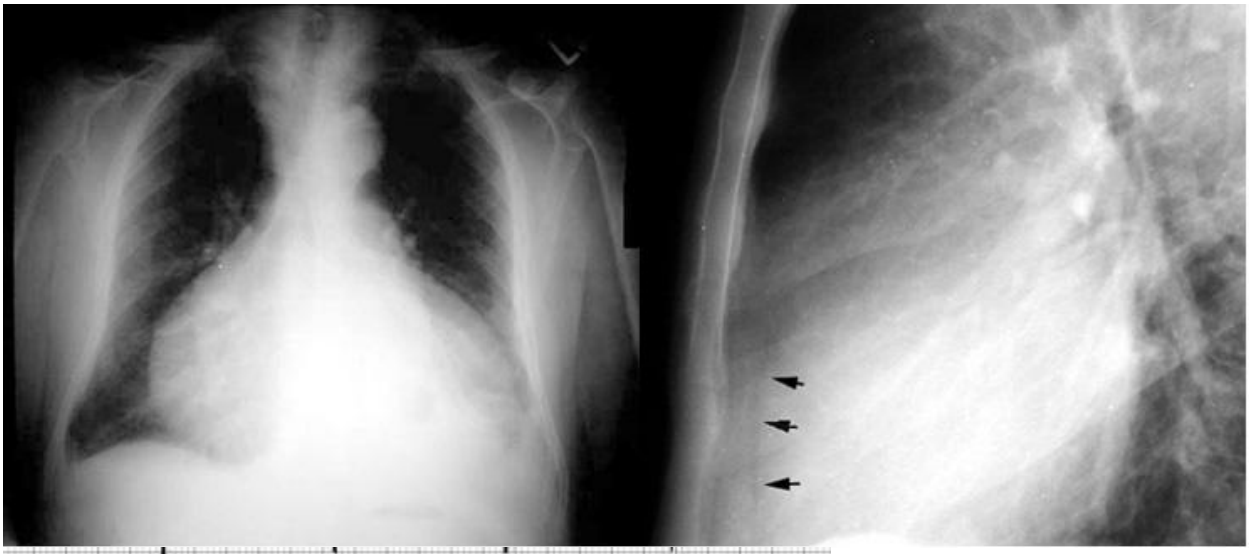
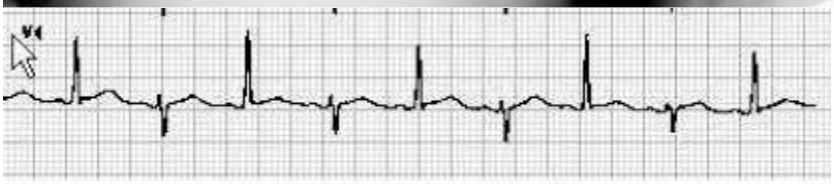




Electrical Alternans
showing alternating QRS complex axis

Next

Aortic Dissection [Tintinalli's Ch 48 pp 327]

Risk Factors Include:

- Male sex
- Age over 50 years
- Poorly controlled hypertension
- Cocaine or amphetamine use
- Bicuspid aortic valve or prior aortic valve replacement
- Connective tissue disorders, including: Marfan's and Ehlers-Danlos
- Pregnancy

Clinical Presentation to Consider an Aortic Dissection

- 'ripping' or 'tearing' sensation in chest
- May radiate to interscapular area of back
- Most often sudden onset and maximal at onset
- May migrate above and below the diaphragm, depending on location of dissection
- Secondary symptoms may result from occlusion of branch arteries caused by the dissecting flap
  - Stroke
  - Acute MI
  - Limb ischemia
- Physical Exam Findings
  these are insensitive and non-specific

        - unilateral pulse deficit of: carotid, radial, femoral artery LR 5.7
        - focal neuro deficits can occur in 17% of cases
        - new murmur of aortic insufficiency
    - Investigations
        - A normal CXR 'lowers' likelihood of dissection but doesn't exclude it
            - Widened mediastinum or abnormal aortic contour
            - Pleural effusions
            - Aortic intimal calcification displacement
        - Negative D-dimer lower likelihood, but also cannot exclude
        - ECG changes are fairly common. Up to 50% have ST or TW changes
        - Troponins are non-specific but do suggest worse prognosis
        - Need a CT Aortogram or Transoesophageal echocardiogram to definitively confirm
    - Initial Management includes: [Tin Ch 59 pp415]
        - Antihypertensives, with negative inotropic agent
            - This is to reduce blood pressure without increasing shear forces which can worsen the tearing
            - Beta-blockade is the usual with short-acting preferred
                - Propranolol, Labetolol or Esmolol
                - Usually a sBP 120-130 initially and 100-120 if possible without causing hypotensive symptoms
            - Then vasodilators like Nitroprussive may be added after
            - They likely need higher level of care, so if you need to ship for this get it going, or upstairs to the ICU

Let's talk Pneumothorax

Classically, a spontaneous pneumothorax occurs in tall, slender males. Of course penetrating trauma is also quite a risk for primary pneumothorax.

Risk Factors are

- Smoking
- Chronic lung diseases, such as asthma or COPD

Up to 3% develop a tension pneumothorax.

Clinical Presentation to Consider a Spontaneous Pneumothorax

- Sudden-onset
- Sharp and/or pleuritic chest pain with dyspnoea
    - The pleuritic component may resolve after 24 hours
- Sinus tachycardia is the most common physical finding
- Classic physical findings are:
    - Ipsilateral decreased breath sounds
    - Hyperresonance to percussion on side with pneumothorax
    - Decreased or absent tactile fremitus

Investigations

- CXR: look for loss of lung markings in the periphery and a pleural line running parallel to the chest wall
- Ultrasound: one of the great uses for bedside ultrasound. Very sensitive and specific, looking for absence of lung sliding in dependent areas of the chest.
- Chest CT of course

Management

If a tension pneumothorax is suspected, based on significant dyspnea, hypotension, tracheal deviation, etc, you may need to intervene more acutely with a needle decompression, followed by a chest tube.

If they are haemodynamically stable we can use some less invasive management options:

- Oxygen administration can help increase resorption of the pleural air
- For small and stable pneumothoraces, we can also elect to just observe and reassess:
    - Observe at least 4 hours with repeat CXR for progression/regression
    - Should return in 24h for repeat assessment

Oesophageal Rupture

[Tintinalli's Ch 48 pp 328]

aka 'boerhaaves'

This is a full-thickness tear of the oesophagus. Classically presents following forceful vomiting.

Diagnosis

Clinical suspicion and differentiating from ACS is key to avoiding delay. CT or the chest or emergency endoscopy are the best means for definitive diagnosis.

Clinical Presentation

Usually they are going to appear sick and may also have a few general associated symptoms:

- tachycardia
- febrile
- dyspnea
- diaphoresis

Physical exam may be helpful if you find the specific finding of 'Hamman's Crunch' which is a crepitus, varying with the heartbeat when auscultating the precordium. It is obviously rare and occurs with pneumomediastinum.

Investigations:

- CXR (a normal cannot rule it out, but a few findings might push you to look closer)
    - may show a pleural effusion, with the left side being more common than the right
    - pneumothorax

- pneumomediastinum
- pneumoperitoneum, or
- subcutaneous air

- If you suspect an oesophageal rupture, you need a CT with water-soluble contrast for definitive diagnosis.

Management

1. Resuscitation for shock
2. Broad spectrum IV antibiotics
3. Emergency surgical consult as soon as you suspect an oesophageal rupture

Objective Three

In a patient with unexplained chest pain, rule out ischemic heart disease.*

Risk Factors for Ischemic Heart Disease

Unless they are crashing, you're likely to have a chance to peruse your incoming patient's medical history quickly before they arrive. Of course one of the do-not-miss diagnoses for the patient presenting with chest pain is ACS or acute coronary syndromes, in particular an active infarct. For this, it's useful to remember the usual risk factors for coronary artery disease:

- Age over 40
- Being male, or a post-menopausal female
- Hypertension
- Tobacco use
- Hypercholesterolemia
- Diabetes
- Central obesity
- Family history of CAD:
    - MI in male family member before 55 years of age
    - MI in female family member before 65 years of age
- Sedentary lifestyle

Cocaine use can also be associated with acute myocardial infarct due to vasospasm.

Characteristics of cardiac chest pain

JAMA 'Rational Clinical Examination' series in 2015 analysed this question regarding history as well as the chest pain symptoms.

Of course, no one item is going to tell us if this is cardiac or not, and so it is presented as Likelihood ratios that are to be taken in summation to determine pre-test probability. These were assessed separately as items that are useful on history and those that are useful on exam.

On History

1. Abnormal prior stress test LR 3.1
2. Peripheral arterial disease LR 2.7
3. Prior CAD or MI, LR 2.0 and 1.6 respectively
4. The rest are even lower and include the usual suspects:
    a. age over 40,
    b. diabetes,
    c. being male sex or post-menopausal female,
    d. hyperlipidemia,
    e. HTN,
    f. tobacco use,
    g. fam hx of CAD,
    h. truncal obesity,
    i. sedentary lifestyle
    j. cocaine use

Obviously none of these likelihood ratios are particularly compelling in isolation. So they need to be taken collectively.

Table 1. Performance of Cardiac Risk Factors in Diagnosing Acute Coronary Syndrome[a]

	No.		% (95% CI)						%[b]	
Test	Studies	Patients	Sensitivity	Specificity	LR+ (95% CI)	I^2, %	LR− (95% CI)	I^2, %	PPV	NPV
Abnormal prior stress[c,61]	1	1777	12 (8-16)	96 (95-97)	3.1 (2.0-4.7)		0.92 (0.88-0.96)		32	12
Peripheral arterial disease[21,23,49]	3	6034	7.5 (2-11)	97 (95-99)	2.7 (1.5-4.8)	0	0.96 (0.94-0.98)	64	29	13
Prior CAD[37,40,49,57,60]	5	6396	41 (13-69)	79 (60-98)	2.0 (1.4-2.6)	87	0.75 (0.56-0.93)	96	23	10
Prior myocardial infarction[d]	9	10 491	28 (21-36)	82 (78-86)	1.6 (1.4-1.7)	42	0.88 (0.81-0.93)	81	19	12
Diabetes[e]	9	10 237	26 (21-32)	82 (77-85)	1.4 (1.3-1.6)	4	0.90 (0.86-0.94)	45	17	12
Cerebrovascular disease[21,23,49,70]	4	6682	10 (8-13)	93 (91-94)	1.4 (1.1-1.8)	18	0.97 (0.94-0.99)	14	17	13
Men[f]	12	21 113	66 (62-76)	50 (44-51)	1.3 (1.2-1.3)	65	0.70 (0.64-0.77)	39	16	9
Hyperlipidemia[g]	10	10 288	42 (31-55)	67 (56-79)	1.3 (1.1-1.5)	70	0.85 (0.77-0.93)	69	16	11
Hypertension[h]	11	10 931	59 (53-66)	52 (44-60)	1.2 (1.1-1.3)	51	0.78 (0.72-0.85)	29	15	10
Any tobacco use[i]	9	7 381	38 (28-47)	65 (55-75)	1.1 (0.9-1.3)	75	0.96 (0.85-1.1)	77	14	13
Family history of CAD[21,23,40,49,51,54,58]	7	8 717	37 (26-47)	64 (58-71)	1.0 (0.9-1.2)	54	0.99 (0.91-1.1)	65	13	13
Obesity[21,41,60]	3	4887	40 (26-55)	68 (48-84)	1.0 (0.9-1.2)	45	0.99 (0.88-1.1)	44	13	13
Prior CABG[23,31,58,70]	4	5902	9.1 (6-14)	91 (87-94)	0.97 (0.5-2.1)	77	1.00 (0.92-1.1)	77	13	13

Clinical Exam – Characteristic of ACS Chest Pain

1. Radiation to both arms was most convincing with a LR 2.6
2. Pain similar to previous ischemic events LR 2.2
3. A change in the pain pattern over the past 24h – LR 2.0
4. Typical chest pain for them LR 1.9
5. Worse with exertion up to LR 1.8
6. Then the rest are 1.5 or less, including: radiation to neck or jaw, radiation to L arm, radiation to R arm, diaphoresis, dyspnea, abrupt onset.

Table 2. Performance of Chest Pain Characteristics in Diagnosing Acute Coronary Syndrome[a]

	No.		% (95% CI)						%[c]	
Test	Studies	Patients	Sensitivity	Specificity	LR+ (95% CI)	I^2, %[b]	LR− (95% CI)	I^2, %[b]	PPV	NPV
Radiation to both arms[49]	1	2718	11 (8.3-15)	96 (95-96)	2.6 (1.8-3.7)		0.93 (0.89-0.96)		28	12
Pain similar to prior ischemia[49]	1	2718	47 (42-53)	79 (77-80)	2.2 (2.0-2.6)		0.67 (0.60-0.74)		25	9
Change in pattern over prior 24 h[49]	1	2718	27 (23-32)	86 (85-88)	2.0 (1.6-2.5)		0.84 (0.79-0.90)		23	11
"Typical" chest pain[d,47,49,54,60,62,71]	6	14 584	66 (58-74)	66 (49-83)	1.9 (0.94-2.9)	98	0.52 (0.35-0.69)	95	22	7
Worse with exertion[e,49,73]	2	5049	38-53	73-777	1.5-1.8		0.66-0.83		18-21	9-11
Radiation to neck or jaw[37,49,60]	3	4018	24 (15-36)	84 (76-90)	1.5 (1.3-1.8)	0	0.91 (0.87-0.95)	7.2	18	12
Recent episode of similar pain[73]	1	2331	55 (50-60)	56 (54-59)	1.3 (1.1-1.4)		0.80 (0.71-0.90)		16	11
Radiation to left arm[37,47,49]	3	13 613	40 (28-54)	69 (61-76)	1.3 (1.2-1.4)	0	0.88 (0.81-0.96)	69	16	12
Radiation to right arm[49]	1	2718	5.4 (3.4-8.3)	96 (95-97)	1.3 (0.78-2.1)		0.99 (0.96-1.0)		16	13
Associated diaphoresis[e,49,60]	2	3249	24-28	79-82	1.3-1.4		0.91-0.93		16-17	12-12
Associated dyspnea[49,60,62]	3	3648	45 (42-49)	61 (59-63)	1.2 (1.1-1.3)	0	0.89 (0.82-0.96)	0	15	12
Abrupt onset[49]	1	2718	76 (71-80)	32 (30-34)	1.1 (1.0-1.2)		0.75 (0.61-0.91)		14	10
Any improvement with nitroglycerin[40,66,73]	3	3218	71 (23-95)	35 (44-86)	1.1 (0.93-1.3)	86	0.90 (0.85-0.96)	0	14	12
"Typical" radiation[e,f,54,62]	2	560	25-32	69-96	1.0-5.7		0.78-0.98		13-46	10-13
Burning pain[e,49,60]	2	3249	12-16	84-92	1.0-1.4		0.97-1.0		13-17	13-13
Associated nausea/vomiting[e,49,60]	2	3249	21-22	77-80	0.92-1.1		0.98-1.0		12-14	13-13
Associated palpitations[60]	1	3487	6.0 (3.5-10)	91 (88-94)	0.71 (0.37-1.3)		1.0 (0.98-1.1)		10	13
Associated syncope[73]	1	2331	9.0 (6.4-12)	84 (82-85)	0.55 (0.39-0.76)		1.1 (1.1-1.1)		8	14
Pleuritic pain[e,37,49]	2	3487	18-36	78-93	0.35-0.61		1.1-1.2		6.6-8.4	14-15

Abbreviations: LR+, positive likelihood ratio; LR−, negative likelihood ratio.

[d] "Typical" chest pain was defined by the individual studies, or when studies

Of course, we're talking about 'ruling out' ischemic heart disease. So aside from our awesome history and physical, we are going to need some investigations to feel confident:

- ECG:
    - New, or presumably new ST segment deviations, >1mm in at least two anatomically contiguous leads
    - T wave inversion in multiple precordial leads
- Troponins:

- Any elevation in troponins
- Many authorities will have pathways for this that include repeating the troponin to assess for delta and possibly application of a rule such as the HEART score

The management of a diagnosed STEMI or NSTEMI is covered in Topic 56, but generally this involves ASA chewed, nitroglycerin if they're not hypertensive or it's not an inferior infarct, heparin and clopidogrel, and either fibrinolysis or cardiac catheterization. Cocaine associated ACS needs repeat dosing of benzodiazepines.

Outro for Part I

That marks the end of Chest Pain part one.

Thanks for sticking with us on that. Loads of info, but I think a lot of it is clinically relevant in addition to exam relevant, which is nice.

We won't waste too much of your time, please stick with us for part 2 to come next week.

Introduction for Part Two

Hello again. Welcome back to The GenerEhlist 105 Topics podcast, part two of Key Topic, Chest Pain.

That's Caleb Dusdal, and I'm Chris Cochrane. IF you haven't listened to part 1, go back and do that now as that includes objective 1 to 3. Today we'll cover objectives 4 and 5.

Objective Four

Given an appropriate history of chest pain suggestive of herpes zoster infection, hiatal hernia, reflux, esophageal spasm, infections, or peptic ulcer disease:

a) **Propose the diagnosis.**
b) **Do an appropriate work-up/follow-up to confirm the suspected diagnosis.**

Herpes Zoster

This is one key reason why it is important to fully inspect your patient presenting with chest pain. It would be embarrassing to miss this as a cause. Like actually look at their chest.

As you know this is due to reactivation of latent varicella-zoster from childhood chicken pox that has been lying in wait in the ganglion of the spinal nerve.

- Presentation
  usually pain or dysesthesia precedes visual evidence by 3-5 days.
  This is followed by erythematous papules, which progress to clusters of vesicles on an erythematous base
  And remember the pathophysiology, these will always present in a dermatomal area. Most often thoracic, but can also be in the trigeminal branches of the face.
  These vesicles turn to pustules, and then crust over in about one week.

Diagnosis

This is going to be a clinical diagnosis based on the history of chicken pox or previous shingles flares, and the appearance of the lesions. A swab could be taken from a freshly opened vesicle for NAAT, but this really isn't necessary.
If the first or second branches of the trigeminal nerve are involved, have a close look at the eyes for dendritic lesions.

Management

This a self limited condition, but long term neuralgic pain can persist. Antivirals can help with:

- Reducing healing time
- Decreasing new lesion formation
- Reducing risk of post-herpetic neuralgia

Any benefit we hope to see requires this be started within 72 hours of symptom onset, which means 72 hours from the initial discomfort, not lesion formation.
For the Immunocompetent Patient:

- Acyclovir 800mg PO five times daily x 7 days
  or
- Valcyclovir 1 gram PO tid for 7 days
  Immunocompromised:
- Acyclovir 10mg/kg IV q8h for 7-10 days
- (Valcyclovir should not be Rx due to risk of HUS)

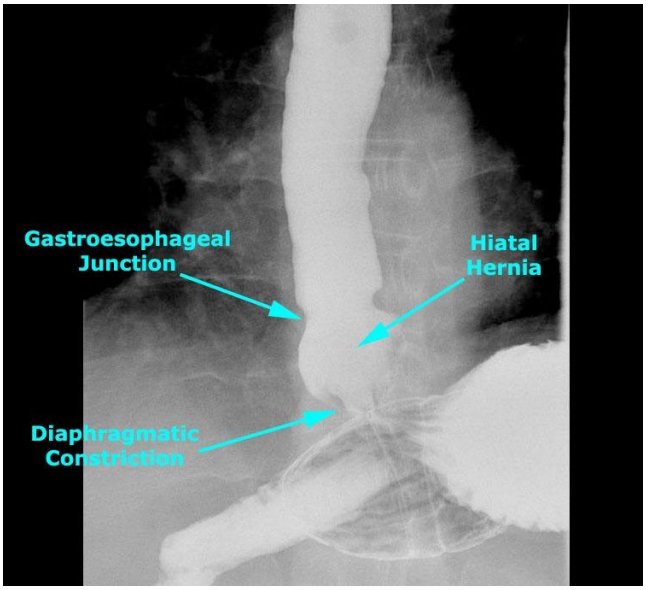
The zoster vaccine markedly reduces morbidity from herpes zoster and post-herpetic neuralgia among older adults.

Hiatal Hernia [Merck Manual 19th Ed Sec2 pp 126]

This is a protrusion of the stomach up through the diaphragm. Most are asymptomatic, but may increase incidence of acid reflux.
Sliding hiatal hernia is the most common, with a paraoesophageal variety being less common.

Diagnosis

The connection between symptoms and the hernia are not entirely clear. In fact, over 40% of cases are discovered incidentally on x-ray.
Definitive diagnosis, particularly for smaller ones, is with barium swallow.



Clinical Presentation
most are asyptomatic.
But retrosternal chest pain can occur.
Occult or massive GI haemorrhage can occur with either type.
Paraoesophageal type can incarcerate and strangulate as a rare complication.

Management
PPI or H2 blocker can be trialed

- Asymptomatic sliding hiatal hernia requires no intervention aside from symptom management for GERD
- A paraoesophageal hernia should be reduced surgically because of the risk for strangulation

Reflux

Commonly accredited to incompetence of the lower oesophageal sphincter, allowing gastric contents into the oesophagus causing the burning retrosternal pain.

Prolonged exposure of the oesophagus to gastric acidic contents can lead to oesophagitis, strictures, or even metaplasia and cancers.

Diagnosis
Clinical in vast majority of cases

Clinical Presentation

- 'heartburn' retrosternal burning pain with or without regurgitation of contents into the mouth.
- if an oesophagitis develops may see: odynophagia or even oesophageal haemorrhage
- resulting strictures can cause progressive dysphagia for solids
- PUD is usually localized to xiphoid region
- Infants with this present with vomiting, irritability, anorexia

Management

If symptoms are irregular or only in specific contexts, they can first trial antacids prn.

- Lifestyle measures should always be used:
  - Elevate head of bed, no meals before lying down or going to sleep.
  - Cessation of: alcohol, fats, chocolate, anticholinergic meds, smoking, spicy foods
  - Weight loss

Most often a trial of PPI or H2 blockers is used, and if they respond, the diagnosis is presumed.

Those unresponsive to empiric therapies above, may require endoscopy to assess for structural complications, with biopsy to assess for H Pylori infection, and dysplasia suggesting Barrett's.

Based on a recent CMAJ publication, the Canadian Task Force recommends NOT screening folks with chronic GERD for Barrett's or Adenocarcinoma https://canadiantaskforce.ca/new-guideline-recommends-against-screening-for-esophageal-adenocarcinoma-in-people-with-chronic-gerd/

Esophageal spasms can occur, giving upper abdominal or chest pain, and they may be related to reflux. They will not kill you, so rule out the other causes of pain first before landing on this diagnosis.

Peptic ulcer disease is like the more painful scary cousin of reflux. Again this can give you upper abdominal or chest pain, usually more constant. There may be abdominal tenderness or evidence of peritonitis of the ulcer erodes significantly. If that happens these patients will be sick. In the more milder forms, treat like reflux with ppi's. Again, testing for H pylori is indicated, if other causes are excluded.

Objective Five

Given a suspected diagnosis of pulmonary embolism:

a) **Do not rule out the diagnosis solely on the basis of a test with low sensitivity and specificity.**
b) **Begin appropriate treatment immediately.**

Since we moved PE from the earlier objective, we're including more than just objective 5 here.

Risk factors that might up your suspicion (think of your Well's score):

- recent surgery – especially orthopaedic
- recent trauma
- prolonged immobility > 8 hours
- active cancer
- on oral contraceptives or hormone replacement therapy (particularly if current smoker)
- procoagulating syndromes
- or a history of VTEs

However, up to 50% of first time VTE are deemed unprovoked

A clinical scenario suggestive of this is, often sudden onset of:

- sharp chest pain, which may worsen with inspiration aka 'pleuritic'. Some will report chest wall tenderness
- dyspnea
- hypoxaemia
- syncope, or shock
- may be associated haemoptysis or cough
- they may also be febrile and have a unilateral leg swelling currently or on recent history

Suggestive Physical exam findings:

- tachypnea, tachycardia and hypoxaemia
- distended jugular veins or hypotension if significant R heart dysfunction

- While we often hear about the legendary S1Q3T3 finding, but the most common ECG finding for pulmonary embolism is sinus tachycardia. May also see TWI in V1-V4 if significant right heart strain

A high pre-test probability needs to get a CT-PA

Low pre-test probability can be essentially ruled out with a negative D-dimer of PERC rule

Management even prior to diagnosing the PE

- Anticoagulation is the treatment
- If massive PE, defined as sBP <90 for longer than 15 minutes, or a more than 40mmHg drop from their sBP baseline, then fibrinolysis should be considered if no contraindications
    - If available in your region, surgical embolectomy may be considered in young patients with massive, proximal PE causing haemodynamic instability

Rather than pick on one test, we are going to run through the recommended pathway from Thrombosis Canada for a Pulmonary Embolism.

If your gestalt suggests a PE, and they are haemodynamically stable, skip everything and send them for a CT-PA.

If they're stable but it's not exactly clinically clear, Thrombosis Canada suggests running the major variables for a DVT/PE through the Wells Score. If they score above 4.5, then you cannot say it is low-likelihood and further steps are needed. If it is below 4.5 on Wells, then you can use the PERC score. Technically the algorithm doesn't say PERC score, but does leave room for interpretation of "ASSESS CLINICAL PROBABILITY"

Table 1: Wells Score* for PE

Variable	Points
Clinical signs and symptoms of DVT	3
Previous DVT or PE	1.5
Immobilization for >3 days or surgery within 4 weeks	1.5
Heart rate >100 beats/minute	1.5
Hemoptysis	1
Malignancy	1
No alternative diagnosis more likely than PE	3
Total Score*	

*Total Score: PE unlikely <4.5; PE likely ≥4.5

So, say your patient scores 2.5 on the Wells because they had cancer and a previous VTE. This is considered 'PE unlikely' and so we can run it through the PERC score.

Now this is a rule-out score, so it is veeeery sensitive. If your patient scores zero here, we can comfortably exclude a Pulmonary Embolism as less than 2% likely.

However, if your patient with low-pretest probability based on gestalt/Wells Score gets even one point on the PERC, then you need to get a D-Dimer. This is quite sensitive and can also rule-out a PE if it is negative. The cut offs that have been used are:

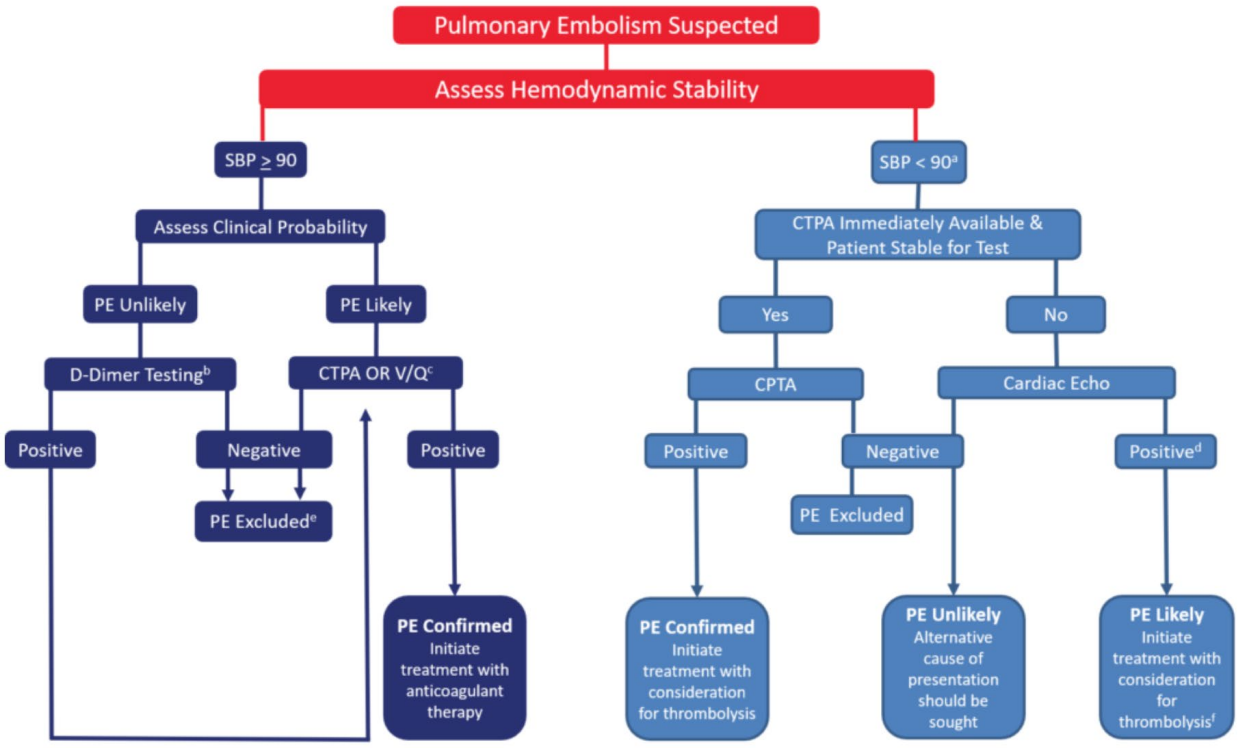
- Age x 10ug/L for patients over 50yo, or
- 500ug/L for everyone under 50 years of age.

Now, if your D-Dimer is positive, you need to get a CT-PA

If your Wells Score was 'high probability' or 4.5 points or higher, then you're going straight to CT-PA as this is the best test.

Table 2: PE Rule-out Criteria (PERC) for patients with low pretest probability for PE

Clinical characteristic	Meets criteria	Does not meet criteria
Age <50	0	1
Initial heart rate <100 beats/min	0	1
Initial SaO2 >94% on room air	0	1
No unilateral leg swelling	0	1
No hemoptysis	0	1
No surgery or trauma ≤4 weeks	0	1
No history of VTE	0	1
No estrogen use	0	1



This algorithm also includes an arm for if they can't be sent for a CT, because they're too unstable for an inhospital CT or because they're unstable and your site doesn't have a CT scanner. In these cases you'll need to use all your other means of diagnosing a PE like POCUS and ECG, then make some hard decisions regarding treatment with a thrombolytic. That brings us to management of PE's.

c) Begin appropriate treatment immediately.

Unless bleed risk is high such as the patient is actively bleeding or they are immediately post-operative, quick acting anticoagulation should be initiated for anyone with high probability of a Pulmonary Embolism. Meaning, before the confirmatory testing with CT-PA is done.

If they have low or intermediate pre-test probability, then this can be held as long as testing can be done shortly: within 24 hours for low risk, or 4 hours for intermediate risk.

Anticoagulation options are:

- If thrombolysis for haemodynamic instability is being considered, then IV unfractionated heparin is preferred
- Otherwise a DOAC as monotherapy is generally preferred: Apixaban or Rivaroxaban
- Low molecular weight heparin bridge to Dabigatran and Edoxaban, or to warfarin are options.
- Low molecular weight heparin monotherapy is still recommended for patients with active cancer

All VTEs should be treated with anticoagulation for at least 3 months, unless there is no apparent trigger or it is recurrent, in which case they may need longer treatment. Talk to your Haematology colleagues.

Thrombosis Canada https://thrombosiscanada.ca/clinicalguides/#

Part Two Outro

That's a wrap!

Shout out to the fellow generalists who have reached out to help, we are excited to show you what they've made in the coming months, and also to those who have put up some 5 star ratings onto the Apple Podcasts app. Very much appreciated.

Referenced Works